Mathematical Modelling and Analysis
Volume 13 Number 4, 2008, pages 539–552
Doi:10.3846/1392-6292.2008.13.539-552
 ISSN 1392-6292 print, ISSN 1648-3510 online

ON THE COMPLEXITIES OF MODELING HIV/AIDS IN SOUTHERN AFRICA

F. NYABADZA

South African Center of Epidemiological Modeling and Applications
19 Jonkershoek Road, Stellenbosch, 7600, South Africa
E-mail: nyabadzaf@sun.ac.za

Received August 11, 2007; revised February 3, 2008; published online December 1, 2008

Abstract. In this paper problems associated with the modeling of HIV/AIDS in Southern Africa are presented. A mathematical model is presented to highlight the three major challenges of modeling HIV/AIDS, i.e condom use, vertical transmission and treatment. The model analysis for the case, where the treatment parameter $\rho = 0$, is presented in terms of the model reproduction number R and threshold parameters R_T and R_A that show the contribution of vertical transmission. It is shown that if R, R_T, $R_A < 1$, then the disease free equilibrium point is both locally asymptotically and globally stable. Numerical simulations for the model are presented to determine the role of some key epidemiological parameters of the model.

Key words: Complexities, interventions, reproduction number, stability, equilibrium points.

1 INTRODUCTION

Southern Africa remains the epicenter of the global AIDS epidemic. Of the estimated 24.5 million people who were living with HIV in sub-Saharan Africa at the end of 2005, 46% were from Southern Africa [12]. The epidemic however seems to be stabilizing at high prevalence levels. Mathematical models have been used to model the dynamics of HIV/AIDS [1, 7, 9, 13]. Incorporation of interventions in these models has attracted significant attention in recent years [8, 10, 14]. The epidemiology of HIV/AIDS has moved beyond the virus and the risk factors associated with its transmission to a more detailed understanding of the mechanisms associated with the spread, distribution and impact of any intervention on the population. In HIV epidemiology, mathematical models can describe the position of individuals within networks of sexual partners via which infections spread, allowing an identification of risks of acquiring and transmitting the infection. The population patterns of HIV incidence can be simulated based upon descriptions of patterns of sexual behavior and viral biology which are then compared with observed patterns. Consequently, the

impact of health policies, such as poor access to care, delayed treatment or the use of screening for asymptomatic cases can be calculated [3]. The process of presenting a model to describe the spread of HIV is accompanied by underlying assumptions and the need for data to estimate parameter values for the qualitative and quantitative predictions that can then be compared with experimental or observed patterns. The outcomes are significant for health planning purposes and disease management.

Scientists have given different theories and explanations for the rapid spread of HIV/AIDS in Southern Africa, some based on, biological and social aspects, economic and political processes [4, 15]. Many complexities such as regional diversities in cultures, sexual behavior and migration patterns remain unexplained by these theories [2]. One can then argue that the spread of HIV/AIDS is not only influenced by an individual's actions but also by political, social norms and economic standards of a society. The dynamics of HIV/AIDS is thus multidimensional and can not be focused from a single approach.

Literature on HIV/AIDS documents that Southern Africa is hardest hit and as such, there is urgent need for interventions. Any proposed intervention needs greater understanding of the social networks, economic and political situations, beliefs and the role of the ever evolving cultural paradigms. In this paper we discuss the complexities associated with modeling HIV/AIDS in Southern Africa. We also look at the emerging issues and challenge mathematicians to include these issues in their models with aim of developing appropriate approaches to HIV/AIDS prevention and management in high disease transmission settings such as Southern Africa. For the benefit of mathematical modeling, we attempt to answer the following questions: Why is HIV/AIDS such a big challenge in Southern Africa? What are the main factors that significantly drive the epidemic? Can the dynamics of HIV/AIDS be adequately modeled mathematically?

This paper is arranged as follows. Section 2 considers an example of a mathematical model that includes treatment and intervention strategies on the unfolding HIV/ADIS pandemic. A model with no treatment of individuals with AIDS is analyzed in Section 3. In Section 4, we consider a model with treatment of individuals with AIDS. Numerical simulations are considered in Section 5 and and some concluding remarks are given in Section 6.

2 Model Formulation

The model follows some ideas presented in [10]. The disease divides the population into classes of individuals that are susceptible, with density $S(t)$, infected, with density $I(t)$, those that are under treatment, with density $T(t)$ and those with AIDS, with density $A(t)$. HIV transmission is assumed to be heterosexual, so that the population under consideration is sexually active. Population mixing is assumed to be homogeneous and the total population size $N(t)$ of sexually active individuals is given by

$$N(t) = S(t) + I(t) + T(t) + A(t).$$

Individuals move from one class to the other as their status with respect to the disease changes. Sexually active individuals are recruited at a constant rate π, the natural death rate $\mu > 0$, is assumed to be proportional to the number of individuals in each class and a disease related death rate ($d > 0$) proportional to the number of individuals in the AIDS class. The recruited individuals are assumed to be susceptible. Upon a sexual encounter, without protection, with an infected individual, from the $I(t)$, $T(t)$ and $A(t)$ compartments, a susceptible individual will become infected with probabilities β_1, β_2 and β_3 respectively. To model the effects of condom use, we assume that condoms have an efficacy ϵ_1 and compliance by individuals using them being α_1 ($0 \leq \alpha_1,\ \epsilon_1 \leq 1$) so that the product $p_1 = \alpha_1\epsilon_1$ measures the level of protection against HIV by the use of condoms. Here p_1 is defined as the condom induced preventability [7]. Thus $(1 - p_1)$ measures the condom induced preventability failure. Hence susceptible individuals are infected at a rate $c(1-p_1)\lambda S(t)$, where c is the rate of acquisition of new partners and λ is the average risk of infection given by

$$\lambda = \frac{\beta_1 I(t) + \beta_2 T(t) + \beta_3 A(t)}{N(t)}. \tag{2.1}$$

We assume individuals seek treatment at a rate ($\sigma > 0$). In Southern Africa, especially in rural communities, it is common to have individuals progressing to AIDS without being tested. We shall assume that the rate of progression to the AIDS class from the I class is given by $\nu_1 > 0$. Treatment reduces the rate of progression to AIDS and sometimes reverses the progression resulting in amelioration. We assume individuals from the T class progress to AIDS at a rate $\gamma > 0$. To take into consideration how this progression is affected by treatment, we assume a drug efficiency ϵ_2 for ARV drugs and adherence to treatment α_2. α_2 measures the proportion of individuals that take their drugs consistently and correctly. By correctly we mean taking the drugs in their correct amounts at the correct times. We define the product of drug efficiency and adherence, ($p_2 = \epsilon_2\alpha_2$), as the drug induced preventability of progression to AIDS by an HIV infected individual per given time. This parameter measures the level of reduction in the severity of HIV when an individual is taking ARV drugs. It is however common to have non-adherence to treatment in Southern Africa, due to, non-affordability of the drugs, forgetting, being away from home, being too busy, or even the feeling that one is okay. Like in any part of the world, HIV treatment adherence will remain a significant problem in growing populations of substance abusing and economically disadvantaged persons living with HIV/AIDS [5]. So individuals progress to AIDS at a treatment adjusted rate $\nu_2 = \gamma(1 - p_2)$.

To model the role of vertical transmission, we include virus free births by HIV infected mothers at a rate $b(1 - \epsilon)e^{-\mu_0}(I + T + A)$, where b is the birth rate and ϵ is the fraction of offsprings born with the virus. If we assume that individuals with AIDS can spread the disease, we can equally assume that they can fall pregnant and are capable of producing children. A proportion q of children born with the virus are subjected to treatment and when they survive the maturation age, τ (between 12 and 15 years), progress to the class of those under treatment while a proportion $(1 - q)$, progress to the AIDS class. The

probability of surviving the juvenile stage is given by $e^{-\mu_0\tau}$, where μ_0 is the natural death rate of the juveniles [6].

To model treatment of individuals in the AIDS class, we introduce a constant treatment parameter $\rho > 0$. Treated individuals are assumed to get better and move to the T-class. This is particularly true in the case of Southern Africa where treatment is only offered by governments when individuals' CD_4T cell count is around 200 cells/mm^3. We however have individuals who can access private medical facilities and can start treatment when the CD_4T cell is way above 200 cells/mm^3. Our model is thus governed by the following nonlinear system of differential equations,

$$\dot{S} = \pi + b(1-\epsilon)e^{-\mu_0\tau}I_A - c(1-p_1)\lambda S - \mu S, \tag{2.2}$$

$$\dot{I} = c(1-p_1)\lambda S - (\mu+\sigma+\nu_1)I, \tag{2.3}$$

$$\dot{T} = m_1 I + m_{41}A - (\mu+\nu_2)(1-R_T)T, \tag{2.4}$$

$$\dot{A} = m_2 T + m_3 I - (\mu+d+\rho)(1-R_A)A, \tag{2.5}$$

$$S(0) = S_0,\ \ I(0) = I_0,\ \ T(0) = T_0,\ \ A(0) = A_0,$$

where $I_A = I + T + A$,

$$m_1 = \sigma + bq\epsilon e^{-\mu_0\tau},\ m_2 = \nu_2 + b(1-q)\epsilon e^{-\mu_0\tau},\ m_3 = \nu_1 + b(1-q)\epsilon e^{-\mu_0\tau},$$

$$m_{41} = \rho + bq\epsilon e^{-\mu_0\tau},\ R_T = \frac{bq\epsilon e^{-\mu_0\tau}}{\mu+\nu_2},\ \ R_A = \frac{b(1-q)\epsilon e^{-\mu_0\tau}}{\mu+d}.$$

The rate of change of the population N, is given by

$$\dot{N} = \pi + be^{-\mu_0\tau}I_A - \mu N - dA. \tag{2.6}$$

The growth of the sexually active population is due to recruitment and those that survive the juvenile stage under the condition $be^{-\mu_0\tau}$. Continuity of the right hand side of equations (2.2)–(2.5) and their derivatives implies that the model is well posed.

3 Model Analysis (the case $\rho = 0$)

We begin by considering the case in which the is no treatment of individuals in the AIDS class, i.e the case $\rho = 0$, but treatment is offered after case by case detection when individuals are tested. The model equations are thus given by (2.2)–(2.5), where $m_{41} := m_4 = bq\epsilon e^{-\mu_0\tau}$ and the other parameters remain the same.

3.1 Equilibria and Model Reproduction Number

Since $N(t) = S(t) + I(t) + T(t) + A(t)$, we can rewrite these equations as

$$\dot{N} = \pi + be^{-\mu_0\tau}I_A - \mu N - dA, \tag{3.1}$$

$$\dot{I} = c(1-p_1)\lambda(N - I_A) - (\mu+\sigma+\nu_1)I, \tag{3.2}$$

$$\dot{T} = m_1 I + m_4 A - (\mu + \nu_2)(1 - R_T)T, \tag{3.3}$$

$$\dot{A} = m_2 T + m_3 I - (\mu + d)(1 - R_A)A, \tag{3.4}$$

$$N(0) = S_0,\ I(0) = I_0,\ \ T(0) = T_0,\ \ A(0) = A_0,$$

In the absence of infection, the population size approaches π/μ.

We now consider system (3.1)–(3.4) at equilibrium. Equation (3.4), gives

$$A = \frac{1}{(\mu + d)(1 - R_A)} \left(m_2 T + m_3 I\right). \tag{3.5}$$

Substituting (3.5) into (3.3) we have

$$T = \phi_1 I, \quad \phi_1 = \frac{m_1(\mu + d)(1 - R_A) + m_3 m_4}{(\mu + d)(\mu + \nu_2)(1 - R_T)(1 - R_A) - m_2 m_4}. \tag{3.6}$$

We can thus write (3.5) in terms of I only, such that

$$A = \phi_2 I, \quad \phi_2 = \frac{m_1 m_2 + m_3(\mu + \nu_2)(1 - R_T)}{(\mu + d)(\mu + \nu_2)(1 - R_T)(1 - R_A) - m_2 m_4}. \tag{3.7}$$

From (2.1), we have

$$\lambda = \psi \frac{I}{N}, \tag{3.8}$$

where $\psi = \beta_1 + \phi_1 \beta_2 + \phi_2 \beta_3$. From equations (3.6) and (3.7), we also have

$$I_A = \xi I, \quad \xi = 1 + \phi_1 + \phi_2. \tag{3.9}$$

From equations (3.8), (3.9) and (3.2) we get a quadratic equation in I whose solutions are $I = 0$ and

$$I = \frac{1}{\xi}\left[1 - \frac{1}{R}\right] N,$$

where R is the model reproduction number given by

$$R = \frac{c(1 - p_1)}{\mu + \sigma + \nu_1}\psi.$$

It is defined as the number of secondary cases that are caused by one primary case introduced into a population that is wholly susceptible. $I = 0$ corresponds to the disease free equilibrium point E_0, given by $E_0 = (\pi/\mu, 0, 0, 0)$. For $I \neq 0$ we have the endemic equilibrium point given by $E_1(N^*, I^*, T^*, A^*)$, where

$$N^* = \frac{\pi R \xi}{\mu R \xi + (d\phi_2 - b\xi e^{-\mu_0 \tau})(R - 1)}, \quad T^* = \phi_1 I^*,$$

$$A^* = \phi_2 I^*, \quad I^* = \frac{1}{\xi}\left(1 - \frac{1}{R}\right) N^*.$$

Firstly, we give a proposition on conditions that guarantee the positivity of ϕ_1 and ϕ_2, and then give a theorem on the existence of the endemic equilibrium point.

Proposition 1. *If $R_A < 1$ and $R_T < 1$ then ϕ_1 and ϕ_2 are positive.*

Proof. Substituting the parameter expressions of m_2 and m_4 into the denominators of ϕ_1 and ϕ_2, we can easily establish the relation

$$R_T < \frac{\mu + d}{\mu + d + \nu_2}(1 - R_A) < 1.$$

Since the numerators are positive, the result follows. □

The following theorem summarizes the existence of the model equilibria.

Theorem 1. *The system* (3.1)–(3.4) *has a disease free equilibrium point that exists for all values of R and a unique endemic equilibrium point E_1, whenever $R > 1$, $R_T < 1$ and $R_A < 1$.*

The basic reproduction number of infection is known to provide the necessary condition for the eradication of an epidemic. If it is less than unity, the disease can be eradicated, otherwise it will persist in the population. If we set $\epsilon = 0$, the model reproduction number R reduces to R_0 determined in [10]. The model has two important threshold parameters, R_T and R_A. As in [6], these parameters define the reproduction numbers for the demographic replacement of the infectives under treatment and those with AIDS respectively through vertical transmission. Equation (3.5) only holds for $R_A < 1$ and equation (3.6) holds for $R_A < 1$ and $R_T < 1$. The conditions $R_T < 1$ and $R_A < 1$, imply that the demographic replacement of the infectives can not lead to an epidemic, other factors need to be taken into consideration. The inclusion of these parameters in the net model reproduction number, will help to determine the contribution of vertical transmission to the overall spread of the epidemic.

3.2 Stability analysis of E_0

3.2.1 Local stability of the disease-free equilibrium

Local stability of the disease free equilibrium point can be obtained by computing the variational matrix of system (3.1)-(3.4) at the disease free equilibrium point. We thus have,

$$J_{E_0} = \begin{pmatrix} -\mu & be^{-\mu_0\tau} & be^{-\mu_0\tau} & be^{-\mu_0\tau} - d \\ 0 & (\mu+\sigma+\nu_1)(R_1-1) & c(1-p_1)\beta_2 & c(1-p_1)\beta_3 \\ 0 & m_1 & (\mu+\nu_2)(R_T-1) & m_4 \\ 0 & m_3 & m_2 & (\mu+d)(R_A-1) \end{pmatrix}.$$

The eigenvalues of J_{E_0} are $(-\mu)$ and the solutions of the the characteristic equation

$$P(\vartheta) = \vartheta^3 + a_2\vartheta^2 + a_1\vartheta + a_0 = 0,$$

where

$$\begin{aligned} a_2 &= (\mu+\sigma+\nu_1)(1-R_1) + (\mu+\nu_2)(1-R_T) + (\mu+d)(1-R_A), \\ a_1 &= (\mu+\sigma+\nu_1)(1-R_1)[(\mu+\nu_2)(1-R_T) + (\mu+d)(1-R_A)] \\ &\quad + [(\mu+d)(\mu+\nu_2)(1-R_A)(1-R_T) - m_2m_4](1-\Re), \\ a_0 &= (\mu+\sigma+\nu_1)[(\mu+d)(\mu+\nu_2)(1-R_A)(1-R_T) - m_2m_4](1-R), \end{aligned}$$

and

$$\Re = \frac{m_1 c(1-p_1)\beta_2 + m_3 c(1-p_1)\beta_3}{(\mu+d)(\mu+\nu_2)(1-R_A)(1-R_T) - m_2 m_4}.$$

We can observe that a_0 and a_2 are positive for $R < 1$ and $R_T < 1$ and $R_A < 1$. By comparing $\Re$ to the model reproduction number we can also observe that $a_1 > 0$ for $R < 1$. We now state the theorem on the local stability of E_0, using the Routh-Hurwitz criteria.

Theorem 2. *Let $R_T < 1$ and $R_A < 1$. The disease free equilibrium point E_0 is locally asymptotically stable for $R < 1$ provided $a_1 a_2 - a_0 > 0$ otherwise it is unstable.*

3.2.2 Global stability of the disease-free equilibrium

To prove globally stability of the disease-free equilibrium point, we use the approach in [11]. For a bounded real-valued function g on the interval $[0, \infty)$, we define

$$g_\infty = \lim_{t\to\infty} \inf g(t), \quad g^\infty = \lim_{t\to\infty} \sup g(t)$$

and therefore we state the following lemma.

Lemma 1. (Thieme 1993[11]) *Let $g : [0, \infty) \to \mathbb{R}$ be bounded and twice differentiable with a bounded second derivative. Let $t_n \to \infty$ and $g(t_n)$ converges to g_∞ or g^∞ for $n \to \infty$, then $g'(t_n) \to 0$, $n \to \infty$.*

We thus have the following theorem on the global stability of E_0.

Theorem 3. *If $R_T < 1$ and $R_A < 1$, the disease free equilibrium point E_0 is globally asymptotically stable for $R < 1$.*

Proof. Suppose $R < 1$, we choose a sequence $t_n \to \infty$ such that $T(t_n) \to T^\infty$, $\dfrac{dT(t_n)}{dt} \to 0$, then $T^\infty \le \phi_1 I^\infty$. This result follows directly from the derivation of (3.6). Choosing a second sequence $s_n \to \infty$ such that $A(s_n) \to A^\infty$ and $\dfrac{dA(s_n)}{dt} \to 0$, then $A^\infty \le \phi_2 I^\infty$. Choosing a third sequence $r_n \to \infty$ such that $I(r_n) \to I^\infty$ and $\dfrac{dI(r_n)}{dt} \to 0$ then,

$$c(1-p_1)\psi \frac{S^*}{N^*} I^\infty - (\mu+\sigma+\nu_1) I^\infty \ge 0. \tag{3.10}$$

Since $S^*/N^* = 1$, equation (3.10) reduces to $(\mu+\sigma+\nu_1)(R-1)I^\infty \ge 0$.

Also, since $R < 1$ it implies that $I^\infty \le 0$, which is only possible if $I^\infty = 0$. Since $I^\infty \ge 0$, we thus have

$$I^\infty = I_\infty = 0 \Rightarrow I(t) \to 0 \quad \text{as} \quad t \to \infty.$$

From estimates for T^∞ and A^∞ we have $T(t) \to 0$ and $A(t) \to 0$ as $t \to \infty$. It follows from equation (3.1) that $N \ge \Pi/\mu$.

But for $N > \Pi/\mu$ it follows that $\frac{dN}{dt} \leq 0$. Therefore we consider the solution for which $N^{\infty} \leq \frac{\Pi}{\mu}$, then

$$N_{\infty} = N^{\infty} = \frac{\Pi}{\mu}.$$

Hence E_0 is globally asymptotically stable. □

The stability of the interior equilibrium point can be invstigated using the characteristic equation corresponding to the jacobian matrix at the endemic equilibrium point. The characteristic equation is a fourth-degree polynomial. Using a symbolic algebra program, it can be shown that the Routh–Hurwitz criterion is satisfied if $R > 1$ and $R_T < 1$ and $R_A < 1$. The endemic equilibrium point is thus locally asymptotically stable.

4 Model Analysis (the case $\rho \neq 0$)

In this section we consider system (2.3)–(2.6). It has two equilibria, i.e. E_0 the disease free equilibrium point and $E_2 = (\bar{N}, \bar{I}, \bar{T}, \bar{A})$, where E_0 is defined as in the case $\rho = 0$ and

$$\bar{N} = \frac{\pi R_\rho \chi}{\mu R_\rho \chi + (d\varphi_2 - b\chi e^{-\mu_0 \tau})(R_\rho - 1)}, \quad \bar{T} = \varphi_1 \bar{I},$$

$$\bar{A} = \varphi_2 \bar{I}, \quad \bar{I} = \frac{1}{\chi}\left(1 - \frac{1}{R_\rho}\right)\bar{N}, \quad \chi = 1 + \varphi_1 + \varphi_2,$$

$$\varphi_1 = \frac{m_1(\mu + d + \rho)(1 - R_A) + m_3 m_{41}}{(\mu + d + \rho)(\mu + \nu_2)(1 - R_T)(1 - R_A) - m_2 m_{41}},$$

$$\varphi_2 = \frac{m_1 m_2 + m_3(\mu + \nu_2)(1 - R_T)}{(\mu + d + \rho)(\mu + \nu_2)(1 - R_T)(1 - R_A) - m_2 m_{41}},$$

R_ρ is the model reproduction number, written in a compact form as

$$R_\rho = \frac{c(1 - p_1)}{\mu + \sigma + \nu_1}\bar{\psi},$$

where $\bar{\psi} = \beta_1 + \varphi_1\beta_2 + \varphi_2\beta_3$. In an expanded form $R_\rho = R_{01} + R_{02} + R_{03}$:

$$\begin{aligned}
R_{01} &= \frac{c(1 - p_1)\beta_1}{(\mu + \sigma + \nu_1)}, \\
R_{02} &= \frac{c(1 - p_1)\beta_2[m_3 m_{41} + m_1(d + \mu + \rho)(1 - R_A)]}{(\mu + \sigma + \nu_1)[(d + \mu + \rho)(1 - R_A)(1 - R_T)(\mu + \nu_2) - m_2 m_{41}]}, \\
R_{03} &= \frac{c(1 - p_1)\beta_3[m_1 m_2 + m_3(\mu + \nu_2)(1 - R_T)]}{(\mu + \sigma + \nu_1)[(d + \mu + \rho)(1 - R_A)(1 - R_T)(\mu + \nu_2) - m_2 m_{41}]}.
\end{aligned}$$

Clearly if $\rho = 0$, we have $R_\rho = R$. The following theorem is valid on the existence of the endemic equilibrium point E_2.

Theorem 4. *The equilibrium point E_2 exists for $R_\rho > 1$.*

The stability analysis of the equilibria of (2.3)–(2.6) can be carried out as done for the case $\rho = 0$. This model focusses on the implications of treating individuals with AIDS. It is known that treatment leads to decreased infectivity and increased duration of infectiousness. We ask the question: what are the implications of treating individuals with AIDS? We attempt to answer this question using numerical simulations for this model.

5 Numerical Simulations

In this section the results of numerical simulations for the system (2.2)–(2.5) are given. The parameter values are from [6, 10]. For the model with no treatment of individuals with AIDS, i.e the case $\rho = 0$, Figure 1(a) shows the change in prevalence as we increase condom use. Modeling condom use is synonymous to modeling behavior change. Increased condom use is a measure of increased behavior change. We can clearly see that for the chosen parameter values, increasing condom use (behavior change) leads to the reduction in prevalence. The prevalence curves shows a sharp decline for high values of p_1. For instance, if the reproduction number is 1.5118, the prevalence levels off at 25% if no other interventions are instituted but if 90% of the individuals use condoms consistently then there will be a sharp decline in the prevalence and the reproduction number ($R = 0.3042$). Similar curves can be plotted to investigate the effects of increasing or decreasing the number of sexual partners. Reducing the values of c leads to a decline the prevalence and the reproduction number.

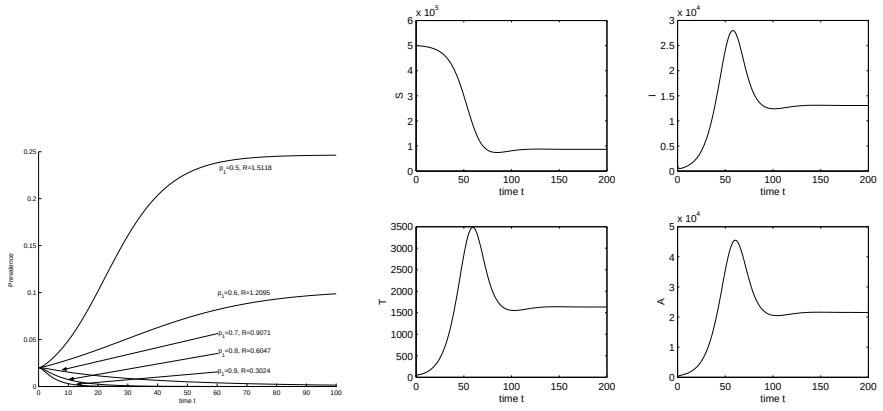

Figure 1. Results of simulations for the given parameter values $\mu = 0.02,\ \nu_1 = 0.6,\ \nu_2 = 0.7,\ \epsilon = 0.06,\ b = 0.03,\ \sigma = 0.09,\ \pi = 10000,\ d = 0.4,\ \beta_1 = 0.004,\ \beta_2 = 0.04,\ \beta_3 = 0.3,\ \mu_0 = 0.001,\ c = 4,\ q = 0.01,\ \rho = 0$: a) the prevalence curve for $\tau = 13\ years$, b) profiles of populations for $p_1 = 0.5,\ \tau = 15\ years$.

Figure 1(b) shows the population dynamics for high values of c (in this particular case $c = 4$) for the case $\rho = 0$. The population in each class settles

to an endemic equilibrium over time. Increasing the number of sexual partners can lead to endemicity of HIV/AIDS in the population.

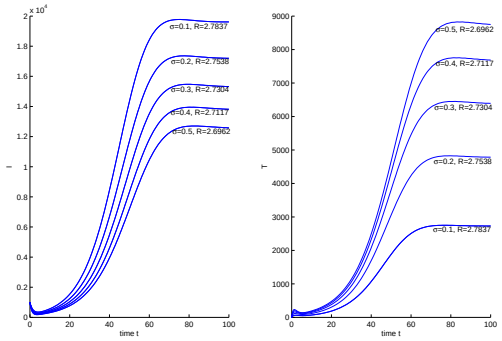

Figure 2. Profile of the T and I classes for the following parameter values $\mu = 0.06,\ p_1 = 0.5,\ \nu_1 = 0.6,\ \nu_2 = 0.7,\ \epsilon = 0.06,\ b = 0.03,\ \pi = 10000,\ d = 0.04,\ \beta_1 = 0.4,\ \beta_2 = 0.04,\ \beta_3 = 0.3,\ \mu_0 = 0.001,\ \tau = 13\ years,\ c = 4,\ \rho = 0$ and $q = 0.01$.

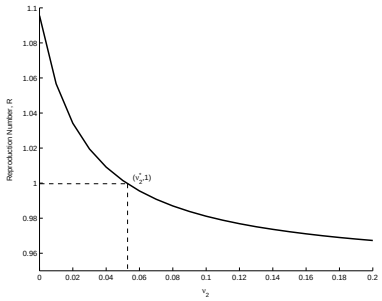

Figure 3. Profile of populations for the following parameter values $\mu = 0.03,\ p_1 = 0.7,\ \nu_1 = 0.6,\ \epsilon = 0.3,\ b = 0.03,\ \sigma = 0.6,\ \pi = 10000,\ d = 0.4,\ \beta_1 = 0.4,\ \beta_2 = 0.08,\ \beta_3 = 0.3,\ \mu_0 = 0.03,\ \tau = 13\ years,\ c = 2,\ \rho = 0$ and $q = 0.01$.

The rate at which individuals join the class of those under treatment σ, is of significant importance, especially in Southern Africa. It is a parameter that depicts the decreasing levels of stigma surrounding HIV/AIDS. Higher values of σ are a sign of decreased stigma. One can not join the class without testing for HIV. We can also take the parameter σ, to measure the response of individuals to the HIV testing campaigns. Testing for HIV is valuable for those who are positive because it raises the need to change their behavior so as not to spread the virus. It is a measure of early HIV diagnosis that will lead to early treatment. Figure 2 show the dynamics of the infected individuals and those under treatment when we vary σ.

Figure 3 depicts the variation of the reproduction number with ν_2 for the case $\rho = 0$. For any given set of parameter values we can always determine the

minimum treatment rate ν_2^*. The epidemic will persist of $\nu_2 < \nu_2^*$.

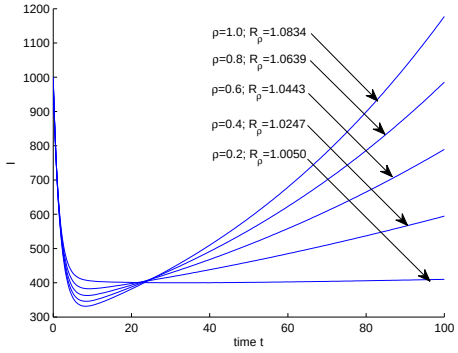

Figure 4. Profile of infectives as ρ changes, $\mu = 0.02$, $p_1 = 0.5$, $\nu_1 = 0.6$, $\nu_2 = 0.7$, $\epsilon = 0.6$, $b = 0.03$, $\sigma = 0.09$, $\pi = 10000$, $d = 0.4$, $\beta_1 = 0.4$, $\beta_2 = 0.04$, $\beta_3 = 0.3$, $\mu_0 = 0.001$, $\tau = 13$ *years*, $c = 1$, and $q = 0.01$.

For the case $\rho \neq 0$, we note that, increasing the value of ρ leads to an increase in the reproduction number R_ρ. It is interesting to note that a massive role out of treatment is beneficial for a specific period of time but in the long run, can cause massive increases in the number of infectives. This brings an important phenomenon with regard to treatment of HIV/AIDS. The longer individuals stay in the system the more chances there are of them transmitting the infection if no behavioral change occurs. We can thus say as long as there is 'recycling of infectives', the HIV/AIDS pandemic will persist. We consider 'recycling of infectives' to mean a process where an infective is about to exit a system and is brought back into the system, still being infectious. This is done by considering the change in the reproduction number R_ρ as ρ changes. Figure 4 shows how the number of infectives change with increasing treatment.

6 Discussion and Conclusion

In this paper, some of the major challenges that surround HIV/AIDS modeling in Southern Africa are discussed. The paper presents specific aspects that mathematical modelers should consider when designing mathematical models on HIV/AIDS that are specific to Southern Africa. Three major challenges are highlighted in the mathematical model. These are, vertical transmission, treatment and condom use. The basis for HIV infection prevention are based on a clear understanding of the aetiology and natural progression the disease.

Vertical transmission is a major cause of paediatric HIV infection in Southern Africa. Interventions to prevent vertical transmission may be complicated by cultural, social and economic dimensions of the region. It becomes therefore difficult to quantify parameters that relate to vertical transmission. On the other hand, while condoms are highly effective barriers to HIV transmission, they are not completely effective in preventing the transmission of HIV

infections. Consistent use of condoms remains a major challenge and quantifying parameters that relate to consistent use of condoms is therefore difficult. Condom efficacy rates vary from one region to the other. The large range of efficiency rates is related to incorrect or inconsistent use of the condoms. This makes quantification of condom use more difficult. Treatment of HIV has improved the lives of many AIDS suffers in Southern Africa. It also has improved the outcomes of opportunistic infections such as tuberculosis which has reemerged as a major cause of death around the world. Treatment of AIDS has reduced stigma and encouraged testing. With such important benefits it is clear, that models that include treatment as an intervention strategy should be validated with the correct data. The data on treatment is however hampered by variations in drug efficacy, adherence by patients, toxicity, availability, nutrition and human behavior. All these factors determine the success of a treatment program. A major question arises; can we factor all these challenges in our model using accurate data?

While many mathematical models need validation using data collected in health care facilities, challenges remain on data accuracy, availability and its usefulness. Most of the data collected serves well for the purposes of service delivery evaluation and management but may not be entirely useful for research purposes. Very few countries in Southern Africa have data collection policies that support research work. Most of the data collection is donor funded and the data may remain the property of the funding agent and thus may not be available to other researchers. A data base, where researchers can obtain information on HIV/AIDS for Southern Africa may serve the purpose.

The interconnectedness of the challenges facing Southern Africa in confronting the HIV pandemic admittedly makes modeling difficult. One can however group the challenges to simplify the modeling approach. Following infection with HIV, the rate of clinical disease progression varies between individuals. Factors such as host susceptibility, genetics and immune function, nutrition, health care and co-infections, as well as viral genetic variability may affect the rate of progression to AIDS. Knowledge about HIV and the immune system has increased in recent years. It is now well understood that progression to AIDS is linked to the decline in the $CD4^+$ T-lymphocytes. This requires a cell dynamics model and as such can not be incorporated into the given example.

Treatment of those infected is shouldered by the governments, which are struggling to cope with brain drain, poverty and other related issues. Service delivery has not been an easy task as the number of AIDS cases and deaths increase. A number of children have been orphaned by the disease. Adoption in the African culture is not well developed. Many cultural settings prohibit parenting a child whose genealogy is not known. Orphanages remain the only option but they are also faced with funding problems and one can only imagine the next generation of parents from these orphanages.

In the model considered as an example, we have considered the effect of decreased infectivity, due to treatment, and increased duration of infectiousness by 'recycling of infectives'. We have demonstrated through numerical simulations that the effect of increased duration of infectiousness may be to increase

the pool of transmitters of infection. ARV administration may result in an increase in life expectancy of individuals over time and this may result in an increase in a pool of transmitters of the disease. Our result has been obtained in the absence of education on prevention. The situation may of course improve with effective education on prevention and other factors that reduce the risks of acquiring infection.

Acknowledgements

The author would like to thank SACEMA for supporting this work.

References

[1] S. M. Blower and A. R. McLean. Prophylactic vaccines, risk behavior change, and the probability of eradicating HIV in san francisco. *Science*, **265**:1451–1454, 1994.

[2] D. Brummer. Labour migration and HIV/AIDS in Southern Africa. *International Organization for Migration Regional Office for Southern Africa*, 2002.

[3] G.P. Garnett. An introduction to mathematical models of sexually transmitted disease epidemiology. *Sexually Transmitted Infections*, **78**:7–12, 2002.

[4] C. W. Hunt. Social vs biological: theories on transmission of AIDS in Africa. *Social Science and Medicine*, **42**(9):1283–1296, 1996.

[5] J.A. Kelly and S.C. Kalichman. Behavioral research in HIV/AIDS primary and secondary prevention: Recent advances and future directions. *Journal of Consulting and Clinical Psychology*, **70**(3):626–639, 2002.

[6] M. Kgosimore and E.M. Lungu. The ffects of vertical transmission on the spread of HIV/AIDS in the presence of treatment. *Journal of Mathematicl Biosciences and Engineering*, **3**(2):297–312, 2006.

[7] M.S. Moghadas, A.B. Gumel, R.G. Mcleod and R. Gordon. Could condoms stop the AIDS epidemic. *Journal of Theoretical Medicine*, **5**:171–181, 2003.

[8] N.J. Nagelkerke, P. Jha, S. de Vlas, E. Korenromp, S. Moses, J. Blanchard and F. Plummer. *Modelling HIV/AIDS epidemics in Botswana and India: The effects of interventions*. CMH Working Papers Series, Paper No. WG5:4, 2001.

[9] R. Naresh and A. Tripathi. Modelling and analysis of HIV-TB co-infection in a variable size population. *Math. Model. Anal.*, **10**(3):275–286, 2005.

[10] F. Nyabadza. A mathematical model for combating HIV/AIDS in Southern Africa: Will multiple strategies work? *Journal of Biological Systems*, **14**:357–372, 2006.

[11] H.R. Thieme. Persistence under relaxed point-dissipativity (with applications to an endemic model). *SIAM Journal of Mathematical Analysis*, **24**:407–435, 1993.

[12] UNAIDS/WHO. *Report on the global AIDS epidemic 2006*. UNAIDS/WHO Report on the global AIDS epidemic 2006, Retreaved 25 July 2007 from www.unaids.org/en/HIV_data/2006GlobalReport

[13] S. Del Valle, A.M. Evangelista, M.C. Velasco, C.M. Kribs-Zaleta and S. Hsu Schimtz. Effects of education, vaccination and treatment on HIV transmission in homosexuals with genetic heterogeneity. *Mathematical Biosciences*, **187**:111–133, 2004.

[14] C. van Vliet, E.I. Meester, E. L. Korenromp, B. Singer, R. Bakker and J. Habbema. Focusing strategies of condom use against HIV in different behavioural strategies: an evalution based on simmulation model. *Bulletin of the World Health Organization*, **79**(5):442–453, 2001.

[15] D. Webb. *HIV and AIDS in Africa*. London: Pluto Press, 1997.