5. Assessing the effects of drug use on the transmission dynamics of HIV/AIDS

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Abstract. Drug misuse has been recognized to have a significant impact on the spread of HIV/AIDS epidemic. A deterministic model to theoretically assess the contribution of drug misuse to the spread of HIV/AIDS is investigated. The basic reproduction number of the model is determined and stability of equilibria analysed. The disease-free equilibrium point is shown to be globally asymptotically stable when its corresponding basic reproduction number is less than unity. The Centre Manifold theory is used to show that the endemic equilibrium point is locally asymptotically stable when its corresponding reproduction number is greater than unity. The results obtained from the analysis of the basic reproduction number show that drug use increases the basic reproduction number. This suggests that drug misuse has the potential to fuel the epidemic. Results from numerical simulations show that HIV infected drug abusing individuals progress to the AIDS stage and die at a faster rate than their corresponding HIV infected parts.

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1. Introduction

Infectious diseases have for centuries ranked with wars and famine as major challenges to human progress and survival [28]. HIV, the Human Immunodeficiency Virus, is the etiological agent of Acquired Immunodeficiency Syndrome (AIDS) which is most often transmitted either through sexual intercourse with an infected individual or through injecting drug use. As a global killer, AIDS now threatens to surpass the Black Death of the fourteenth century and the 1918-1920 influenza pandemic, each of which killed at least 50 million people [24, 25]. HIV, which has infected more than 60 million people globally [52], is the largest single cause of human immune deficiency and markedly increases vulnerability to a wide range of opportunistic pathogens including herpes viruses, tuberculosis and hepatitis to name a few. In adults, there is long and variable latent period associated with HIV infection and the onset of HIV related diseases including AIDS. HIV/AIDS have killed more than 25 million people since it was first recognized in 1981 making it one of the most destructive epidemics in history [53]. It remains one of the leading causes of death in the world, with its effect most devastating in sub-Saharan Africa, where HIV prevalence ranges between 12% and 42% [40], and the region has remained the epidemiological locus of the disease.

In the early stages of the pandemic, HIV transmission occurred mainly through unprotected sex, but there are reports of transmission attributable to drug injection [41, 42]. Drug use-related HIV transmission has become a major concern in Africa as well. This situation is most dramatic in Mauritius where the HIV epidemic is driven primarily by injecting drug. High-risk behavior such as the use of non-sterile injecting equipment and unprotected sex are common in injecting drug user populations, in whom HIV prevalence is high. Up to half of the injecting drug users tested in Mombasa and Nairobi (Kenya) were found to be HIV positive, as were 26% in Zanzibar and 28% in South Africa [32, 35]. Injecting drug use is a growing phenomenon in South Africa, where injecting of heroin has increased in recent years in the Gauteng and Mpumalanga provinces, while the trend appears to be fluctuating in the port city of Cape Town [38]. As a result, heroin injection has come to play a predominant role in fueling the AIDS epidemic in South Africa [37]. Unfortunately, in the Southern Africa region, there is a widespread lack of awareness about where to access HIV treatment and preventive services, and numerous barriers to accessing appropriate HIV and drug interventions [37]. Consequently, multiple risk behaviours in vulnerable populations and lack of access to HIV prevention services could accelerate the spread of HIV [36].
Studies suggest that cocaine and heroin increase HIV replication and suppress immune function [45]. Drug misuse is a major factor in the spread of HIV infection, being linked with unsafe sexual activity, as individuals who use drugs or engage in high-risk behaviors associated with drug use increase their risk not only of contracting and transmitting HIV, but also hepatitis B and C, tuberculosis, as well as a number of sexually transmitted diseases, including syphilis, chlamydia, trichomoniasis, gonorrhea, and genital herpes. Injecting drug users (IDUs) are also commonly susceptible to skin infections at the site of injection and to bacterial and viral infections, such as bacterial pneumonia and endocarditis, which, if left untreated, can lead to serious health problems [33]. Behaviors associated with drug use are among the main factors in the spread of HIV infection in the United States. Similarly, in Mauritius, 75% of sex workers reported injecting drug and 23% said they never used non-sterile injecting equipment [41]. Among drug-using sex workers in Mauritius who had previously been tested for HIV, 13% were infected with the virus, yet 68% of the sex workers said they had consistently used condoms during the previous three months [42]. There is therefore a stronger need for prevention efforts focusing on injecting drug users, and especially those who are also sex workers [9].

Drugs can change the way the brain works, disrupting the parts of the brain that people use to weigh risks and benefits when making decisions [21]. Drug misuse and increased spread of HIV/AIDS are correlated. In fact, drug and alcohol use can also be dangerous for people who are taking antiretroviral medications (ARVs), because they are less likely to take all of their medications, and street drugs may have dangerous interactions with ARVs. Lack of adherence to treatment recommendations in general is so widespread that no grouping of sociodemographic or psychosocial characteristics can reliably predict it [12]. There is potential for serious interactions between drugs and ARVs which can lead to under or overdoses of ARVs and which can be fatal. Using alcohol or drugs before or during sexual activity greatly increases the chances that one will not follow safer sex guidelines. There is also evidence of a strong relation between alcohol use and HIV infection: drinkers have a 70% increased risk of being HIV-positive when compared with nondrinkers [10]. Substance use can lead to multiple partners which can again increase one's infection risk as well as the number of persons to whom infection can be transmitted. Also, drug misuse causes many problems both to individuals and to societies, including loss of productivity, transmission of infectious diseases, crime, family and social disorder, and excessive health care expenditures [26]. Drug misuse can disproportionately exacerbate HIV transmission, and
therefore efforts to mitigate drug misuse may likewise help stem the HIV/AIDS epidemic [39].

HIV/AIDS has received significant attention from the mathematical standpoint, and a wide range of papers have been written on its transmission dynamics and the impact of interventions on its prevalence [47, 46, 48, 50, 51, 14]. There is a great deal of work on developing and studying the properties models of the transmission of the sexual spread of HIV [11, 30] and in particular the importance of heterogeneous mixing [17, 16], contact structure [17], random allocation model for the transmission of HIV by needle sharing among a group of intravenous drug users who are friends or relatives (buddy-users) [13]. While Behrens et al [19] consider treatment in a dynamic model of drug initiation which we do not, they conclude that prevention can temper drug prevalence and consumption, but drug treatment's effectiveness depends critically on the stage in the epidemic in which it is employed, as reducing the number of heavy users in the early stages of an epidemic can be counter-productive if it masks the risks of drug use, thereby removing a disincentive to initiation. The interdependence between drug misuse and HIV is the main motive behind this study. The resulting model may be termed the two epidemics: external epidemic (injecting drug use) and, homogeneous epidemic model for HIV/AIDS [20]. Greenhalgh [15] study the effects of heterogeneity on the spread of HIV and AIDS among a population of injecting drug users, while we focus on the population level impact of the latter on the former. This study has some fundamental difference from those found in the literature. Volz et al., [55] estimate the contact network structure for an Atlanta community with heterogeneous sexual and drug-related risk behaviors and build a detailed transmission model for HIV through this population. We formulate mathematical models to study the effects of drug misuse on the transmission dynamics of HIV/AIDS, obtain formulas for the reproductive ratio by investigating the stability of the disease-free equilibrium and determine its global stability when the reproductive ratio is less than or equal to one. Study of the existence of the endemic equilibrium and partial results for the local stability of the endemic equilibrium are presented. Choosing certain parameter values, numerical simulations are carried out and conclusions based on the simulation results are drawn.

The paper is organized as follows. In Section 2, the model description and its basic properties are presented. In Section 3, stability analysis of the model is carried out. In order to investigate the relative importance of the different factors responsible for initial disease transmission, a sensitivity analysis of the factors driving drug misuse is carried out. Numerical simulations of the model are reported in Section 4, and the paper concludes with a Discussion in Section 5.
2. Model description

The model sub-divides the total sexually active human population at time \( t \), denoted by \( N \), into the following sub-populations of susceptible drug misusers \( S_D(t) \), susceptible non-drug misusers \( S_N(t) \), infected drug misusers \( I_D(t) \), infected non-drug misusers \( I_N(t) \) (with no symptoms of AIDS), drug misusers AIDS cases \( A_D(t) \), non-drug misusers AIDS cases \( A_N(t) \): AIDS patients in this model are assumed to be sexually inactive, but drug misusers AIDS patients \( A_D(t) \) are assumed to influence the dynamics of the virus through drug sharing by injections since they are now addicted. Individuals are referred to as drug users or non drug users only in connection with their behavior and in particular drug users to non drug user infectivity rate is greater than non-drug user to drug user. The population is assumed to be uniform and homogeneously mixing. The total population is given by;

\[
N = S_D(t) + S_N(t) + I_D(t) + I_N(t) + A_D(t) \tag{1}
\]

It is assumed that sexually active susceptible individuals are recruited into the population through aging and migration at a constant rate \( \Lambda \): A proportion \( \pi_0 \) of these individuals are assumed to be drug misusers \( S_D(t) \) while the complementary \( (1 - \pi_0) = \pi_1 \) denotes the non-drug misusers \( S_N(t) \). Susceptible drug misusers and non-drug misusers acquire HIV infection at rates

\[
\lambda_D = \frac{\beta c(\eta I_D(t) + I_N(t) + \theta A_D(t))}{N(t)} \quad \text{and} \quad \lambda_N = \frac{\beta c(I_D(t) + I_N(t))}{N(t)}, \text{ respectively} \tag{2}
\]

In this case, \( \beta \) is the effective sexual contact and/or drug sharing injection rate, \( c \) is the rate of acquiring new partners (sexual and/or drug injecting). The modification parameters for the effect of drug misuse are \( \eta \) and \( \theta \). Infected drug users transmit HIV to susceptible drug users at a higher rate than they transmit HIV to susceptible non-drug users because they can transmit the virus to the former group both through sexual intercourse and through needle sharing. The parameter \( \eta > 1 \) models the fact that HIV positive drug users infect more people as they infect through needle sharing and sex. Likewise, drug users with AIDS can transmit HIV to susceptible drug users because of needle sharing and the impairment of judgment under drug addiction, whereas this is not possible for susceptible non-drug users. The term \( \theta A_D(t) \) represents this infection route, where the parameter \( \theta \in (0, 1) \) accounts for the reduced infectiousness of drug users who are in the AIDS stage due to their no longer being sexually active.
Hence, this equation embodies that drug users have a higher risk of contracting HIV than non-drug users because drug users have an increase probability of infection through drug sharing by use of injections and needles. HIV infected individuals progress to AIDS stage at a constant rate $\gamma > 0$ for drug users and non-drug users. The natural death rate $\mu > 0$ is assumed to be proportional to the number in each class. AIDS patients have an additional disease-induced mortality $\nu > 0$. It is also assumed that drug users AIDS patients will cease to use drugs due to their deteriorating health and also because they may constantly be under monitoring, and thus, they will move into the class of non-drug users AIDS patients at a constant rate $\phi > 0$.

The model flow diagram is shown in Figure 1.

Putting the formulations and the assumptions together gives the following deterministic system of non-linear ordinary differential equations,
This model makes a number of assumptions that we will now describe. First, the population is assumed to be uniform and homogeneously mixing, although we note that models where transmission is restricted to occur through sexual partnerships give different predictions [48, 49]. Secondly, it is assumed that sexual risk behaviours are the same for drug users as for non-drug users, though in reality they may be different. Thirdly, it is assumed that drug users in the AIDS stage transmit at a reduced rate to susceptible drug users, although conceivably this transmission rate could actually be higher during the AIDS stage because the viral load is much higher during AIDS than during earlier stages of HIV infection. Fourth, we have not attempted to include a detailed model of how individuals enter or leave the drug using population or how their HIV status influence their mixing patterns, except for the term $\phi$ in the above equations representing drug-using AIDS patients leaving the drug using population.

2.1. Model basic properties

In this section, we study the basic results of solutions of model system (3), which are essential in the proofs of stability results.

**Lemma 1.** The equations preserve positivity of solutions.

**Proof.** The vectorfield given by the right hand side of (3) points inward on the boundary of $\mathbb{R}_+^n \setminus \{0\}$. For example, if $I_D = 0$ then $\dot{I}_D = \lambda_D S_D \geq 0$. All the other components are similar.

**Lemma 2.** Each non-negative solution is bounded in $L^1$-norm by $\max \{N(0), \Lambda/\mu\}$

**Proof.** The norm $L^1$ norm of each non-negative solution is $N$ and it satisfies the inequality $N' \leq \Lambda - \mu N$. Solutions to the equation $M' = \Lambda - \mu M$ are
monotone increasing and bounded by $\Lambda/\mu$ if $M(0) < \Lambda/\mu$. They are monotone decreasing and bounded above if $M(0) \geq \Lambda/\mu$. Since $N' \leq M'$ the claim follows.

**Corollary 1.** The region

$$\Phi = \left\{ (S_D, S_N, I_D, I_N, A_D, A_N) \in \mathbb{R}^6_+ : N \leq \frac{\Lambda}{\mu} \right\}.$$  \hfill (4)

is invariant and attracting for system (3).

**Theorem 1.** For every non-zero, non-negative initial value, solutions of model system (3) exist for all times.

Proof. Local existence of solutions follows from standard arguments since the right hand side of (3) is locally Lipschitz. Global existence follows from the a-priori bounds.

The model has a number of invariant sets that correspond to epidemiologically limiting cases of the problem. The effect of drug misuse on the spread of HIV/AIDS is modelled via the additional infection pathways with corresponding parameters $\gamma_i$ and $\delta$ and the recruitment of individuals into the two classes is determined by $\pi_0$, $\pi_1$.

If we set $\eta = 0$, $\theta = 0$, the we can simply add the respective compartments $S = S_D + S_A$ and so on, and we obtain a standard SIR-model. If, on the other hand, we allow only one group to enter the system, then we obtain invariant sets.

**Lemma 3.** If $\pi_0 = 0$ then the set $\{S_D = I_D = A_D = 0\}$ is invariant and attracting for system (3). If $\pi_1 = 0$ then the set $\{S_N = I_N = 0\}$ is invariant and the set $\{S_N = I_N = 0, A_N = \frac{\phi}{\mu + \psi}A_D\}$ is attracting.

### 3. Equilibrium states, reproductive ratio and stability

In this section, we compute the equilibrium states, namely the disease-free equilibrium (DFE) and the endemic equilibrium (EE), and investigate their stability using the basic reproductive number.

#### 3.1. Disease-free equilibrium and the basic reproduction number

Model system (3) has a DFE given by,
The linear stability of $\mathcal{E}^0$ is governed by the basic reproduction number $\mathcal{R}_0$ which is defined as the spectral radius of the next generation matrix [54]. The basic reproduction number can often be interpreted as the expected number of secondary infections produced by a single infectious individual during his/her entire infectious period. However, in our model an infectious individual can be in one of the three classes $I_D$, $I_N$, $A_D$ and the expected number of secondary infections depends on the class. We consider the different possibilities in detail.

Following van den Driessche and Watmough [54], we calculate the next generation matrix as

$$
\mathcal{K} = \beta c \begin{bmatrix}
\frac{\pi_0}{\gamma + \mu} & \frac{\theta \pi_0}{\gamma + \mu} & \frac{\pi_0}{\phi + \mu + \nu} & 0 \\
0 & \frac{\pi_1}{\gamma + \mu} & \frac{\pi_1}{\gamma + \mu} & 0 \\
0 & 0 & 0 & 0
\end{bmatrix}.
$$

The rows and columns refer to $I_D$, $I_N$, $A_D$ in that order. Since individuals in class $A_N$ do not contribute to new infections, we can ignore them for $K$. The $(i, j)$-entry of this matrix is the expected number of secondary infections in class $i$ resulting from a single primary infective in class $j$. We consider two special cases first.

**Case 1: There is no drug misuse in the community**

We set $\pi_0 = 0$. Then the matrix $K$ has rank one and the spectral radius is given by

$$
\mathcal{R}_{0_N} = \frac{\beta c}{\gamma + \mu}.
$$

This reproductive rate sometimes referred to as *the back of the napkin* [1] is simply the ratio of the per capita rate of infection and the average lifetime of an individual in class $I_N$. It is defined as the number of secondary HIV cases produced by a single infected individual during his/her entire infectious period in a totally naive (susceptible) population in the absence of drug misuse individuals.
Case 2: The entire population is made up of drug misusers

In this case, we have $\pi_1 = 0$. This model is similar to the one in [18], but there are some fundamental differences. While their model focuses on individual's response to a drug, ours is derived from a population in which HIV/AIDS is prevalent.

The matrix $K$ has rank one, and the spectral radius is given by

$$\mathcal{R}_{0D} = \frac{\beta c \eta}{\gamma + \mu} + \frac{\gamma}{(\gamma + \mu)(\phi + \mu + \nu)} \frac{\beta c \theta}{(\phi + \mu + \nu)} = \mathcal{R}_{0N} \left( \eta + \frac{\gamma \theta}{(\phi + \mu + \nu)} \right).$$

The first term in this expression is the expected number of secondary cases infected while the individual was in the $I_D$-class. The second term is the probability that the individual progresses to the AIDS stage before they die, multiplied by the expected number of secondary infections during the average time spent in the AIDS class. Under our assumption $\eta > 1$ and/or $\theta > 0$ we obtain $\mathcal{R}_{0D} > \mathcal{R}_{0N}$; Hence, the additional pathways of disease transmission for drug-misusers increase the likelihood of disease spread.

Case 3: The general case

When $\pi_0, \pi_1 > 0$, the matrix $K$ has rank two and its spectral radius can be calculated explicitly as

$$\mathcal{R}_{DN} = \mathcal{R}_{0N} \left[ \frac{\eta \pi_0 + \pi_1}{2} + \frac{\gamma \theta \pi_0}{2h_3} + \frac{1}{2} \sqrt{\left( \eta \pi_0 - \pi_1 \right) + \frac{\gamma \theta \pi_0}{h_3}} \right]^2 + 4\pi_0 \pi_1,$$

with $h_3 = \phi + \mu + \nu$ throughout the paper. The next result follows from Theorem 2 in [54].

**Theorem 2.** The disease-free equilibrium of model (3) is locally asymptotically stable if $\mathcal{R}_{DN} < 1$, and unstable otherwise.

Using a theorem from Castillo-Chavez et al. [5], we can even show global stability of the DFE in the case that the reproduction number is less than unity.

**Theorem 3.** The DFE $E_0$ of our model system (3) is globally asymptotically stable provided $\mathcal{R}_0 < 1$

**Proof.** Following Castillo-Chavez et al. [5], we write system (3) in the form
where $X = (S_D, S_N)$ and $Y = (I_D, I_N, A_D, A_N)$. Here $X \in \mathbb{R}_+^2$ denotes (its components) the number of uninfected individuals and $Y \in \mathbb{R}_4^+$ denoting (its components) the number of infected individuals. The disease-free equilibrium is now denoted by $\mathcal{E}^0 = (X_0, 0)$ where $X_0 = \left( \frac{\pi_0 \Lambda}{\mu}, \frac{\pi_1 \Lambda}{\mu} \right)$. We have to prove that the two conditions

(H1) For $X'(t) = F(X, 0)$, $X$ is a globally asymptotically stable

(H2) $G(X, Y) = UY - \hat{G}(X, Y)$, $\hat{G}(X, Y) \geq 0$ for $(X, Y) \in \Phi_1$, (11)

are satisfied where $\Phi_1$ is a positively invariant attracting domain.

Consider $F(X, 0) = \left[ \begin{array}{cc} \frac{\pi_0 \Lambda - \mu S_D}{\pi_1 \Lambda - \mu S_N} & \end{array} \right]$.

$$U = \begin{bmatrix} -h_1 + \eta \beta c \pi_0 & \beta c \pi_0 & \theta \beta c \pi_0 & 0 \\ \beta c \pi_1 & \beta c \pi_1 - h_1 & 0 & 0 \\ 0 & 0 & -h_3 & 0 \\ \gamma & \gamma & \phi & -(\mu + \nu) \end{bmatrix}$$ (12)

and

$$\hat{G}(X, Y) = \begin{bmatrix} \hat{G}_1(X, Y) \\ \hat{G}_2(X, Y) \\ \hat{G}_3(X, Y) \\ \hat{G}_4(X, Y) \end{bmatrix} = \begin{bmatrix} \beta c (\eta I_D + I_N + \theta A_D) \left( \frac{\pi_0}{N} - \frac{S_D}{N} \right) \\ \beta c (I_D + I_N) \left( \frac{\pi_1}{N} - \frac{S_N}{N} \right) \\ 0 \\ 0 \end{bmatrix}$$

Therefore, $\hat{G}(X, Y) \geq 0$ whenever $\pi_0 \geq \frac{S_D}{N}$ and $\pi_1 \geq \frac{S_N}{N}$, implying that $\mathcal{E}^0$ is globally asymptotically stable for $\mathcal{R}_{DN} < 1$ in

$$\Phi_1 = \left\{ (S_D, S_N) \in \mathbb{R}_+^2 : S_D \leq \frac{\pi_0 A}{\mu}, S_N \leq \frac{\pi_1 A}{\mu} \right\} \subset \Phi,$$

which is also positively invariant and attracting.

Theorem 3 has obvious public health importance since it tells us that the disease can be eradicated completely from the community in the long run, whenever $\mathcal{R}_{DN} < 1$

### 3.2. Endemic equilibria and stability analysis

Model system (3) has three possible endemic equilibria: the drug free endemic equilibrium with a population of non drug misusers only, the
endemic equilibrium when the whole population is made up of drug misusers and the equilibrium where drug misusers and non drug misusers co-exist, herein referred to as the interior equilibrium point.

3.2.1. Drug free endemic equilibrium

We set \( \pi_1 = 1 \) so that there are no drug misusers entering the community. Since the subspace \( \{ S_D = I_D = A_D = 0 \} \) is attracting, we only consider the three dimensional system of \( S_N, I_N, A_N \): The equation for \( A_N \) decouples, and hence we are left with a two-dimensional system. The basic reproduction number is \( R_{0_N} = c\beta/(\gamma + \mu) \). The endemic equilibrium is given by

\[
S^*_N = \frac{\Lambda}{\beta(1 - \frac{1}{R_{0_N}}) - \mu}, \quad I^*_N = (R_{0_N} - 1)S^*_N, \quad A^*_N = \frac{\gamma}{\mu + \nu}I^*_N. \tag{13}
\]

This state exists and is unique if \( R_{0_N} > 1 \).

**Theorem 4.** If \( \pi_1 = 1 \) and \( R_{0_N} > 1 \) then the unique endemic equilibrium is globally asymptotically stable for the model system (3).

**Proof.** Since all compartments with drug abusing individuals will approach zero, it suffices to prove the claim for the invariant subspace \( \{ S_D = I_D = A_D = 0 \} \), and, in fact, for the two-dimensional system of \( S_N, I_N \) only. The Jacobi matrix of this two-dimensional system at the endemic equilibrium \( (S^*_N, I^*_N) \) is given by

\[
J(S^*_N, I^*_N) = \begin{bmatrix}
-c\beta(1 - \alpha)^2 - \mu & c\alpha\beta \\
c\beta(1 - \alpha)^2 & c\alpha\beta - (\gamma + \mu)
\end{bmatrix}, \tag{14}
\]

where \( \alpha = 1/R_{0_N} \). After a lot of algebra, we find that the trace of \( J \) is negative and the determinant is both positive, hence the steady state is locally asymptotically stable. More precisely,

\[
\text{tr}(J) = c\beta \left( \frac{1}{R_{0_N}} - 1 \right) - \mu, \quad \det(J) = c\beta \gamma \left( 1 - \frac{1}{R_{0_N}} \right)^2 + \mu(\gamma + \mu)(R_{0_N} - 1). \tag{15}
\]

To show global stability, we first note that the domain \( \{ S_N, I_N \geq 0, S_N + I_N < \Lambda/\mu \} \) is positively invariant and attracting for the
two-dimensional system. Then we use the negative criterion by Bendixon and Dulac to rule out the existence of periodic orbits. We multiply by the function $1/I_N$, so that

\[
\frac{S'_N}{I_N} = \frac{\Lambda}{I_N} - \frac{c\beta S_N}{S_N + I_N} - \mu \frac{S_N}{I_N}, \quad \frac{I'_N}{I_N} = \frac{c\beta S_N}{S_N + I_N} - (\mu + \gamma). \tag{16}
\]

Differentiating the right hand side of the first equation with respect to $S_N$ and the right hand side of the second equation with respect to $I_N$ we obtain

\[
-\frac{c\beta I_N}{(S_N + I_N)^2} - \frac{\mu}{I_N} < 0, \quad \text{and} \quad \frac{c\beta S_N}{(S_N + I_N)^2} < 0. \tag{17}
\]

Since the sum of these two expressions is negative, there are no periodic orbits.

### 3.2.2. Drug misuse only endemic equilibrium

This occurs when $S_N = I_N = A_N = 0$, $\pi_1 = 0$ and is given by

\[
E_2 = (S_D^*, 0, I_D^*, 0, A_D^*, 0) \quad \text{where} \quad S_D^* = \frac{\Lambda}{\mu + \lambda_D^*}, \quad I_D^* = \frac{\Lambda \lambda_D^*}{(\mu + \lambda_D^*)(\gamma + \mu)}, \quad A_D^* = \frac{\Lambda \gamma \lambda_D^*}{(\mu + \lambda_D^*)(\gamma + \mu)(\mu + \nu)}, \tag{18}
\]

in terms of the equilibrium value of the force of infection $\lambda_D^*$. Substituting (18) into the equation for the force of infection $\lambda_D^*$ we have

\[
\lambda_D^* g(\lambda_D^*) = \lambda_D^* (K_1 \lambda_D^* + K_2) = 0 \tag{19}
\]

where $\lambda_D^* = 0$ corresponds to the disease-free equilibrium and $g(\lambda_D^*) = 0$ corresponds to the existence of endemic equilibrium point where,

\[
K_1 = \frac{\mu + \nu + \gamma}{(\mu + \nu)(\mu + \gamma)}, \quad K_2 = 1 - R_{0_D}. \tag{20}
\]

Noting that, $K_1$ is always positive and $K_2$ is negative or positive depending on whether $R_{0_D}$ is greater or smaller than 1.

**Theorem 5.** The endemic equilibrium $E_2$ exists whenever $R_{0_D} > 1$. 
Proof. By examining the linear equation \( K_1 \lambda_N^* + K_2 = 0 \), we have that

\[
\lambda_D^* = -\frac{B_2}{B_1} = \frac{\mathcal{R}_{0D} - 1}{K_1}
\]  

(21)

But the disease is endemic when the force of infection \( \lambda_D^* > 0 \) which implies \( \mathcal{R}_{0D} > 1 \). Therefore, the endemic equilibrium \( \mathcal{E}_2 \) exists whenever \( \mathcal{R}_{0D} > 1 \).

To determine the local asymptotic stability of the point \( \mathcal{E}_2 \), we apply the Centre Manifold theory. Let us make the following change of variables in order to apply the Center Manifold theory \( S_D = x_1, I_D = x_2 \) and \( A_D = x_3 \), \( (S_N = I_N = A_N = 0, \pi_0 = 1) \) so that \( \bar{N} = \sum_{n=1}^{3} x_n \). We now use the vector notation \( X = (x_1, x_2, x_3)^T \) Then, the model system (3) can be written in the form \( \frac{dX}{dt} = F = (f_1, f_2, f_3)^T \), such that

\[
x'_1(t) = f_1 = \Lambda - \frac{\beta c(\eta x_2 + \theta x_3)}{\sum_{n=1}^{3} x_n} x_1 - \mu x_1,
\]

\[
x'_2(t) = f_2 = \frac{\beta c(\eta x_2 + \theta x_3)}{\sum_{n=1}^{3} x_n} x_1 - (\gamma + \mu) x_2,
\]

\[
x'_3(t) = f_3 = \gamma x_2 - (\mu + \nu) x_3.
\]

(22)

System (22) is dynamics in invariant subspaces (the boundaries) of the full model which are also attracting/absorbing. If \( \pi_0 = 0 \), then, there is no inflow into the susceptible drug misusers class and at the long run, this class will be depreciated as it ages, while for \( \pi_0 = 1 \), similar conclusion holds for the susceptible non-drug misusers class. There are the borderline (extreme) cases which should be excluded. Thus, \( \pi_0 \in (0, 1) \).

The Jacobian matrix of system (22) at \( \mathcal{E}_{20} = \mathcal{E}_{10} \) is given by

\[
J(\mathcal{E}_{20}) = \begin{bmatrix}
-\mu & \eta \beta c & -\theta \beta c \\
0 & \eta \beta c - (\gamma + \mu) & \theta \beta c \\
0 & \gamma & -(\mu + \nu)
\end{bmatrix},
\]

(23)

from which it can be shown that the reproduction number is given by
If \( \beta \) is taken as a bifurcation parameter and if we consider the case \( \mathcal{R}_{0p} = 1 \) and solve for \( \beta \), we obtain the bifurcation point

\[
\beta = \beta_2^* = \frac{(\gamma + \mu)(\mu + \nu)}{c(\gamma \theta + \eta(\mu + \nu))} \tag{25}
\]

The linearized system of the transformed equation (22) with \( \beta = \beta_2^* \), has a simple zero eigenvalue. Hence, the Centre Manifold theory can be used to analyze the dynamics of (22) near \( \beta = \beta_2^* \) [4]. It can be shown that the Jacobian of (22) at \( \beta = \beta_2^* \) has a right eigenvector associated with the zero eigenvalue given by \( w = [w_1, w_2, w_3]^T \), where

\[
w_1 = -\frac{\beta_2^* c(\eta(\mu + \nu) + \gamma \theta) w_2}{\mu(\mu + \nu)}, \quad w_2 = w_2 > 0, \quad w_3 = \frac{(\eta \beta_2^* c - \gamma - \mu) w_2}{\theta \beta_2^* c} = \frac{\gamma w_2}{\mu + \nu}. \tag{26}
\]

The left eigenvector of \( J(\mathcal{E}_{20}) \) associated with the zero eigenvalue at \( \beta = \beta_2^* \) is given by

\[
z = [z_1, z_2, z_3]^T, \quad \text{where}
\]

\[
z_1 = 0, \quad z_2 = z_2 > 0, \quad z_3 = \frac{(\eta \beta_2^* c - \gamma - \mu) z_2}{\gamma} = \frac{\theta \beta_2^* c z_2}{\mu + \nu} > 0. \tag{27}
\]

**Computations of the bifurcation coefficients \( a \) and \( b \):**

For the sign of \( b \), it is associated the following non-vanishing partial derivatives of \( F \),

\[
\frac{\partial^2 f_1}{\partial x_2 \partial \beta_2^*} = -c \eta, \quad \frac{\partial^2 f_1}{\partial x_3 \partial \beta_2^*} = -c \theta, \quad \frac{\partial^2 f_2}{\partial x_2 \partial \beta_2^*} = c \eta, \quad \frac{\partial^2 f_2}{\partial x_3 \partial \beta_2^*} = c \theta. \tag{28}
\]

From (28) it follows that,

\[
b = \frac{(\eta(\mu + \nu) + \gamma \theta) c z_2 w_2}{\mu + \nu} > 0. \tag{29}
\]

For the sign of \( a \) it is associated with the following non-vanishing partial derivatives of \( F \)
\[
\frac{\partial^2 f_2}{\partial x_2^2} = -\frac{2\eta \beta^2 \mu}{\Lambda}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_3} = -\frac{2c \beta^2 \theta \mu}{\Delta}, \quad \frac{\partial^2 f_2}{\partial x_3^2} = -\frac{2gb^2 \theta}{\Delta}
\]  \hspace{1cm} (30)

From (30) it follows that

\[
a = -\frac{2b \mu c(\mu + \nu)(\mu + \nu + \gamma(\mu + \nu))z_2w_2^2}{(\mu + \nu)^2 \Delta}
\]  \hspace{1cm} (31)

Thus \( a < 0 \) and \( b > 0 \) and using Theorem 4.1 [6] item (iv), we have established the following result.

**Theorem 6.** The unique endemic equilibrium \( \mathcal{E}_2 \) guaranteed by Theorem 4.1 [6] is locally asymptotically stable for \( R_{0D} > 1 \) but close to 1.

The model will exhibit the phenomenon of backward bifurcation if the parameter \( a \) is positive, since \( b \) is in this case is always positive. Since \( a < 0 \), the drug misuse only model does not undergo this phenomenon and the modification parameters \( \eta \) and \( \theta \) do not influence the sign of a \( \left( \frac{\partial a}{\partial \eta} < 0, \frac{\partial a}{\partial \theta} < 0 \right) \)

It is nevertheless important to note that \( \frac{\partial R_{0D}}{\partial \eta} > 0, \frac{\partial R_{0D}}{\partial \theta} > 0 \), and consequently, drug misuse will always have a negative impact on the transmission dynamics of HIV/AIDS in the community.

### 3.2.3. Interior equilibrium point

We now present the endemic equilibrium where both drug misusers and non drug misusers co-exist. This is denoted by \( \mathcal{E}_3 \) with

\[
\mathcal{E}_3 = (S_{D}^{**}, S_{N}^{**}, I_{D}^{**}, I_{N}^{**}, A_{D}^{**}, A_{N}^{**})
\]

where

\[
S_{D}^{**} = \frac{\pi_0 \Lambda}{\lambda_{D}^{**} + \mu}, \quad S_{N}^{**} = \frac{\pi_1 \Lambda}{\lambda_{N}^{**} + \mu}, \quad I_{D}^{**} = \frac{\Lambda \pi_0 \lambda_{D}^{**}}{(\gamma + \mu)(\mu + \lambda_{D}^{**})},
\]

\[
I_{N}^{**} = \frac{\Lambda \pi_1 \lambda_{N}^{**}}{(\gamma + \mu)(\mu + \lambda_{N}^{**})}, \quad A_{D}^{**} = \frac{\Lambda \pi_0 \lambda_{D}^{**}}{(\gamma + \mu)(\mu + \lambda_{D}^{**})(\phi + \mu + \nu)},
\]

\[
A_{N}^{**} = \frac{\Lambda \lambda_{N}^{**} \pi_1}{(\mu + \gamma)(\lambda_{N}^{**} + \mu)(\mu + \gamma)} + \frac{\phi \Lambda \pi_0 \lambda_{D}^{**}}{(\gamma + \mu)(\mu + \lambda_{D}^{**})(\phi + \mu + \nu)(\mu + \nu)}
\]  \hspace{1cm} (32)

in terms of the equilibrium value of the forces of infection \( \lambda_{D}^{**} \) and \( \lambda_{N}^{**} \) When \( \mathcal{E}^0 \) is global stable (cf. Theorem 3) then \( \mathcal{E}_3 \) is unique. Using the Centre
Manifold theory to establish the local asymptotic stability of $E_3$, we make the following change of variables $S_D = x_1, S_N = x_2, I_D = x_3, I_N = x_4, A_D = x_5$, and $A_N = x_6$, so that $N = \sum_{n=1}^{\infty} x_n$. We now use the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6)^T$. Then, model system (3) can be written in the form $\frac{dX}{dt} = F = (f_1, f_2, f_3, f_4, f_5, f_6)^T$, such that

\[ x'_1(t) = f_1 = \pi_0 \Lambda - \frac{\beta c(\eta x_3 + x_4 + \theta x_5)}{\sum_{n=1}^{\infty} x_n} x_1 - \mu x_1, \]

\[ x'_2(t) = f_2 = \pi_1 \Lambda - \frac{\beta c(x_3 + x_4)}{\sum_{n=1}^{\infty} x_n} x_2 - \mu x_2, \]

\[ x'_3(t) = f_3 = \frac{\beta c(\eta x_3 + x_4 + \theta x_5)}{\sum_{n=1}^{\infty} x_n} x_1 - (\gamma + \mu) x_3, \]

\[ x'_4(t) = f_4 = \frac{\beta c(x_3 + x_4)}{\sum_{n=1}^{\infty} x_n} x_2 - (\gamma + \mu) x_4, \]

\[ x'_5(t) = f_5 = \gamma x_3 - (\phi + \mu + \nu) x_5, \]

\[ x'_6(t) = f_6 = \gamma x_4 + \phi x_5 - (\mu + \nu) x_6. \]

The Jacobian matrix of the system (33) is given by

\[
J(E^0) = \begin{bmatrix}
-\mu & 0 & -\eta \beta c \pi_0 & -\beta c \pi_0 & -\theta \beta c \pi_0 & 0 \\
0 & -\mu & -\beta c \pi_1 & -\beta c \pi_1 & 0 & 0 \\
0 & 0 & \eta \beta c \pi_0 - (\gamma + \mu) & \beta c \pi_0 & \theta \beta c \pi_0 & 0 \\
0 & 0 & \beta c \pi_1 & \beta c \pi_1 - (\gamma + \mu) & 0 & 0 \\
0 & 0 & \gamma & 0 & -\phi - \mu + \nu & 0 \\
0 & 0 & 0 & \gamma & \phi & -(\mu + \nu)
\end{bmatrix}
\]  

(34)

From (34), it can be shown that the reproduction number in the presence of drug misuse is given by (9). If $\beta$ is taken as a bifurcation parameter and if we consider the case $R_{DN} = 1$ and solve for $\beta$ gives

\[
\beta = \beta^*_3 = \frac{2h_1^2 h_3}{c \left( \gamma_1 h_3 + (\gamma + \eta_3) h_1 \pi_0 + (\gamma + \eta_3) h_1 \pi_0 + \sqrt{(\gamma_1 h_3 + (\gamma + \eta_3) h_1 \pi_0)^2 - 4 \pi_0 \pi_1 h_4^2 h_3 (\gamma + (\eta - 1) h_3)} \right)},
\]

(35)

where $h_1 = \gamma + \mu$. Then, the linearized system of the transformed equation (33) with $\beta = \beta^*_3$ has a simple zero eigenvalue. Hence, the centre manifold theory can be used to analyze the dynamics of (33) near $\beta = \beta^*_3$ [4]. It can be
shown that the Jacobian of (33) at $\beta = \beta_3^*$ has a right eigenvector associated with the zero eigenvalue given by $m = (m_1, m_2, m_3, m_4, m_5, m_6)^T$, where

$$m_1 = -\beta_3^* c \pi_0 ((\beta_3^* c \pi_1 - \gamma - \mu)(\gamma \theta + (\phi + \mu + \nu) \eta) + (\phi + \mu + \nu) \beta_3^* c \pi_1) m_3,$$

$$m_2 = \frac{(2 \beta_3^* c \pi_1 - \gamma - \mu) \beta_3^* c \pi_1 m_3}{\mu (\beta_3^* c \pi_1 - \gamma - \mu)}, \quad m_3 = m_3 > 0, \quad m_4 = \frac{\beta_3^* c \pi_1 m_3}{\beta_3^* c \pi_1 - \gamma - \mu};$$

$$m_5 = \frac{\gamma m_2}{\phi + \mu + \nu}, \quad m_6 = \frac{((\beta_3^* c \pi_1 - \gamma - \mu)(\phi + \mu + \nu) \beta_3^* c \pi_1) \gamma m_3}{(\mu + \nu) (\phi + \mu + \nu) (\beta_3^* c \pi_1 - \gamma - \mu)}.$$

(36)

The left eigenvector of $J(\mathcal{E}^0)$ associated with the zero eigenvalue at $\beta = \beta_3^*$ is given by

$$n = (n_1, n_2, n_3, n_4, n_5, n_6)^T,$$

where

$$n_1 = n_2 = n_6 = 0, \quad n_3 = n_3 > 0, \quad n_4 = \frac{\beta_3^* c \pi_0 n_3}{\beta_3^* c \pi_1 - \gamma - \mu}, \quad n_5 = \frac{\theta \beta_3^* c \pi_0 n_3}{\phi + \mu + \nu}.$$  

(37)

The non zero partial derivatives of $F$ associated with $b$ are

$$\frac{\partial^2 f_3}{\partial x_3 \partial \beta_3^*} = c \eta \pi_0, \quad \frac{\partial^2 f_4}{\partial x_4 \partial \beta_3^*} = c \pi_0, \quad \frac{\partial^2 f_3}{\partial x_3 \partial \beta_3^*} = c \theta \pi_0, \quad \frac{\partial^2 f_4}{\partial x_4 \partial \beta_3^*} = \frac{\partial^2 f_4}{\partial x_4 \partial \beta_3^*} = c \pi_1.$$ 

(38)

It follows from (38) that,

$$b = \frac{(2 \beta_3^* c \pi_1 - \gamma - \mu) \beta_3^* c^2 \pi_0 \pi_1 m_3 m_3}{(\beta_3^* c \pi_1 - \gamma - \mu)^2} + \frac{((\beta_3^* c \pi_1 - \gamma - \mu)(\phi + \mu + \nu) + \theta \gamma + \beta_3^* c \pi_1 (\phi + \mu + \nu)) c \pi_0 m_3 n_3}{(\mu + \nu) (\beta_3^* c \pi_1 - \gamma - \mu)}.$$ 

(39)

It is immediate that $b > 0$ for $\beta_3^* c \pi_1 > \gamma + \mu$ and $b < 0$ for some $\beta_3^* c \pi_1 < \gamma + \mu$. This sign of $b$ in general is to be expected for such models because, in essence, using $\beta$ for a bifurcation parameter ensures $b > 0$. Following the same computation approach as in the previous section, the bifurcation coefficient $a$ is given as

$$a = \frac{2 \beta_3^* c \pi_0}{\Lambda} \left[ m_3 m_3 (m_3 - n_4) + m_3 m_4 (m_3 - n_4) + \eta (m_3 m_5 + \eta m_3^2 + (1 + \eta) m_3 m_4) \right]$$

$$- \frac{2 \beta_3^* c \pi_0}{\Lambda} \left[(1 + \eta) m_4 m_5 + (\theta + \eta) m_3 m_5 + \eta m_3 m_6 + m_4 (m_4 + m_6) + \eta m_5 (m_5 + m_6) \right]$$

$$- \frac{2 \beta_3^* c \pi_0}{\Lambda} \left[ m_1 m_3 (m_4 - n_3) + n_4 (m_3^2 + (m_3 + m_4) (m_4 + m_5 + m_6)) - (m_1 m_4 + \eta m_1 m_5) m_3 \right].$$ 

(40)
Thus, \( a < 0 \) for \( \beta_3^e \pi_1 > \gamma + \mu \). Hence, \( a < 0 \) and \( b > 0 \) for \( \beta_3^e \pi_1 > \gamma + \mu \) and using Theorem 4.1 [6], we establish the following result.

**Theorem 7.** If \( \beta_3^e \pi_1 > \gamma + \mu \) the endemic equilibrium \( \mathcal{E}_3 \) is locally asymptotically stable for \( R_{DN} > 1 \).

Since \( \mathcal{E}_3 \) is unique and \( \mathcal{E}^f \) is globally asymptotically stable (which excludes any possibility of the phenomenon of backward bifurcation occurring), then there can only be a a forward or transcritical bifurcation at \( R_{DN} = 1 \). Thus, we expect \( \mathcal{E}_3 \) to be globally asymptotically stable (by using an average Lyapunov function Theorem and some dynamical system theory [23]).

The model exhibits the phenomenon of backward bifurcation if the parameter \( a \) is positive, since \( b \) is automatically positive because using \( \beta \) as the bifurcation parameter ensures this. We note however that \( \frac{\partial a}{\partial \theta} = \frac{2\beta_3^e c \mu}{\Lambda} \mathcal{H} \), Where \( \mathcal{H} = \pi_1 m_3 \left( m_1 n_3 - \frac{\beta_1^e m_2}{\mu} (\eta m_3 - m_4) \right) - \pi_0 n_3 [m_3 (m_2 + m_3 + m_5 + m_6) + m_4 (m_3 + m_5)] \). Using the model parameters, we find numerically that \( \mathcal{H} < 0 \). Therefore the model system (3) cannot undergo backward bifurcation (as \( \beta_3^e \pi_1 > \gamma + \mu \) which then implies that \( a < 0 \)).

In order to quantify by how much this affects initial disease transmission, a sensitivity analysis is carried out next.

### 3.3. Sensitivity analysis

Here, we wish to investigate the relative importance of the different factors responsible for initial disease transmission, which is directly related to the magnitude of \( R_{DN} \). We follow the approach in [7]. Using the parameter values in Table 1, the sensitivity index of \( R_{DN} = 3.856 \) with respect to the effect

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recruitment rate</td>
<td>( \Lambda )</td>
<td>0.029 yr(^{-1} )</td>
<td>[2]</td>
</tr>
<tr>
<td>Natural mortality rate</td>
<td>( \mu )</td>
<td>0.02 yr(^{-1} )</td>
<td>[2]</td>
</tr>
<tr>
<td>Proportion recruited into drug misusers</td>
<td>( \pi_0 )</td>
<td>0.5</td>
<td>Assumed</td>
</tr>
<tr>
<td>Natural rate of progression to AIDS</td>
<td>( \gamma )</td>
<td>0.1 yr(^{-1} )</td>
<td>[2]</td>
</tr>
<tr>
<td>Modification parameter</td>
<td>( \eta )</td>
<td>1.02 yr(^{-1} )</td>
<td>Assumed</td>
</tr>
<tr>
<td>AIDS related death rate</td>
<td>( \nu )</td>
<td>0.4 yr(^{-1} )</td>
<td>[2]</td>
</tr>
<tr>
<td>Product of effective contact rate for HIV infection and probability of HIV transmission per contact</td>
<td>( \beta c )</td>
<td>0.011-0.05 yr(^{-1} )</td>
<td>[22]</td>
</tr>
<tr>
<td>Modification parameter</td>
<td>( \theta )</td>
<td>0.1 yr(^{-1} )</td>
<td>Assumed</td>
</tr>
<tr>
<td>Rate of quitting drug misuse as a result of advanced AIDS</td>
<td>( \phi )</td>
<td>0.05 yr(^{-1} )</td>
<td>Assumed</td>
</tr>
</tbody>
</table>
of drug misuse which is characterized by the parameters $\eta > 1$ and $\theta > 0$ are obtained. On the other hand, the sensitivity index of $R_{DN}$ with respect to the deceleration parameter $\theta = 0.1$ (which accounts for the reduced infectiousness of drug users who are in the AIDS stage and assumed to have a reduced role in sexual activities) is 0.561. Thus, an increase of 10% in this parameter will lead to a 5.61% increase in secondary transmission (mainly due to needles sharing).

Also, the sensitivity of $\eta > 1$ (which accounts for possible increase in disease transmission by HIV positive drug users who infect more people via both needle sharing and sexual contact) with respect to $R_{DN}$ is 5.67. Therefore, a 10% increase in the parameter $\eta$ leads to a staggering 56.7% in secondary transmission driven by drug misusers.

We note that even for a value of $\eta$ as low as 1.001, we still have an increase of more than 50% (56.02%) in secondary transmission. These values inform us on the degree how worse drug misuse impact negatively on the transmission dynamics of HIV. We note however that $\eta$ is not very sensitive on its own as long as its value is slightly greater than unity. Since care should be taken in choosing parameters which are or may not directly be observable, our analysis informs us that for modification parameters assumed to be greater than unity, we should choose them as closer to one as possible in order not to overestimate by how much worse things could be. For parameters whose values are by hypothesis less than unity, we choose them as close to zero as possible, especially in the case of HIV/AIDS for which most individuals in the AIDS stage are sexually incapacitated due to the terminal form of the disease.

Here we ask: Is it possible to control the epidemic by targeting drug misusers only? To answer this question, we investigate the role of drug misuse on the initial disease transmission. $R_{0N} = 3.33 < R_{DN}$ thus drug misuse seems to impact negatively on the initial disease spread by 15.7% only. Even without drug misuse, the epidemic still spread since the basic reproduction number $R_{0N}$ is greater than unity. Also, the prevalence of drug misuse is measured by the parameter $\pi_0$, and the sensitivity of RDN to the later is 0.758, and a 10% increase in drug misuse prevalence will lead to approximately 7.58% increase in initial disease transmission. Thus, drug misuse has a negative impact on HIV transmission in the community and consequently contributes in driving the epidemic. In particular, a 60% reduction of in°ow of drug misusers gives a sensitivity index of 0.344. That is, for $\pi_0 = 0.2$ we achieve a 31.1% net reduction in initial disease transmission (in this case, $R_{DN} = 2.03$). Therefore, it is possible to slightly control the epidemic by targeting drug misusers only, even though the infection still spreads at a reduced rate without drug misuse.
4. Numerical simulations

We carry out numerical simulations to show the effects of drug misuse in the transmission dynamics of HIV/AIDS. The parameter values used for numerical simulations are in Table 1, some from the literature and others assumed for the purpose of illustration. The fourth order Runge-Kutta numerical scheme coded in C++ programming language is used for the numerical simulations of model system (3).

Figure 2 is a graphical representation showing the effect varying the contribution $\gamma_i$ of HIV positive drug misusers who are not yet showing symptoms of AIDS. Figure 2 (a) shows that the population of susceptible drug misusers is depleted more thoroughly than the population of susceptible non-drug users because of the higher infection rates in the former group. Interestingly, the value

![Figure 2](image_url)

**Figure 2.** Simulations of model (3) showing plots of drug misusers and non-drug misusers in relation to HIV infection as a function of time with varying $\gamma_i$. (a) susceptible population. (b) HIV positive population without AIDS. (c) AIDS patients. The modification parameter starts from $\gamma_i = 1$ with step size 0.5. Parameter values used are as in Table 1.
of $r_f$ has a significant effect on how many drug users are infected, but it also has some impact on how many susceptible non-drug users are infected, as well as a strong impact on the time evolution of the outbreak in both drug users and non-drug users. Similarly, Figure 2(b) shows that the peak in infected persons is reached more quickly for drug users than for non-drug users. The equilibrium number of infected drug users is lower than the equilibrium number of infected non-drug users because of the term $\phi$ in the equations that represents infected drug users leaving the drug-using population. Finally, Figure 2(c) indicates that AIDS cases peak sooner in the drug using population than in the non-drug using population. Again, the equilibrium number of drug users with AIDS is lower than the equilibrium number of non-drug users with AIDS because of the $\phi$ term in the equations. Notably, the value of $r_f$ does not have a significant impact on the equilibrium number of AIDS cases in either group, although it does have a significant impact on the time evolution.

We evaluate the effect of varying the contribution $\theta$ of drug misusers who are in the AIDS stage in Figure 3. The results shown in Figure 3 follow a trend

![Figure 3](image-url)

**Figure 3.** Simulations of model (3) showing plots of drug misusers and non-drug misusers in relation to HIV infection as a function of time with varying $\mu$. (a) susceptible population. (b) HIV positive population without AIDS. (c) AIDS patients. The modification parameter starts from $\mu = 0$ with step size 0.25. Parameters values used are as in Table 1.
similar to those in Figure 2. This suggests that drug misuse contributes to the increase of HIV/AIDS pandemic, although the effect of varying $\theta$ is not as pronounced as the effect of varying.

5. Conclusion

A mathematical model for the transmission dynamics of HIV in the context of drug misuse (with two sub-populations: drug abusing individuals and non-drug users) is formulated and its mathematical properties investigated in order to assess the impact of drug misuse on the transmission dynamics of HIV/AIDS has been proposed and investigated. The disease-free equilibrium is shown to be globally asymptotically stable when the corresponding reproduction number is less than unity. Using the Centre Manifold theory, each equilibrium point was shown to be locally asymptotically when its corresponding reproduction number is greater than unity. A sensitivity of the reproduction number to the level of drug misuse is carried out showing that drug misuse has the potential to increase disease transmission through the basic reproduction number $R_{0N}$. Thus, we argue that controlling drug misuse can contribute in curtailling (reducing the spread of) the epidemic, as drug misuse has the potential to fuel the HIV/AIDS pandemic and should be given prominence in any HIV control programme.

Results from the numerical simulations suggest that drug misuse has a significant impact on HIV prevalence in both drug using populations and non-drug using populations, under the assumption that the two groups are mixing with one another. The stability of these equilibria has clear public health consequences as there exists the potential for the disease to persist in the population. Also, HIV prevalence and AIDS cases grow more rapidly in drug using populations than in non-drug using populations. This implies that efforts to curtail drug misuse could help reduce HIV incidence in both drug user and non-drug user populations. Strategies to reduce/curtail drug misuse should carefully be considered in communities where drug misuse is growing and where there is potential for further HIV transmission. In this respect, mass educational campaigns can prove very beneficial [29, 31].

This study is not exhaustive and can be extended in various ways. For instance, since the factor $\gamma$ is due to increased sexual activity, $N$ can be the total contacts and $\gamma$ can then be incorporated into $N$ as per the work of Nold on contact rates and mixing ([34] and the references therein).
References


33. NIDA InfoFacts: Drug misuse and the link to HIV/AIDS and Other Infectious Diseases, 1-4.


