Is the world doing enough for the poor? A case of HIV/AIDS testing and counselling


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Abstract. A HIV/AIDS model with testing and counselling in resource limited settings is proposed and investigated. The disease threshold quantities are determined and stability of the various equilibria analysed. Of public health concern, theoretical analysis of the reproduction number suggests that HIV positive individuals who know their status and have not disclosed it have a great potential to fuel the epidemic. Numerical simulations are also carried out and we observe that in the end there will be more people who know their HIV status than those who do not know. Further, this suggests the future of the epidemic will be driven by people who know their status and have not disclosed it. It therefore seems that more needs to be done in terms of counselling in resource-limited settings as far as the HIV/AIDS epidemic is concerned.

Keywords: HIV/AIDS, reproduction number, resource-limited settings, testing and counselling

1 Introduction

The HIV/AIDS epidemic has now attained epidemic proportion and can be considered a global pandemic, it is by any account overwhelmingly an African one. Sub-Saharan Africa is home to 24 of the 25 countries with the world’s highest HIV levels. At the close of 2001, an estimated 40 million people worldwide-37.2 million adults and 2.7 million children younger than 15 years were living with HIV/AIDS, and more than 70% (28.1 million) of these people live in sub-Saharan Africa; another 15 percent (6.1 million) live in South and Southeast Asia (www.rapeoutcry.co.za/resources/hiv.doc). South Africa, a country with the highest incidence of rape and child rape in the world, has 19.94% or approximately one in nine of its 41 million inhabitants with HIV/AIDS, as opposed to one in five for Botswana (35.8%), and 25.25% and 25.06% for Swaziland and Zimbabwe, respectively (www.rapeoutcry.co.za/resources/hiv.doc). Infection rates are stabilizing in parts of the continent, but they are on the rise in most of the south[29].

In Africa, HIV is transmitted mostly through sexual intercourse. Currently, despite the availability of voluntary counselling and testing in most countries in sub-Saharan Africa, the HIV/AIDS epidemic continues to grow. This brings us to the following question. Is HIV/AIDS testing and counselling enough to control the spread of the epidemic? There is an urgent need to address some cultural practices and norms in Africa. In many sub-Saharan Africa countries, myths like sleeping with virgin would cure HIV/AIDS led to an increase in the number of rape cases by people who know their HIV positive status[1, 6, 16]. Virgin cleansing is a myth that has occurred since at least the sixteenth century, when Europeans believed that they could rid themselves of a sexually transmitted disease (syphilis and gonorrhea) by transferring it to a virgin through sexual intercourse[15]. Although the exact prevalence of this belief is unclear, it is believed to occur worldwide.

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However, the majority of modern reported cases of this event have occurred in sub-Saharan Africa\cite{15, 20}. Sex with an uninfected virgin or a disable person does not cure a person infected with any disease. Such contacts expose the uninfected individual to HIV and other sexually transmitted disease, and as such may contribute in spreading the disease in physically vulnerable individuals because they are easy targets\cite{15}. It has been suggested that infant rape and virgin cleansing may capture some of the dynamics of HIV/AIDS crisis\cite{18}. The impact of these will be worth a separate investigation in a future study. The “virgin cleansing/rape” myth\cite{17} has gained considerable notoriety as the perceived reason for certain sexual abuse and child molestation occurrences, especially in Africa\cite{13, 15}. In this case, knowing one’s status seems to suggest a voluntary spread of the infection. In order to gainfully benefit from testing and counselling, there is a strong need for status disclosure. Public health messages have traditionally urged disclosure to all sexual and drug using partners. In reality, some HIV positive persons may choose not to disclose due to fears of rejection or harm, feelings of shame, desires to maintain secrecy, feelings that with safer sex there is no need for disclosure, fatalism, perceived community norms against disclosure, and beliefs that individuals are responsible for protecting themselves\cite{30}. The relationship between disclosure, sexual risk behaviors and potential transmission of HIV varies. Research findings have presented a mixed picture\cite{25}. Some studies have found that increased disclosure is associated with reduced sexual risk behavior\cite{23}. Other studies show that disclosure does not always alter risk taking behaviors\cite{10}. Even with disclosure, unsafe sex sometimes occurs. Some people engage in safer sex behaviors without any discussion of HIV status\cite{19}.

It is well-known that drug abuse can disproportionately exacerbate HIV transmission\cite{28}, and therefore efforts to mitigate drug abuse may likewise help stem the HIV/AIDS epidemic. Also, poverty and gender inequality are so manifestly related to HIV transmission and outcomes. How do we address these issues in HIV/AIDS prevention and care? The best framework for analysis and evaluation would need to be robustly grounded in a broad biosocial understanding, since the phenomena it attempts to describe are nothing if not both biological and social\cite{27}. Poverty reduction guarantees food security and can contribute in reducing the spread of HIV/AIDS (the effects of poverty and drug abuse taken one at a time are being addressed in subsequent studies). But, much still needs to be done especially in poor resource settings\cite{12} as poverty has a multiplier effect on nutrition. In many societies, gender norms and gender dynamics influence people’s attitudes to sex, sexuality, risk taking and fidelity. Thus, gender-inequality plays an important role in the transmission dynamics of HIV/AIDS\cite{21}. For these reasons, HIV/AIDS is inherently a poverty- drug abuse- and gender-based issue and needs to be seen in this light if it is to be addressed effectively. All these factors can easily be addressed if policies are tailored to reduce or eradicate drug abuse, poverty and gender inequality. Besides, one major factor which to the best of our knowledge has never been taken into consideration in HIV modelling is the social classification of infected individuals into those who know their status and those who do not. Prevention effectiveness should be tailored to address HIV/AIDS in communities taking into account their cultural beliefs. This ideal and innovative way of addressing the spread of the epidemic will likely be more welcoming as it will have relevance and potential contribution to the development of the community as it attempts to educate individuals on the downfalls of superstition. This is challenging, but not impossible to convince individuals by using the umbrella of poverty, gender inequality and drug abuse in order to address the social myths. HIV/AIDS models are numerous in the literature \cite{3–5}. For a comprehensive review, see \cite{14, 22}, although this may be the first model that attempts to look into classifying infective individuals into those who know and those who do not know their status in the presence of myths and absence of antiretroviral therapy, a possible scenario in some communities in the developing world.

The rest of this paper is organized as follows. In the next section, the model and its basic properties (boundedness and positivity) are presented. In Section 3, we determine sufficient conditions for the local and global stability of the disease-free as well as of the local stability of the endemic equilibrium. Section 4 numerically assesses the long-term driver (status awareness) of the HIV/AIDS. The last Section concludes the paper.

2 Model description

We wish to investigate a question of public health concern, namely: why despite years of testing and

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counselling is HIV/AIDS still spreading in poverty striken communities (like a wild fire on a windy day)? In order to address this seemingly naive question, the total population is divided into four compartments according to individuals disease status: susceptibles \( S(t) \), HIV positive individuals who do not know their status \( I_1(t) \), HIV positive individuals aware of their status \( I_2(t) \) and the AIDS patients \( A(t) \). Thus, the total population is given by:

\[
N(t) = S(t) + I_1(t) + I_2(t) + A(t).
\]

(1)

At any time, new recruits enter the human population through birth or immigration at constant rate \( \Lambda \). There is a constant natural death rate \( \mu \) in each subclass. The force of infection associated with HIV infection, denoted by \( \lambda \), is given by

\[
\lambda(t) = \frac{\beta c(I_1(t) + \eta I_2(t) + \theta A(t))}{N(t)},
\]

(2)

with \( \beta \) being the probability of HIV transmission per sexual contact; \( c \) is the effective contact rate for HIV infection to occur; \( \eta > 1 \) models the fact that HIV positive individuals who know their status spread the disease more than those who do not know their status as they are afraid of dying alone as well as believing in myths like sleeping with a virgin will cure your AIDS\(^{15,20} \); \( \theta \in (0, 1) \) models the fact that AIDS patients are not as sexually active as healthy individuals, and that given their ill health they do not attract many sexual partners. In this case we assume an individual who do not know his/her status will take precautions to minimize chances of getting infected. Susceptibles are infected with HIV at a rate \( \lambda \) and a proportion \( p \) will know their status through testin and move to the class of HIV positive individuals who know their status \( I_2(t) \). The complementary proportion \( (1 - p) \) will move into \( I_1(t) \). Individuals in both HIV positive classes progress to the AIDS stage at a rate \( \rho \). Of the individuals in \( I_1(t) \), they will know their status a rate \( \delta \) and move into \( I_2(t) \). Individuals in \( A(t) \) have an additional disease induced death rate \( \nu \). The structure of the model system (3) is given in Fig. 1.

\[
\begin{align*}
S'(t) &= \Lambda - (\lambda + \mu)S, \\
I_1'(t) &= (1 - p)\lambda S - (\mu + \rho + \delta)I_1, \\
I_2'(t) &= p\lambda S + \delta I_1 - (\mu + \rho)I_2, \\
A'(t) &= \rho(I_1 + I_2) - (\mu + \nu)A.
\end{align*}
\]

(3)
The model has initial conditions given by
\[ S(0) = S_0 \geq 0, \ I_1(0) = I_{10} \geq 0, \ I_2(0) = I_{20} \geq 0, \ A(0) = A_0 \geq 0. \]  
(4)

Based on biological considerations, the proposed model system (3) will be studied in the following region
\[ \mathcal{G} = \left\{ (S, I_1, I_2, A) \in \mathbb{R}_+^4 : N(t) \leq \frac{A}{\mu} \right\}, \]
(5)
which is positively invariant and attracting with respect to model system (3).

\section{Positivity and boundedness of solutions}

Model system (3) describes human population and therefore it is necessary to prove that all the variables \( S(t), \ I_1(t), \ I_2(t), \ A(t) \) are non-negative for all time. Solutions of the model system (3) with positive initial data remains positive for all time \( t \geq 0 \) and are bounded in \( \mathcal{G} \).

\textbf{Theorem 1.} Let \( S(t) \geq 0, \ I_1(t) \geq 0, \ I_2(t) \geq 0, \ A(t) \geq 0 \). The solutions \( S, \ I_1, \ I_2, \ A \) of model system (3) are positive for \( t \geq 0 \). For the model system (3), the region \( \mathcal{G} \) is positively invariant and all solutions starting in \( \mathcal{G} \) approach, enter, or stay in \( \mathcal{G} \).

\textbf{Proof.} Under the given initial conditions, it is easy to prove that each solution component of model system (3) is positive, otherwise by contradiction that there exists a first time
\[ t_1 : S(t_1) = 0, \ S'(t_1) < 0 \] and \( S(t) > 0 \), \( I_1(t) > 0 \), \( I_2(t) > 0 \), \( A(t) > 0 \) for \( 0 < t < t_1 \) or there exists a \( t_2 : I_1(t_2) = 0, \ I_1'(t_2) < 0 \) and \( S(t) > 0 \), \( I_1(t) > 0 \), \( I_2(t) > 0 \), \( A(t) > 0 \) for \( 0 < t < t_2 \) or there exists a \( t_3 : I_2(t_3) = 0, \ I_2'(t_3) < 0 \) and \( S(t) > 0 \), \( I_1(t) > 0 \), \( I_2(t) > 0 \), \( A(t) > 0 \) for \( 0 < t < t_3 \) or there exists a \( t_4 : A(t_4) = 0, \ A'(t_4) < 0 \) and \( S(t) > 0 \), \( I_1(t) > 0 \), \( I_2(t) > 0 \), \( A(t) > 0 \) for \( 0 < t < t_4 \).

In the first case we have,
\[ S'(t_1) = A > 0, \]
(6)
which is a contradiction and consequently \( S \) remains positive. In the second case we have,
\[ I_1'(t_2) = (1 - p)S(t_2)\lambda(t_2) > 0, \]
(7)
which is a contradiction, therefore \( I_1 \) is positive. In the third case we have,
\[ I_2'(t_3) = pS(t_3)\lambda(t_3) + \delta I_1(t_3) > 0, \]
(8)
which is also a contradiction, so \( I_T \) remains positive. In the final case we have,
\[ A'(t_4) = \rho(I_1(t_4) + I_2(t_4)) > 0, \]
(9)
which is a contradiction, that is, \( A \) remains positive. Thus, in all cases \( S, \ I_1, \ I_2, \ A \) remain positive. Since \( N(t) \geq A(t) \), then
\[ A - (\mu + \nu)N \leq N'(t) \leq A - \mu N \]
(10)
implies that \( N(t) \) is bounded and all solutions starting in \( \mathcal{G} \) approach, enter or stay in \( \mathcal{G} \).

\section{Model analysis}

In this section, we focus on the stability of the equilibria of system (3) and we begin by considering the disease-free equilibrium.

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3.1 Disease-free equilibrium and stability analysis

The disease-free equilibrium of model system (3) is given by

$$\mathcal{U}_0 = (S_0, I_1, I_2, A_0) = \left( \frac{\Lambda}{\mu}, 0, 0, 0 \right).$$

(11)

3.1.1 $\mathcal{R}_T$ and local stability of $\mathcal{U}_0$

The basic reproduction number is defined as the number of secondary infections generated by a single infectious individual during his or her entire infectious period when introduced into a completely naive population\[^{[11]}\]. Herein, the disease threshold quantity, $\mathcal{R}_T$ is defined as the number of new HIV infections produced by a single HIV/AIDS case in a population in which some people know their HIV status. Following the approach of \[^{[11]}\], we have

$$\mathcal{R}_T = \frac{\beta c \{(\mu + \rho)[(\mu + \nu)(1 - p + \eta p) + \theta p] + \delta(\mu + \nu + \theta p)\}}{\mu + \nu}(\mu + \rho)(\mu + \delta + \rho).$$

(14)

Theorem 2 follows from Theorem 2 in \[^{[11]}\].

**Theorem 2.** The disease-free equilibrium $\mathcal{U}_0$ is locally asymptotically stable when $\mathcal{R}_T < 1$, and unstable otherwise.

Following \[^{[8]}\], we now list two conditions that if met, also guarantee the global asymptotic stability of the disease-free equilibrium. Rewriting model system (3) as

$$\frac{dX}{dt} = F(X, Z), \quad \frac{dZ}{dt} = G(X, Z), \quad G(X, 0) = 0,$$

(15)

where $X = (S)$ and $Z = (I_1, I_2, A)$, with $X \in \mathbb{R}_+$ denoting (its components) the number of uninfected individuals and $Z \in \mathbb{R}_+^3$ denoting (its components) the number of infected individuals including both the HIV positive only and the AIDS cases. The disease-free equilibrium is now denoted by,

$$\mathcal{U}_0 = (X^*, 0), \text{ where } X^* = \frac{\Lambda}{\mu}. $$

(16)

The conditions (H1) and (H2) in Eq. (17) must be met to guarantee local asymptotic stability.

$$H1: \text{ For } \frac{dX}{dt} = F(X, 0), \text{ } X^* \text{ is globally asymptotic stable (g.a.s)}$

$$H2: \text{ } G(X, Z) = BZ - \tilde{G}(X, Z), \quad \tilde{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \mathcal{S},$$

(17)

where, $B = D_Z G(X^*, 0)$ is an $M$-matrix (the off diagonal elements of $B$ are nonnegative) and $\mathcal{S}$ is the region where the model makes biological sense. If system (15) satisfies the conditions in (17), then, Theorem 3 holds.

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Theorem 3. The fixed point $U_0 = (X^*, 0)$ is a global asymptotic stable equilibrium of system (15) provided that $R_T < 1$ and assumptions in (17) are satisfied.

Proof. From Theorem 2, $U_0$ is locally asymptotically stable if $R_T < 1$. Consider

$$F(X, 0) = \begin{bmatrix} A - \mu S \end{bmatrix},$$

$$G(X, Z) = BZ - \hat{G}(X, Z), \quad B = \begin{bmatrix} (1 - p)\beta c - (\mu + \rho + \delta) & (1 - p)\eta \beta c & (1 - p)\theta \beta c \\ \rho \beta c + \delta & \rho \eta \beta c - (\mu + \rho) & \rho \theta \beta c \\ \rho & \rho & -(\mu + \nu) \end{bmatrix}.$$  (19)

Then,

$$\hat{G}(X, Z) = \begin{bmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \\ \hat{G}_3(X, Z) \end{bmatrix} = \begin{bmatrix} (1 - p)\beta c(I_1 + \eta I_2 + \theta A)(1 - \frac{S}{N}) \\ p\beta c(I_1 + \eta I_2 + \theta A)(1 - \frac{S}{N}) \\ 0 \end{bmatrix}.$$  (20)

Thus, $\hat{G}(X, Z) \geq 0$ implying that $U_0$ is globally asymptotically stable for $R_T < 1$.

### 3.1.2 Analysis of the reproduction number ($R_T$)

We now theoretically assess the effects that individuals unaware of their status as well as all individuals who know their status have on the number of HIV/AIDS cases.

**Case 1.** All people do not know their status

In this case $\delta = 0$ and $\eta = 1$ such that

$$\lim_{(\delta, \eta) \rightarrow (0, 1)} R_T = R_0 = \frac{\beta c(\mu + \nu + \theta \rho)}{(\mu + \nu)(\mu + \rho)},$$  (21)

which is known as the HIV/AIDS basic reproduction number. Expressing $R_0$ in terms of $R_T$ we obtain

$$R_0 = \frac{(\mu + \rho)((\mu + \nu)(1 - p + \eta \rho) + \theta \rho) + \delta(\eta(\mu + \nu) + \theta \rho)}{\mu + \nu + \theta \rho} R_T,$$  (22)

from which,

$$\frac{\partial R_0}{\partial R_T} = \frac{(\mu + \rho)((\mu + \nu)(1 - p + \eta \rho) + \theta \rho) + \delta(\eta(\mu + \nu) + \theta \rho)}{\mu + \nu + \theta \rho} > 0.$$  (23)

This suggests that if a proportion of people know their status, there is a potential that such people are intentionally spreading the virus[24]. From this observation, there is a strong need for HIV/AIDS testing and counselling (on status disclosure) to be accompanied by provision of antiretroviral drugs and public health campaigns tailored to address specific issues pertaining to each community.

**Case 2.** All people know their status

If all people know their status then $p = 1$ and $\delta = 0$. In this case $R_T$ becomes $R_{0T}$ which is given as

$$R_{0T} = \frac{\beta c(\eta(\mu + \nu) + \theta \rho)}{(\mu + \nu)(\mu + \rho)} = \frac{\mu + \nu + \theta \rho}{\eta(\mu + \nu) + \theta \rho} R_0,$$  (24)

from which it can be shown that

$$R_0 = \frac{\eta(\mu + \nu) + \theta \rho}{\mu + \nu + \theta \rho} R_{0T} \Rightarrow \frac{\partial R_0}{\partial R_{0T}} = \frac{\eta(\mu + \nu) + \theta \rho}{\mu + \nu + \theta \rho} > 0.$$  (25)

That is, increasing $R_{0T}$ results in an increase of the basic reproduction number $R_0$ suggesting that if more people know their status, in the absence of antiretroviral therapy they are likely to spread the infection intentionally. Most people who know that they are HIV positive may indulge in more risky sexual behaviour which will in turn contribute to the epidemic spread.

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3.2 Endemic equilibrium and stability analysis

In terms of the force of infection, the endemic equilibrium \( \mathcal{U}^* \) is given by

\[
\mathcal{U}^* = (S^*, I_1^*, I_2^*, A^*)
\]

where \( S^* = \frac{A}{\mu + \lambda^*}, I_1^* = \frac{(1-p)\Lambda^*}{\mu + \rho + \delta}, I_2^* = \frac{\Lambda^*\rho}{\mu + \rho + \delta}, A^* = \frac{\rho\Lambda^*}{\mu + \rho + \delta}. \)

We now employ the Centre Manifold theory\(^7\) to analyse the stability of this equilibrium point as described in Theorem 4.1\(^6\), to establish the local asymptotic stability of the endemic equilibrium. Let us make the following change of variables in order to apply the Center Manifold theory \( S = x_1, I_1 = x_2, I_2 = x_3 \) and \( A = x_4 \), so that \( N = \sum_{n=1}^{4} x_n \). We now use the vector notation \( X = (x_1, x_2, x_3, x_4)^T \). Then, model system (3) can be written in the form \( \frac{dX}{dt} = F = (f_1, f_2, f_3, f_4)^T \), such that

\[
\begin{align*}
x_1'(t) &= f_1 = \lambda - \frac{\beta c(x_2 + \eta x_3 + \theta x_4)}{\sum_{n=1}^{4} x_n} x_1 - \mu x_1, \\
\sum_{n=1}^{4} x_n &= \lambda = \mu + \lambda, \\
\sum_{n=1}^{4} x_n &= \lambda = \mu + \lambda, \\
f_1 &= \frac{\beta c(x_2 + \eta x_3 + \theta x_4)}{\sum_{n=1}^{4} x_n} x_1 - (\mu + \rho) x_2, \\
f_3 &= \frac{\rho \beta c(x_2 + \eta x_3 + \theta x_4)}{\sum_{n=1}^{4} x_n} x_1 + \delta x_2 - (\mu + \rho) x_3, \\
f_4 &= \frac{\rho (x_2 + x_3) - (\mu + \nu) x_4.}
\end{align*}
\]

The Jacobian matrix of system (27) at \( \mathcal{U}_0 \) is given by

\[
J(\mathcal{U}_0) = 
\begin{bmatrix}
-\mu & -\beta c & -\eta \beta c & -\theta \beta c \\
0 & (1-p)\beta c - (\delta + \rho + \mu) & (1-p)\eta \beta c & (1-p)\theta \beta c \\
0 & \rho \beta c + \delta & \rho \eta \beta c - (\mu + \rho) & \rho \theta \beta c \\
0 & \rho & -\rho (\mu + \nu)
\end{bmatrix},
\]

from which it can be shown that the reproduction number is given by Eq. (14). If \( \beta \) is taken as a bifurcation point, consider the case \( R_T = 1 \) and solve for \( \beta \), we obtain

\[
\beta = \beta^* = \frac{(\mu + \nu)(\mu + \rho)(\mu + \delta + \rho)}{c\{(\mu + \rho)[(\mu + \nu)(1 - \rho + \eta) + \theta \rho] + \delta (\eta (\mu + \nu) + \theta \rho)\}}.
\]

Note that the linearised system of the transformed Eq. (27) with \( \beta = \beta^* \), has a simple zero eigenvalue. Hence, the Centre Manifold theory\(^7\) can be used to analyze the dynamics of (27) near \( \beta = \beta^* \). It can be shown that the Jacobian of (27) at \( \beta = \beta^* \) has a right eigenvector associated with the zero eigenvalue given by \( u = [u_1, u_2, u_3, u_4]^T \), where

\[
\begin{align*}
u_1 &= \frac{-\beta c(u_2 + \eta u_3 + \theta u_4)}{\mu}, \\
u_2 &= \frac{\rho \theta \beta^* c - (\mu + \nu)(\rho \eta \beta^* c - \mu - \nu)}{(\mu + \nu)(\rho \eta \beta^* c + \delta) - \rho \theta \beta^* c} u_3, \\
u_3 &= u_3 > 0, \\
u_4 &= \frac{\rho(\mu + \nu)(\rho \eta \beta^* c - \mu - \nu)}{\mu + \nu} u_3.
\end{align*}
\]

The left eigenvector of \( J(\mathcal{U}_0) \) associated with the zero eigenvalue at \( \beta = \beta^* \) is given by \( v = [v_1, v_2, v_3, v_4]^T \), where

\[
\begin{align*}
v_1 &= 0, \\
v_2 &= \frac{-\rho \theta \beta^* c + (\mu + \nu)(\rho \eta \beta^* c - \mu - \rho))}{(1-p)\beta^* c} u_3, \\
v_3 &= u_3 > 0, \\
v_4 &= \frac{\theta (\mu + \nu) u_3}{\theta \rho + \eta (\mu + \nu)}.
\end{align*}
\]

Further, we use Theorem 4 from \(^6\), stated below for elucidation.

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Theorem 4. Consider the following general system of ordinary differential equation equations with a parameter $\phi$

$$\frac{dx}{dt} = f(x, \phi), \quad f : R^n \times R \rightarrow R \text{ and } f \in C^2(R^n \times R),$$

(32)

where 0 is an equilibrium of the system that is $f(0, \phi) = 0$ for all $\phi$ and assume

\textbf{A1}: $A = D_x f(0, 0) = \left( \frac{\partial f}{\partial x_j}(0, 0) \right)$ is the linearisation of system (32) around the equilibrium 0 with $\phi$ evaluated at 0. Zero is a simple eigenvalue of $A$ and other eigenvalues of $A$ have negative real parts;

\textbf{A2}: Matrix $A$ has a right eigenvector $u$ and a left eigenvector $v$ corresponding to the zero eigenvalue.

Let $f_k$ be the $k$th component of $f$ and

$$a = \sum_{k,i,j=1} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0), \quad b = \sum_{k,i=1} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0, 0).$$

(33)

The local dynamics of (32) around 0 are totally governed by $a$ and $b$.

(i) $a > 0$, $b > 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi << 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;

(ii) $a < 0$, $b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 unstable; when $0 < \phi << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;

(iii) $a > 0$, $b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi << 1$, 0 is stable, and a positive unstable equilibrium appears;

(iv) $a < 0$, $b > 0$. When $\phi$ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Computations of the bifurcation parameters $a$ and $b$:

For system (27), the associated non-zero partial derivatives of $F$ at the disease-free equilibrium are given by

$$\begin{align*}
\frac{\partial^2 f_2}{\partial x_2 \partial x_3} &= \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = \frac{(1 - p)(1 + \eta) \beta^* c\mu}{\Lambda}, & \frac{\partial^2 f_2}{\partial x_2^2} &= \frac{-2(1 - p) \beta^* c\mu}{\Lambda}, \\
\frac{\partial^2 f_2}{\partial x_2 \partial x_4} &= \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = \frac{(1 - p)(1 + \theta) \beta^* c\mu}{\Lambda}, & \frac{\partial^2 f_2}{\partial x_2^3} &= \frac{-2(1 - p) \beta^* c\mu}{\Lambda}, \\
\frac{\partial^2 f_2}{\partial x_3 \partial x_4} &= \frac{\partial^2 f_2}{\partial x_4 \partial x_3} = \frac{(1 - p) \beta^* c(\theta + \eta) \mu}{\Lambda}, & \frac{\partial^2 f_2}{\partial x_3^4} &= \frac{-2(1 - p) \beta^* c\theta \mu}{\Lambda}, \\
\frac{\partial^2 f_3}{\partial x_2 \partial x_3} &= \frac{\partial^2 f_3}{\partial x_3 \partial x_2} = \frac{p(1 + \eta) \beta^* c\mu}{\Lambda}, & \frac{\partial^2 f_3}{\partial x_2^2} &= \frac{-2p \beta^* c\mu}{\Lambda}, \\
\frac{\partial^2 f_3}{\partial x_2 \partial x_4} &= \frac{\partial^2 f_3}{\partial x_4 \partial x_2} = \frac{p(1 + \theta) \beta^* c\mu}{\Lambda}, & \frac{\partial^2 f_3}{\partial x_2^3} &= \frac{-2p \beta^* c\mu}{\Lambda}, \\
\frac{\partial^2 f_3}{\partial x_3 \partial x_4} &= \frac{\partial^2 f_3}{\partial x_4 \partial x_3} = \frac{p \beta^* c(\theta + \eta) \mu}{\Lambda}, & \frac{\partial^2 f_3}{\partial x_3^4} &= \frac{-2p \beta^* c\theta \mu}{\Lambda}.
\end{align*}$$

(34)

From (34), it follows that

$$a = -\frac{2 \beta^* c\mu(pv_3 + (1 - p)v_2)}{\Lambda} [u_2(u_2 + u_3 + u_4) + u_3(u_3 + u_4) + u_4^2] < 0.$$  

(35)

Since $a < 0$, this excludes the possibility of any backward bifurcation, consequently, local stability will suggest global stability. This conclusion also stems from the global stability of the disease-free equilibrium. For the sign of $b$, it is associated with the following non-vanishing partial derivatives of $F$.  

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\[
\begin{align*}
\frac{\partial^2 f_1}{\partial x_2 \partial \beta^*} &= -c, & \frac{\partial^2 f_1}{\partial x_3 \partial \beta^*} &= -\eta c, & \frac{\partial^2 f_1}{\partial x_4 \partial \beta^*} &= -\theta c, \\
\frac{\partial^2 f_2}{\partial x_2 \partial \beta^*} &= (1-p)c, & \frac{\partial^2 f_2}{\partial x_3 \partial \beta^*} &= (1-p)\eta c, & \frac{\partial^2 f_2}{\partial x_4 \partial \beta^*} &= (1-p)\theta c, \\
\frac{\partial^2 f_3}{\partial x_2 \partial \beta^*} &= pc, & \frac{\partial^2 f_3}{\partial x_3 \partial \beta^*} &= p\eta c, & \frac{\partial^2 f_3}{\partial x_4 \partial \beta^*} &= p\theta c.
\end{align*}
\]
\[(36)\]

It follows from the expressions in (36) that
\[
b = c(u_2 + \eta u_3 + \theta u_4)(pv_3 + (1-p)v_2) > 0.
\]
\[(37)\]

Thus, \(a < 0\) and \(b > 0\) and using Theorem 4 item \((iv)\), we have established the following result.

**Theorem 5.** The unique endemic equilibrium \(U^*\) guaranteed by Theorem 4 is locally asymptotically stable for \(R_T > 1\), but close to 1.

### 4 Numerical simulations

In this section, we make use of Matlab to analyse the effect of varying initial conditions, varying some model parameters on model system (3) using model parameters in Tab. 1. Fig. 4 is a graphical representation depicting the effects of varying initial conditions. From graphs in Fig. 4, we observe that as long as \(R_T > 1\), varying the initial conditions will not change the final result as in all cases the susceptible, HIV positive unaware of their status, HIV positive who know their status and the AIDS cases will converge asymptotically to the endemic state. This asymptotic state is reached when we have the following scenarios: \(S(t) > I_2(t) > I_1(t) > A(t)\). The results from Fig. 4 suggest that eventually, there will be more people aware of their HIV status than those who are not. This further suggests the future of the HIV/AIDS epidemic will be driven by people who know their status. Hence, the following question: Is the world doing enough for those who test HIV positive in resource limited countries? It seems more needs to be done to encourage abstinence and or positive sexual behavioural change, but without education and incentives, poverty stricken communities may not commit to such behavioural change.

Fig. 4 is a graphical representation showing the effects of varying \(\eta\) and \(\theta\) on the population. The direction of the arrow in Fig. 4 (a) and 4 (c) show a decrease in \(\eta\) and \(\theta\), respectively. Increasing \(\eta\) and \(\theta\), respectively results in a decrease of susceptibles implying more susceptible people getting infected. In Fig. 4 (b) and 4 (d) increasing \(\eta\) and \(\theta\), respectively result in an increase of the HIV positive and AIDS cases in the first years before gradually declining to asymptotic states. In both graphs (Fig. 4 (b) and 4 (d)), HIV positives who do

---

**Table 1. Model parameters and their interpretations**

<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>0.029yr(^{-1})</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Natural mortality rate</td>
<td>(\mu)</td>
<td>0.02yr(^{-1})</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Proportion aware of their status soon after infection</td>
<td>(p)</td>
<td>0.2yr(^{-1})</td>
<td>Assumed</td>
</tr>
<tr>
<td>Proportion aware of their status later</td>
<td>(\delta)</td>
<td>0.1yr(^{-1})</td>
<td>Assumed</td>
</tr>
<tr>
<td>Natural rate of progression to AIDS</td>
<td>(\rho)</td>
<td>0.1yr(^{-1})</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Modification parameter</td>
<td>(\eta)</td>
<td>1.02yr(^{-1})</td>
<td>Assumed</td>
</tr>
<tr>
<td>AIDS related death rate</td>
<td>(\nu)</td>
<td>(0.333-0.4)yr(^{-1})</td>
<td>Bhunu et al. 2009a</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>0.011-0.95yr(^{-1})</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Modification parameter</td>
<td>(\theta)</td>
<td>0.33yr(^{-1})</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

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Fig. 2. Simulations of model system (3) showing plots of susceptibles, AIDS cases, HIV positive individuals not aware of their status and those who are as a function of time with various initial conditions. (a) Varying the susceptible initial population. (b) Varying the initial population of HIV positive people unaware of their status. (c) Varying the initial population of HIV positive people aware of their status. Parameters values used are as in Table 1.

not know their status start off, increasing at a faster rate than those who do know their status. However, as testing and counselling gain ground, more people end up knowing their status. Here we ask: If more people are getting to know their status, why is the epidemic growing in resource limited countries? This tend to suggest that the epidemic is being fuelled by people who know their status, further suggesting that testing and counselling should be accompanied by a positive sexual behavioural change (with possible status disclosure which can be acheive by effective stigma fighting via focus educational campaigns). Status disclosure and or positive behavioural change if taken seriously in resource limited settings, may greatly influence the dynamics of the epidemic. The impact of these is a subject of further investigation elsewhere.

Fig. 4 is a graphical representation depicting the effect of increasing the contact rate $c$ on the susceptible, HIV positive and AIDS populations. Fig. 4 (a) shows an increase in the contact rate $c$ results in the depletion of the susceptible pool as more people get infected. The remaining Figs. 4 (b), 4 (c) and 4 (d) show that

---

Table 1: Model parameters and their interpretations.

<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>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Natural mortality rate</td>
<td>$\mu$</td>
<td>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Natural rate of progression to AIDS</td>
<td>$\rho$</td>
<td>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Proportion aware of their status soon after infection</td>
<td>$\delta$</td>
<td>(0.333-0.4) yr</td>
<td>Assumed</td>
</tr>
<tr>
<td>Proportion aware of their status later</td>
<td>$\eta$</td>
<td>1.0 yr</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>AIDS related death rate</td>
<td>$\nu$</td>
<td>0.1 yr</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Natural rate of progression to AIDS</td>
<td>$\rho$</td>
<td>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Modulation parameter</td>
<td>$\theta$</td>
<td>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Product of effective contact rate for HIV infection and probability of HIV transmission per contact</td>
<td>$\theta$</td>
<td>0.02 yr$^{-1}$</td>
<td>Bhunu et al. 2009a</td>
</tr>
<tr>
<td>Population size</td>
<td>$I$</td>
<td>0.5</td>
<td>Assumed</td>
</tr>
<tr>
<td>Time (years)</td>
<td>$S$</td>
<td>0.1</td>
<td>Bhunu et al. 2009a</td>
</tr>
</tbody>
</table>

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know their status and have somewhat “given up the hope of living” have a great potential to willingly fuel the disease as they progress to the AIDS stage. The disease-free equilibrium was obtained and is stable if and only if \( R_0 < 1 \).

Figure 2 is a graphical representation depicting the effects of varying initial conditions. From graphs in Figure 2, we observe that as long as \( \eta > I \) and \( \theta > Q \), varying the initial conditions will not change the final distribution of the population. The direction of the arrow in Figures 3(a) and 3(c) show a decrease in \( \eta \) and \( \theta \), respectively. Increasing \( \eta \) and \( \theta \), decreasing \( \eta \) and \( \theta \) results in an increase of people who know their status and the AIDS cases will converge asymptotically to the endemic state. This asymptotic state is reached as in all cases the susceptible, HIV positive unaware of their status, HIV positive who know their status and the AIDS cases will converge asymptotically to the endemic state. This asymptotic state is reached as in all cases the susceptible population decreases and the infected population increases.

This further suggests the future of the HIV/AIDS epidemic will be driven by people who know their status. Hence, the following question: Is the world doing enough for those who test HIV positive in resource limited countries? It seems more needs to be done to encourage abstinence and or positive sexual behavioural change, but without education and incentives, poverty striken communities may not commit to such behavioural change.

Increasing \( c \) results in an increase of HIV positive cases unaware of their status, HIV positive cases who know their status and AIDS cases, respectively. An increase of HIV positive translates to an increase of the AIDS cases as HIV positive cases progress to the AIDS stage. Also, Fig. 4 illustrates both the disease-free which occurs when \( R_T < 1 \) and the endemic equilibrium when \( R_T > 1 \).

5 Conclusion

We proposed a deterministic compartmental model in an attempt to investigate the existing problem of HIV/AIDS in poor countries, in the presence of testing and counselling. The disease-free equilibrium was shown to be globally asymptotically stable when the corresponding reproduction number is less than unity. The Centre Manifold theory was employed to show that the endemic equilibrium point is locally asymptotically stable when the corresponding reproduction number is greater than unity. From the analysis of the reproduction number, we conclude that in the absence of antiretroviral therapy and counselling, HIV positive people who know their status and have somewhat “given up the hope of living” have a great potential to willingly fuel the disease as they progress to the AIDS stage.
the epidemic. Since life prolonging drugs are not made easily available to people in poor resource settings, then we ask: Is it morally right to test individuals for HIV only for the reason of taking statistics? This may not be enough in the fight against HIV/AIDS as long as some HIV positive who know their status opt for HIV spreading spree. Should resource limited countries consider compulsory testing and status disclosure of those who are HIV positive as was recently suggested by the Swaziland minister of health[2]? Again this may lead to stigmatisation and many other human rights abuses against those known to be HIV positive. Public health personnel in order to make informed decision should consider community-based counselling via massive educational campaigns that account for the community social norms and beliefs. This can be achieved by providing free and constant supply of antiretrovirals with hope that more HIV positive people can come out in the open or change their behaviour and adopt protective measures. Free antiretroviral drugs roll out plan may stimulate people to go for testing and counselling (a behaviour similar to the famous “Pavlov’s dogs”) once there is assurance of incentives and uninterrupted treatment. From the numerical simulations, at the long run there will be more HIV positive people who know their status than those who do not. Testing and counselling is not enough if not accompanied by individuals positive behavioural change. Therefore,
conventional educational methods should therefore give prominence to social structure and cultural beliefs of the community in their counselling portfolio as well as incentives to encourage behavioural change. It seems the world is not doing enough in this direction, especially in rural communities. Nevertheless, all hope is not lost as [26] conclude in a recent study that eradication of AIDS is feasible, using the tools that we have currently at hand, but action needs to occur immediately, if not, then HIV/AIDS will race beyond our ability to afford it. This study is not exhaustive and can be extended in various ways: since the future trend of HIV/AIDS infection might be driven by those who know their status and willingly transmit the disease, this, we shall investigate critically in a future study. Also, an in-depth analysis of the “myth cleansing” is viable.

References


