

Assessing Effect of Behaviour Change on HIV/AIDS Prevalence Using a Staged Progression Model

Mandidayingeyi H. Pfupajena¹, Chiedza E. Mashiri¹, Mashasha Maxwell¹, Vusimusi Mlilo¹, *Loyce Gonzo¹, Susan Maposa¹, Edina Chandiwana¹, Pauline Mukwembi¹.

¹Department of Mathematics, Midlands State University, Gweru, Zimbabwe

*Corresponding Author's email address: loygonzo@gmail.com

Abstract

A staged progression model was proposed that is an intervention model incorporating behaviour change. Analysis of the intervention model revealed that the disease free equilibrium point is locally stable when the bifurcation parameter $\beta < \beta^*$ near $R_0 = 1$. The endemic equilibrium point was shown to be locally stable using the centre manifold theorem and a Lyapunov function was used to show that the endemic equilibrium point is globally asymptotically stable. Results suggest that positive behaviour change is effective in reducing the HIV/AIDS prevalence for the first few years suggesting that positive behaviour change alone cannot be used effectively to control or eradicate HIV/AIDS. In the case of negative behaviour it was seen that negative behaviour change negatively impacts the population greatly during the first few years of the onset of the disease. However changes in patterns of HIV prevalence in Zimbabwe and most developing countries are quite recent and caution is required on the representativeness of the estimates and to note whether the observed changes are consistent with behaviour change and not the natural course of the HIV epidemic.

Keywords: HIV prevalence, behaviour change, endemic equilibrium, Lyapunov function

Introduction

Over the past quarter of a century the HIV virus has spread to all corners of the globe resulting in one of the deadliest epidemic of modern times. It is of particular concern in most developing countries as more often than not these face the problem of lack of resources and this makes the combating of the disease and its effects less probable. Not only do they face the problem of a depleted work force but there are also additional complications of orphaned children in need of care and protection. Women in developing countries

generally do not have a say in sexual matters resulting in their being unable to negotiate for safe sex practices. All this poses problems for policy implementers and decision making has to be carefully negotiated in the hope that whatever policies they come up with, work for the betterment of their countries. Sub-Saharan Africa continues to bear a disproportionate share of the global HIV burden. In 2007 a total of 2,1 million men, women and children died of AIDS. In mid 2010, about 68% of all people living with HIV resided in Sub-Saharan Africa, a region

constituting only 12% of the global population (UNAIDS, 2011). Zimbabwe has a projected population of about thirteen million and is among the Sub-Saharan African countries worst hit by the HIV/AIDS epidemic (NAC, 2012).

According to the last Zimbabwe Demographic Health Survey (ZDHS 2010-11), fifteen percent (15%) of Zimbabwean adults aged 15-49 are infected with HIV. In the 2005-6, the HIV prevalence rate for adults was 18 percent, thus the national HIV prevalence rate was estimated to have declined by three percentage points over the five-year period between the 2005-6 ZDHS and the 2010-11 ZDHS. However, Zimbabwe's HIV prevalence was pegged at 13, 7% in 2009 meaning there has been an increase to 15% following the completion of the last ZDHS (2010-11). Reports imply or suggest that HIV/AIDS prevalence rate is declining in Zimbabwe (Gregson *et al.*, 2010). Karen Stanecki a senior advisor in the UNAIDS epidemiology monitoring group stressed that the reduced figures in HIV prevalence rate were not a reflection of current conditions and that the present situation in Zimbabwe could reverse the trend (IRIN Africa, 2007). Among other factors, the decline in HIV incidence and prevalence can be attributed to sexual behaviour change. Many Zimbabweans have started to adopt safer sexual behaviours like condom use and having fewer sexual partners. These behavioural changes need to be maintained and adopted by all Zimbabwean adults and youth, married and unmarried, HIV positive and HIV negative (Zimbabwe National Behavioural Change Strategy 2006-2010).

In spite of the level of uncertainty that remains around the epidemic, it is clear

that HIV/AIDS epidemic is a major public health challenge that demands an effective policy response. Understanding the epidemic is an important step in planning and evaluating this response and research might help in this process. Understanding how socio-economic factors and behavioural change influence the course of the epidemic in Zimbabwe is also important and might help in understanding the impact of these various factors and in formulating strategies to combat the epidemic.

HIV/AIDS and Sexual Behaviour

HIV/AIDS has robbed many nations of its sons and daughters, wreaking havoc with people's lives. A lot of resources have been used to try and understand the dynamics of the disease. Researchers have looked at transmission paths as well as possible ways in which to combat the disease with the eventual hope of its complete eradication. The hope of complete eradication stems from the hope that people will realise the implications of their behaviour and how it will help in the fight against the disease. Successful combating of the disease has most usually been attributed to sexual behaviour change.

The HIV epidemic has devastated Africa and understanding behavioural responses to HIV rates is crucial both for predicting the future path of the epidemic and for preventing its future spread (Oster, 2009). Odu *et al.* (2008) reported a poor correlation between knowledge and sexual behaviour since knowledge has been shown not to be enough. Results from the study show that people practise unsafe sex despite

their knowledge of HIV/AIDS. Shumba et al. (2011) suggest that knowledge is not being translated into appropriate changes in sexual behaviour. Young men and women in universities in spite of their knowledge levels on HIV still maintain remarkably high levels of concurrent sexual partnerships. Gender and socio-economic status also play a role in determining one's sexual behaviour. UNAIDS (2010) statistics for Sub-Saharan Africa show that:

- More women than men are living with HIV in Sub-Saharan Africa accounting for 59% of people living with HIV.
- Young women aged 15-24 are as much as eight times likely than men of the same age to be living with HIV.
- Studies among women in Sub-Saharan Africa show that fear of a partner's negative reaction, including abandonment, violence, rejection, loss of economic support and accusations of infidelity were the most commonly reported barriers to HIV testing and disclosure of HIV status.

Shumba et al. (2011) also found out that females were drawn to concurrent relationships by the wealth and generosity of their partners and to obtain sexual satisfaction whilst males were attracted by beauty and the desire to acquire sexual experience. It has been postulated that unprotected paid sex was a more significant factor in early HIV epidemics in Sub-Saharan Africa. However, a review of 68 studies from 18 countries suggests that paid sex can remain an equally important factor in mature epidemics. Mobility can also affect sexual behaviour as it opens up one to certain vulnerabilities.

The Zimbabwean situation

Zimbabwe undertook the Modes of Transmission modelling in 2010, the model shows HIV transmission is pre-dominantly sexually driven, accounting for over 90 percent of new infections (NAC, 2012). People practising high risk sex are a major source of more than half the new HIV infection cases due to low condom use and high sexual networking (NAC, 2012). In view of these arguments it is important to consider behaviour change for the Zimbabwean case as the country has experienced some of the following:

- Spousal separation and mobility are major vulnerability factors in HIV infections and these can lead to risky behaviour as supported by surveys conducted by the Zimbabwean Demographic Health Services (NAC, 2006) and The Antenatal Care Survey of 2003 (NAC, 2006). The economic meltdown in the face of low income earnings saw a number of people leaving the country in search of greener pastures. An estimated 3.4 million Zimbabweans had fled the country by June 2007 (Meldrum, 2007). This in itself creates vulnerability between couples as they spend too much time apart.
- The advent of "Operation Murambatsvina" or "Restore Order" cost some 700 000 Zimbabweans their homes or livelihoods or both and otherwise affected nearly a fifth of the troubled country's population (Africa Report, 2005). This gave rise to an increase in urban to rural migration as most families were displaced thereby increasing the risk of transmission from urban dwellers to rural dwellers.

- Financial instability also contributes towards risky behaviour which may lead to commercial sex. Commercial sex is a facilitator or spreader of AIDS, (Tiruneh, 2009). The economic situation in Zimbabwe has seen the rate of unemployment increasing thereby resulting in financial instability.

Modelling HIV/AIDS dynamics

Salomon and Murray (2001) used back calculation techniques to come up with the HIV/AIDS prevalence rates for four Sub-Saharan African countries including Zimbabwe. These are techniques which reconstruct past patterns of HIV counts and estimate future AIDS counts (Onoja and Oyeloja, 2008). The methods depend on three key components, namely a model for the distribution of the infection, the assumed incubation period besides the observed counts of AIDS cases over time (Onoja and Oyeloja, 2008). The prevalence rate was estimated using EPI-MODEL which is a software programme developed by the World Health Organisation (WHO).

Dorrington and Schneider (2001) used the ASSA 2000 rural and urban model to project the HIV/AIDS prevalence rate in ten African countries including Zimbabwe. The ASSA 2000 rural and urban model is a population projection model which models the demographic impact of heterosexual HIV/AIDS epidemic.

Simwa et al., (2003) dealt with a deterministic model for HIV with three stages of disease progression among infected patients. Using two systems of ordinary differential equations that are coupled through a delay in one of the systems, a compartmental model for the dynamics of the HIV/AIDS epidemic is constructed.

Baryarama and Mugisha (2005) came up with a system of non-linear ordinary differential equations with a variable force of infection. A prevalence equation was formulated which through rearranging and factorisation reduced to a non-autonomous logistic equation which had an explicit solution subject to certain restrictions on the variable coefficients. Baryarama and Mugisha (2007) formulated a single staged model and a staged progression model with the same variable contact rate over time. In both models analytical expressions for the HIV prevalence were obtained. A comparison of the two models was undertaken, it was shown that prevalence from the single stage model were lower than the projections from the staged progression model up to and beyond the peak prevalence.

Johnson et al. (2009) state that mathematical models can be used to assess the likely effects of behavioural change and can play an important role in determining whether observed changes are attributable to changes in sexual behaviour or to some other factors. A key conclusion from the analysis is that concurrent partnerships play a major role in the epidemiology of HIV in South Africa, accounting for three quarters of new HIV infections over the 1990-2000 period.

Methods and Techniques

We modelled the HIV prevalence rate for Zimbabwe using a deterministic staged progression intervention model. The intervention staged progression model incorporates behaviour change. Stability analysis of the computed equilibrium points was carried out. In order to validate the proposed model and analytical results, numerical simulations were carried out. The system of differential equations developed was considered

as initial value problems when the numerical simulations were being done. The numerical method that was used for their solution was a method developed by two German mathematicians, Runge and Kutta. In particular the Fourth-order Runge-Kutta method was used.

Results

Model formulation

The sexually active population is first divided into two risk groups i.e. low risk and high risk group. The low risk group represented by L and the high risk group by H . Risk is perceived in terms of sexual behaviour. Those in the high risk group represent the sexually mature who engage in multiple sexual relationships which can be one of two forms or a mixture of both sequential or serial monogamy and concurrent sexual relationships. Whilst those in the low risk group do not engage in multiple sexual relationships. The sexually active population is then classified into six compartments, high risk susceptibles $S_H(t)$, low risk susceptible $S_L(t)$, high risk infectives $I_H(t)$, low risk infectives $I_L(t)$, pre-AIDS class $P(t)$ and AIDS class $A(t)$. The sexually active are recruited at a rate b . A proportion π enter the low risk susceptible class whilst the remaining proportion $(1 - \pi)$ enter the high risk susceptible class. The susceptible in both risk groups have no contact with the virus but are at risk of getting infected. These compartments increase through the maturation of individuals into the sexually mature age group and decreases due to contagion at a time dependent rate λ going into the low risk and high risk infectives compartments respectively. People may change risk groups whilst in the susceptible compartments or later in the infectives compartments. These rates at which people change risk groups is assumed to be the same at both susceptible and infectives level. Rate of changing from low risk group to high risk group (negative change) is represented by e_1 whilst that of changing from high risk group to low risk group (positive change) is represented by e_2 . Negative change may be due to financial instability, denial by an individual that they could be at risk of acquiring the disease, peer pressure and so on. Positive change may be due to behavioural interventions at either group or individual level in the form of educational messages. Both the low risk and high risk infectives will show AIDS symptoms at a rate ρ_1 . α_1 is the rate at which low risk infectives decrease by getting into the pre-AIDS class and α_2 is the rate at which high risk infectives decrease by getting into the pre-AIDS class, ($\alpha_2 > \alpha_1$). Those in the AIDS class will show AIDS symptoms at a rate ρ_2 . Each compartment has a constant natural death rate μ , proportional to the number in each compartment. Those in the AIDS class have an additional disease induced mortality rate δ . The total variable population size is given by

$$N(t) = S_L(t) + S_H(t) + I_L(t) + I_H(t) + P(t) + A(t) \quad (1)$$

$A(t)$ is redundant since its assumed that those in the AIDS class do not contribute to the infection. The force of infection λ depends on the probability of transmission per partnership

β for high risk infectives, $\beta\eta_1$ for low risk infectives and $\beta\eta_2$ for those in the pre-AIDS class. Without loss of generality its safe to assume that $\beta\eta_2 < \beta\eta_1 < \beta < 1$. The infection rate is given by;

$$\lambda = \frac{\beta I_H + \beta\eta_1 I_L + \beta\eta_2 P}{N} \tag{2}$$

Model assumptions

- population is large enough to be compartmentalised.
- interaction is of standard form.
- transmission of disease is through heterosexual sex.
- those in the AIDS class are sexually inactive and do not contribute towards infection.

The model parameters and variables are defined in Tables 1 and 2.

Table 1: Description of variables of the basic model.

Variable Description

Variable	Description
N	adult population
S _L	susceptible individuals in the low risk group
S _H	susceptible individuals in the high risk group
I _L	infected individuals in low risk group who have not progressed to pre-AIDS stage
I _H	infected individuals in high risk group who have not progressed to pre-AIDS stage
P	infected individuals in the pre-AIDS stage
A	infected individuals with AIDS symptoms

Table 2: Description of the parameters of the basic model.

Parameter	Description
b	recruitment rate of susceptibles
μ	natural mortality rate
δ	AIDS induced mortality rate
π	proportion of newly recruited individuals who enter S _L
1 - π	proportion of newly recruited individuals who enter S _H
α_1	progression rate for low risk group infectives to pre-AIDS class
α_2	progression rate for high risk group infectives to pre-AIDS class
ρ_1	progression rate to AIDS class for infectives in I _L and I _H
ρ_2	progression rate to AIDS class for infected individuals in P
β	transmission coefficient
η_1	modification parameter for I _L
η_2	modification parameter for P

Since the model system monitors human population all state variables are non-negative. The model system of equations are independent of therefore along with the model assumptions this results in the following system of equations;

$$\frac{dS_L}{dt} = b\pi + e_2S_H - (e_1 + \mu)S_L - \lambda S_L \quad (3)$$

$$\frac{dS_H}{dt} = b(1 - \pi) + e_1S_L - (e_2 + \mu)S_H - \lambda S_H \quad (4)$$

$$\frac{dI_L}{dt} = \lambda S_L + e_2I_H - (e_1 + \mu + \alpha_1 + \rho_1)I_L \quad (5)$$

$$\frac{dI_H}{dt} = \lambda S_H + e_1I_L - (e_2 + \mu + \alpha_2 + \rho_1)I_H \quad (6)$$

$$\frac{dP}{dt} = \alpha_1I_L + \alpha_2I_H - (\mu + \rho_2)P \quad (7)$$

$S_L(0) > 0, S_H(0) > 0, I_L(0) \geq 0, I_H(0) \geq 0$ and $P(0) \geq 0$. The total population $N(t)$ will now be given by $N(t) = S_L(t) + S_H(t) + I_L(t) + I_H(t) + P(t)$ (8)

Model analysis

Theorem 1 (positivity of solutions): *The system of equations 3-7 preserve positivity i.e. the solutions $S_L(t), S_H(t), I_L(t), I_H(t), P(t)$ of this system are positive $\forall t \geq 0$*

From 3 we have,

$$\frac{dS_L}{dt} \geq -(\lambda + \mu + e_1)dt \quad (9)$$

$$\int \frac{dS_L}{S_L} \geq -\int (\lambda + \mu + e_1)dt \quad (10)$$

$$\ln S_L \geq B - ((\mu + e_1)t + \int \lambda dt) \quad (11)$$

$$S_L(t) \geq Ce^{-((\mu+e_1)t + \int \lambda dt)} \quad (12)$$

Applying initial conditions yields, $S_L(t) \geq S_L(0)e^{-((\mu+e_1)t + \int \lambda dt)}$ (13)

Since $e^{-((\mu+e_1)t + \int \lambda dt)} > 0$ this implies that $S_L(t)$ is always positive. The same can be done for the remaining equations, therefore the system of equations is always positive.

Invariance/boundedness of solutions

From equation system 3-7 the time derivative of N along a solution path of the system gives:

$$\frac{dN}{dt} = b - \mu N - \rho_1(I_H + I_L) - \rho_2 P \tag{14}$$

$$\frac{dN}{dt} \leq b - \mu N \tag{15}$$

$$\int \frac{d(e^{\mu t} N)}{dt} \leq \int b e^{\mu t} dt \tag{16}$$

$$N \leq \frac{b}{\mu} + C e^{-\mu t} \tag{17}$$

Applying initial conditions for $t = 0$, we get $C = N(0) - \frac{b}{\mu}$. Therefore, for $t \geq 0$, thus as

$t \rightarrow \infty, 0 \leq N \leq \frac{b}{\mu}$, and we assume without loss of generality that the population has reached

its limiting value of $\frac{b}{\mu}$. therefore all feasible solutions of the system enter this region which is positively invariant. Existence, uniqueness and continuity results for the system will hold in this region. Therefore the system is epidemiologically well posed.

Equilibria: existence and local stability

At equilibrium point $\frac{dS_L}{dt} = \frac{dS_H}{dt} = \frac{dI_L}{dt} = \frac{dI_H}{dt} = \frac{dP}{dt} = 0$ where in terms of the

equilibrium value of the force of infection λ^* we have,

$$S_H^* = \frac{b[e_1 - G]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)} \tag{18}$$

$$S_L^* = \frac{b[J + e_2]}{(\lambda^* + \mu + e_1 + e_2)} \tag{19}$$

$$I_H^* = \frac{b\lambda^*[Q_1 G - e_1[J + e_2 + Q_1]]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1 e_2 - Q_1 Q_2)} \tag{20}$$

$$I_L^* = \frac{b\lambda^*[e_2[G-e_1]-Q_2[J+e_2]]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1e_2 - Q_1Q_2)} \quad (21)$$

$$P^* = \frac{b\lambda^*[e_2[G-e_1]-\alpha_1[e_2+Q_2J]+Q_1G-e_1\alpha_2J+\mu-e_2-Q_1]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1e_2 - Q_1Q_2)} \quad (22)$$

Where,

$$\lambda^* = \frac{\beta I_H^* + \beta \eta_1 I_L^* + \beta \eta_2 P^*}{N^*} \quad (23)$$

$$Q_1 = (\mu + \alpha_1 + \rho_1 + e_1) \quad (24)$$

$$Q_2 = (\mu + \alpha_1 + \rho_1 + e_2) \quad (25)$$

$$Q_3 = (\mu + \rho_2) \quad (26)$$

$$G = (\lambda^* + \mu)(-1 + \pi) \quad (27)$$

$$J = \pi(\lambda^* + \mu) \quad (28)$$

At disease free equilibrium (DFE), which we will denote by ε_0 , $\lambda^* = 0$ in the system of equations 3-7 substituting for $\lambda^* = 0$ yields DFE given by;

$$\varepsilon_0 = \left(S_L(0), S_H(0), I_L(0), I_H(0), P(0) = \frac{b(\mu\pi + e_2)}{\mu(\mu + e_1 + e_2)}, \frac{b(\mu - \mu\pi + e_1)}{\mu(\mu + e_1 + e_2)}, 0, 0, 0 \right) \quad (29)$$

The linear stability of is governed by the basic reproduction number , defined as the expected number of secondary infections produced by a single case in a wholly susceptible population. Using the Driessche-Watmough method we obtain as;

$R_0 = R_L + R_H + R_P$, where

$$R_L = \left(\frac{e_2}{Q_2} \right) \frac{\beta S_H^*}{Q_1 \left(1 - \frac{e_1 e_2}{Q_1 Q_2} \right)} + \frac{\beta S_L^*}{Q_1 \left(1 - \frac{e_1 e_2}{Q_1 Q_2} \right)} \quad (30)$$

$$R_H = \frac{\beta \eta_1 S_H^*}{Q_2 \left(1 - \frac{e_1 e_2}{Q_1 Q_2} \right)} + \left(\frac{e_1}{Q_1} \right) \frac{\beta \eta_1 S_L^*}{Q_2 \left(1 - \frac{e_1 e_2}{Q_1 Q_2} \right)} \quad (31)$$

$$\begin{aligned}
 R_p = & \left(\frac{e_1}{Q_1}\right)\left(\frac{e_2}{Q_2}\right)\frac{\beta\eta_2 S_H^*}{Q_3\left(1-\frac{e_1 e_2}{Q_1 Q_2}\right)} + \left(\frac{\alpha_1}{Q_1}\right)\frac{\beta\eta_2 S_L^*}{Q_3\left(1-\frac{e_1 e_2}{Q_1 Q_2}\right)} + \left(\frac{\alpha_2}{Q_2}\right)\frac{\beta\eta_2 S_H^*}{Q_3\left(1-\frac{e_1 e_2}{Q_1 Q_2}\right)} \\
 & + \left(\frac{e_1}{Q_1}\right)\left(\frac{\alpha_2}{Q_2}\right)\frac{\beta\eta_2 S_L^*}{Q_3\left(1-\frac{e_1 e_2}{Q_1 Q_2}\right)} \quad (4.48)
 \end{aligned}$$

(32)

These are written in such a way that one can easily trace the contribution of each of the infected classes to HIV transmission. If $R_0 < 1$, DFE is locally asymptotically stable and unstable otherwise. Looking for the existence of multiple endemic equilibrium for the system of equations 3-7 we obtain that the endemic equilibrium point is given by ξ_1 where,

$$\xi_1 = (S_H^*, S_L^*, I_H^*, I_L^*, P^*) \quad (33)$$

$$S_H^* = \frac{b[e_1 - G]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)} \quad (34)$$

$$S_L^* = \frac{b[J + e_2]}{(\lambda^* + \mu + e_1 + e_2)} \quad (35)$$

$$I_H^* = \frac{b\lambda^*[Q_1 G - e_1[J + e_2 + Q_1]]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1 e_2 - Q_1 Q_2)} \quad (36)$$

$$I_L^* = \frac{b\lambda^*[e_2[G - e_1] - Q_2[J + e_2]]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1 e_2 - Q_1 Q_2)} \quad (37)$$

$$P^* = \frac{b\lambda^*[e_2[G - e_1] - \alpha_1[e_2 + Q_2 J] + Q_1 G - e_1 \alpha_2 J + \mu - e_2 - Q_1]}{(\lambda^* + \mu)(\lambda^* + \mu + e_1 + e_2)(e_1 e_2 - Q_1 Q_2)} \quad (38)$$

Substituting the above in the force of infection then has to satisfy the following algebraic equation which is biologically significant if its discriminant is greater than zero.

$$\lambda^*(a\lambda^{*2} + b\lambda^* + c) = 0 \tag{39}$$

λ^*
equal to zero gives DFE. Solving the part in bracket yields

$$a = -Q_1Q_3 - \pi Q_2Q_3 - \pi\alpha_1Q_2 - \pi e_1(Q_3 + \alpha_2) + \pi Q_1Q_3 + (Q_3 + \alpha_1)(\pi - 1)e_2 + (\pi - 1)Q_1\alpha_2 \tag{40}$$

$$b = J_1 - J_2 \tag{41}$$

$$J_1 = \beta\mu\pi e_1 Q_3 + \beta e_1 e_2 Q_3 + \beta\mu Q_1 Q_3 + \beta\mu\eta_1 e_2 Q_3 + \beta e_1 Q_1 Q_3 + \beta\eta_1 e_1 e_2 Q_3 + \beta\mu\eta_1 Q_2 Q_3 + \beta\eta_1 e_2 Q_2 Q_3 + \beta\mu\alpha_1\eta_2 e_2 + \beta\alpha_1\eta_2 e_1 e_2 + \beta\mu\alpha_1\eta_2 Q_2 + \beta\alpha_1\eta_2 e_2 Q_2 + \beta\mu\alpha_2\eta_2 e_2 + \beta\mu\alpha_2\eta_2 Q_1 + \beta\alpha_2\eta_2 e_1 Q_1$$

$$J_2 = \beta\mu\pi e_1 Q_1 Q_3 + \beta\mu\pi\eta_1 e_2 Q_3 + \beta\mu\alpha_1\eta_2 e_2 + \beta\mu\alpha_2\eta_2 Q_1$$

And

$$c = Q_1Q_2Q_3(\mu + e_1 + e_2)(K - 1) \tag{42}$$

Where ,

$$K = \frac{e_1 e_2 (\mu + e_1)}{Q_1 Q_2 (\mu + e_1 + e_2)}$$

If c is positive and K is greater than 1 we have a unique endemic equilibrium point, if b is positive and K is less than 1 we have two endemic equilibrium points and discriminant is greater than zero and b is greater than zero we have one endemic equilibrium point.

The necessary condition that if the basic reproduction number is less than one then DFE is locally asymptotically stable is not sufficient for global disease control. There is stability giving rise to backward bifurcation a situation where a stable endemic state co-exists with DFE when $R_0 < 1$. In particular when $R_0 = 1$ it is expected that the disease will be able to invade in the case of a backward bifurcation. An analysis of the local centre manifold yields a simple criterion for the existence and stability of super and sub threshold endemic equilibria for R_0 near 1. The results are significant for disease control. Since it will also be complicated to analyse the stability of the endemic equilibrium point (EEP), we employ the

centre manifold theorem. Following the work of (Buonomo and Lacitgnola, 2011), focusing on the DFE and investigating the occurrence of trans-critical bifurcation at. We let β be the bifurcation parameter for the local stability of the endemic equilibrium and we solve for β^* . We obtain,

$$\beta^* = \beta$$

$$\beta^* = \frac{\mu Q_1 Q_2 Q_3 \left(1 - \frac{e_1 e_2}{Q_1 Q_2}\right) (\mu + e_1 + e_2)}{b(\mu - \mu\pi + e_1)M + b(\mu\pi + e_2)N} \quad (43)$$

Where,

$$M = (Q_3 e_2 + Q_1 Q_3 \eta_1 + \alpha_1 e_2 \eta_2 + Q_1 \alpha_2 \eta_2)$$

$$N = (Q_2 Q_3 + Q_3 e_1 \eta_1 + Q_2 \alpha_1 \eta_2 + e_1 \alpha_2 \eta_2)$$

It follows that the DFE is locally stable when $\beta < \beta^*$ whereas it loses its stability when $\beta > \beta^*$.

Global stability of EEP

To establish the global stability of the EEP we will employ the method summarised in the theorem given below which states that:

Theorem 2: Let U be a Lyapunov function defined as

$$U = \frac{(S_L - S_L^*)^2}{2} + \frac{\delta_1}{2} (S_H - S_H^*)^2 + \frac{\delta_2}{2} (I_L - I_L^*)^2 + \frac{\delta_3}{2} (I_H - I_H^*)^2 + \frac{\delta_4}{2} (P - P^*)^2 \text{ where}$$

$\delta_1, \delta_2, \delta_3, \delta_4 > 0$ are to be chosen properly such that $U'(E) = 0$ where

$E = (S_L^*, S_H^*, I_L^*, I_H^*, P^*)$ and $U = (S_L, S_H, I_L, I_H, P) > 0$ for all (S_L, S_H, I_L, I_H, P) ,

$P \in \{E\} \frac{dU}{dt} = 0$ implies that E^* of the system is Lyapunov stable and $\frac{dU}{dt} < 0$ implies

that E^* is globally stable.

Thus it is possible to set δ_i such that and the endemic equilibrium is globally stable.

Prevalence

Following the work of (Baryarama and Mugisha, 2007) we define prevalence as the fraction of infectives in the population excluding those with AIDS symptoms i.e.

$$\frac{I_L + I_H + P}{N}$$

We let $\phi_{sp} = \frac{I_L}{N} + \frac{I_H}{N} + \frac{P}{N}$ be the HIV prevalence in the HIV/AIDS model with risk groups.

Then

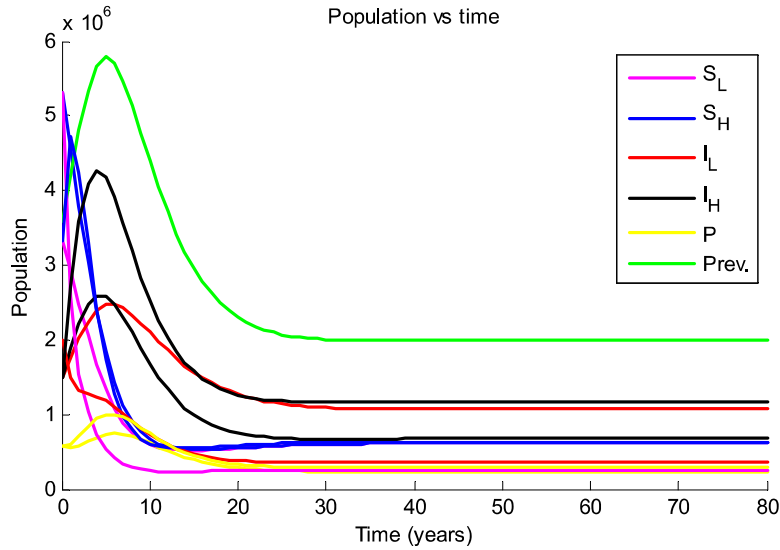
$$\frac{d\phi_{sp}}{dt} = \frac{N \left(\frac{dI_L}{dt} \right) - I_L \left(\frac{dN}{dt} \right)}{N^2} + \frac{N \left(\frac{dI_H}{dt} \right) - I_H \left(\frac{dN}{dt} \right)}{N^2} + \frac{N \left(\frac{dP}{dt} \right) - P \left(\frac{dN}{dt} \right)}{N^2} \quad (44)$$

$$\begin{aligned} \frac{d\phi_{sp}}{dt} &= \frac{1}{N} \left(\frac{dI_L}{N} + \frac{dI_H}{N} + \frac{dP}{N} \right) - \frac{1}{N} \frac{dN}{dt} \left(\frac{I_L}{N} + \frac{I_H}{N} + \frac{P}{N} \right) \\ &= \frac{1}{N} [e_2 I_H + \lambda S_L - Q_1 I_L + e_1 I_L + \lambda S_H - Q_2 I_H + \alpha_1 I_L + \alpha_2 I_H - Q_3 P] \\ &\quad - \frac{1}{N} [(b - \mu N - e_1 (I_L + I_H) - e_2 P) \phi_{sp}] \\ &= \frac{1}{N} [\lambda (S_L + S_H) + (e_1 + \alpha_1) I_L + (e_2 + \alpha_2) I_H - Q_1 I_L - Q_2 I_H - Q_3 P] \\ &\quad - \frac{1}{N} [(b - \mu N - e_1 (I_L + I_H) - e_2 P) \phi_{sp}] \end{aligned}$$

Numerical simulations

For validation numerical simulations for the proposed model were done. Movement within risk groups will be used to describe behaviour change. For parameter values used refer to table 1. The total population of Zimbabwe as of 1997 given by $N = 12.23$ million is used, (NAC, 2012). Initial conditions for the sub-populations are as follows, $S_L = 3323305$, $S_H = 5323305$, $I_L = 1500000$, $I_H = 1500000$, $P = 583390$. Thus the total infected population is 3583390. The recruitment rate into the sexually active population will be taken as $0.032N$, (Baryarama and Mugisha, 2007). Simulations were done using Matlab 7.1.

Assessment of no behaviour change



In the absence of behaviour change it was noted that as the susceptible population declines, the infectives population increases. The infectives and prevalence curves peak initially for about six years and then start to decline, levelling off around the twentieth year.

Assessment of positive behaviour change

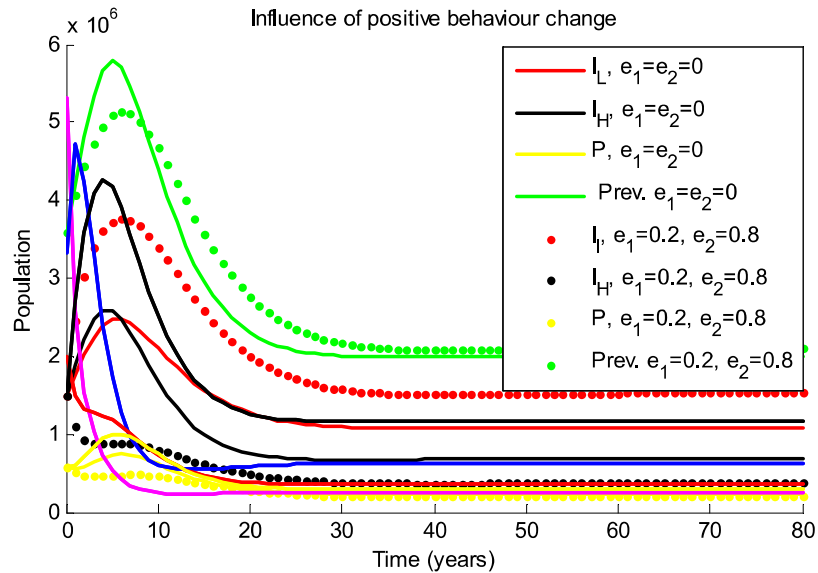


Figure 2: Influence of positive behaviour change

Positive behaviour change occurs when the proportion of individuals in the high risk groups transferring to the low risk groups is more than the proportion of individuals in the low risk groups transferring to the high risk groups (i.e. $e_2 > e_1$). In such a scenario it can be seen that for the first nine years the prevalence rate for no behaviour change is higher than that for positive behaviour change. However from year ten onwards the prevalence rate for positive behaviour change becomes higher.

Assessment of negative behaviour change

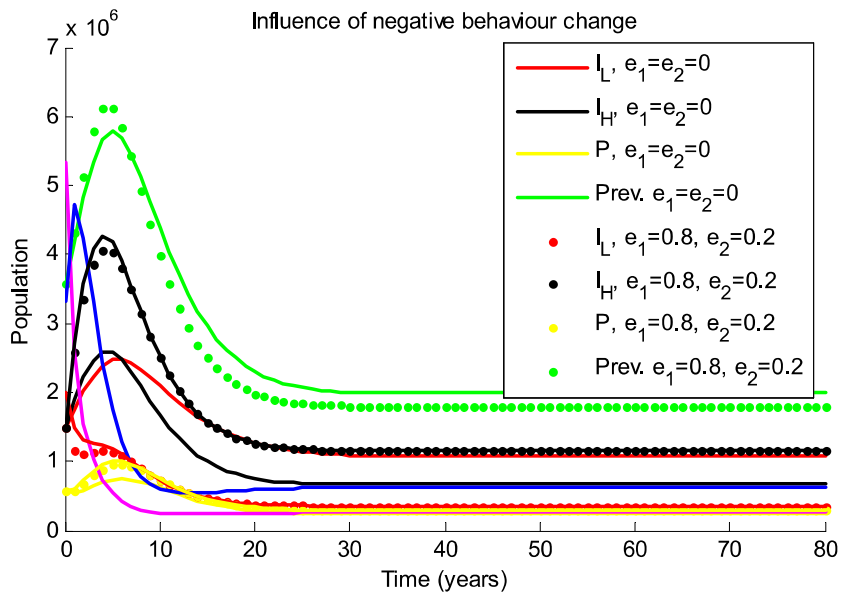


Figure 3: Influence of negative behaviour change.

Negative behaviour change occurs when the proportion of individuals in the low risk groups transferring to the high risk groups is more than the proportion of the individuals in the high risk groups transferring to the low risk groups (i.e. $e_1 > e_2$). In such a scenario for the first seven years the prevalence rate for negative behaviour change is higher than that for no behaviour change. However from year eight onwards the prevalence rate for negative behaviour change becomes lower than that for no behaviour change.

Impact of behaviour change on the reproduction numbers

Table 3: Comparison of reproduction numbers.

Conditions	Reproduction number
$e_1 = e_2 = 0$	$R_0 = 4.8067$
$e_1 = 0.8, e_2 = 0.2$	$R_{en} = 4.5924$
$e_1 = 0.2, e_2 = 0.8$	$R_{ep} = 4.2791$

The basic reproduction number is given by $R_0 = R_L + R_H + R_P$. Using the aforementioned parameters in table 1 and $\pi = 1$ we compare R_0 and the effective reproduction numbers for both positive and negative behaviour change given by R_{ep} and R_{en} respectively, we note that R_{ep} is less than and is greater than . Implication being that behaviour change has an impact on the spread of the disease, with positive behaviour change having a positive impact on the spread since it has the least value.

Discussion and Conclusion

Results suggest that positive behaviour change is effective in reducing the HIV/AIDS prevalence rate for the first nine years suggesting that positive behaviour change alone cannot be used effectively to control or eradicate HIV/AIDS. This is further supported by the value of, though it has the lowest value as compared with the other reproduction numbers it is still much greater than 1 thereby implying that positive behaviour change alone cannot be relied on as a means

of controlling or eradicating HIV/AIDS. However in the case of negative behaviour change HIV/AIDS prevalence tends to be higher than that for no behaviour change for the first seven years meaning that negative behaviour change negatively impacts the population during the first few years of the onset of the disease. Ultimately as people get used to a certain scenario they become complacent with time and might not adhere to strict behavioural practices. Behaviour change has impact for a few years therefore it needs to be coupled with other intervention strategies to be effective over time.

References

Africa Report. 2005. Zimbabwe’s operation Murambatsvina: the tipping point? Report no 97.

BARYARAMA, F., MUGISHA, J.Y.T. 2007. Comparison of single staged and staged progression models for HIV/AIDS. The International Journal of Mathematics and Mathematical Sciences.

BARYARAMA, F, MUGISHA, J.Y.T., LUBOOBI, L., (2005). An HIV/AIDS model with variable

- force of infection and its application to the epidemic in Uganda. *American Journal Of Applied Sciences*.
- BARYARAMA, F., MUGISHA, J.Y.T., LUBOOBI, L. 2006. A mathematical model for the dynamics of HIV/AIDS with gradual behaviour change *Computational and Mathematical Methods in Medicine*,7:1,15-26.
- BUONOMO, B., LACITGNOLA, D. 2008. On the backward bifurcation of a vaccination model with non linear incidence *Non-Linear Analysis: Modelling and Control*, 2011 vol. 16, No 1, 30-46.
- DORRINGTON, R., SCHNEIDER, D. 2001. Fitting the ASSA 2000 urban - rural AIDS and demographic model to 10 sub Saharan countries.
- GREGSON ET AL. 2010. HIV decline in Zimbabwe due to reductions in risky sex? Evidence from a comprehensive epidemiological review. *International Journal of Epidemiology*, 1-13.
- IRIN Africa. 2007. Zimbabwe HIV prevalence decline will it last? [www.irinnews.org / report.aspx?reportid=61177](http://www.irinnews.org/report.aspx?reportid=61177), 12/06/2014.
- JIWO, S. 2009. Evaluating the potential impact of condoms and vaginal microbicides to reduce the spread of HIV.
- JOHNSON ET AL. 2009. Sexual behaviour patterns in SA and their association with the spread of HIV: Insights from a mathematical model *Demographic research* vol. 21 Article 11, 289-340.
- MELDRUM, A. 2007. Refugees flood from Zimbabwe. *The Guardian*.
- National AIDS Council, NAC. 2006. Zimbabwe National HIV and AIDS strategic plan , (ZNASP) 2006-2010.
- National AIDS Council, NAC. 2012. Global AIDS Response Progress Report.
- ODU, B.K., AKANLE, F.F. 2008. Knowledge of HIV/AIDS and sexual behaviour among the youths in South west Nigeria *Human and Social Sciences Journal* 3(1):81-88.
- ONOJA, M.A., OYELOJA, B.A. 2008. Statistical modeling of HIV/AIDS epidemic in north central zone of Nigeria *The Internet Journal Of Infectious Diseases* ISSN:1528-8366.
- OSTER, E. 2014. HIV and sexual behavior change: Why not Africa? www.home.uchicago.edu/hivbehaviour.pdf.
- SHUMBA, A., MAPFUMO, J., CHADEMANA, J. 2011. The prevalence of concurrent sexual partnerships among students in institutions of higher education in Zimbabwe *J Human Ecol*, 34(1):53-5.
- SALOMON, J.A., MURRAY J.L., (July 2001). Modelling HIV/AIDS epidemic in Sub-Saharan Africa using sero-prevalence data from Ante-Natal Clinics *Bulletin of WHO* vol 79, no 7, Geneva.
- TIRUNEH, G. 2009. Determinants of adult HIV/AIDS prevalence in Africa. Do cultural variations matter? *Midsouth Political Science Review* Vol. 10.

- UNAIDS Zimbabwe Country Page:www.unaids.org/en/geographical+area/by+c, 25/06/2015.
- VAN DRIESSCHE, P., WATMOUGH, J., Reproduction numbers and sub threshold endemic equilibria for compartmental models of disease transmission.
- ZIMBABWE NATIONAL AIDS COUNCIL (NAC). 2006. The National Behavioural Change Strategy for Prevention of Sexual Transmission of HIV 2006-2010.
- ZIMBABWE NATIONAL STATISTICS AGENCY (ZIMSTAT) AND ICF INTERNATIONAL. 2012. Zimbabwe Demographic and Health Survey 2010-11. Calverton, Maryland: ZIMSTAT and ICF International1.Inc.