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Comparative Analysis of HIV/AIDS Control Strategies with Mathematical Models

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Abstract In this paper, deterministic and stochastic differential equation models of the vertical/heterosexual transmission dynamics of HIV/AIDS in a population are formulated. The objectives are, first, to compare the effects of condom use, antiretroviral therapy (ART) separately and the combination of both as control strategies; and second, to compare the performance of the two models. The models were solved numerically for varying values of the control parameters and fixed published data. The numerical results show that ART outweighs condom use in performance, with the effective combination of both as a control strategy the best. At the level of model performance, both models compete favorably well, especially, when there is no or little control. However, under effective control strategy, either at ART or condom use level or a combination of both, the sample paths of the stochastic differential equation model demonstrate strong components of stochasticity as some paths tend to the disease-free equilibrium point while the trajectory of the deterministic model shows disease growth. The findings in this study that effective combination of ART and condom use as a control strategy are crucial for the control of HIV/AIDS.

Keywords HIV/AIDS, antiretroviral therapy, condom use, deterministic model, stochastic model, stochastic differential equations, Wiener process and numerical simulation

Introduction

The human immune-deficiency virus (HIV) together with the associated acquired immune deficiency syndrome (AIDS) is still a monster [1-2]. Cumulatively, up to 50.6 million people now suffer from HIV/AIDS across the globe. About 34 million people were living with HIV/AIDS in 2010 (estimates range from 30.9 to 36.9 million) and about 1.8 million people die annually due to opportunistic infections and diseases. This is 0.5% of the world population. 68% of these live in sub-Saharan Africa. HIV/AIDS affects mostly people in the economically productive age range, reducing the work-force, and thereby, constraining development [3].

The basic routes of HIV transmission between persons are well understood. The major routes are through unprotected sexual intercourse (heterosexual and homosexual), mother- to-child or vertical transmission (at birth or through breastfeeding), unsafe injections in medical care, unsafe blood transfusions and shared injection equipment in injectable drug use [2-4].

Several intervention methods are available. These range from sex abstinence, use of condoms, education and use of antiretroviral drugs and counseling. These control measures outdo one another in performance.

Condom use, as an intervention, prevents transmissions that are sexual in nature but is limited in the sense that it does not shield the fetus or the baby against vertical transmission.

As pointed out in Williams et al (2011), the development of antiretroviral drugs to treat HIV has been a singular scientific achievement. Between 1995 and 2009 an estimated 14.4 million life-years has been gained globally among adults on ART but the rate of new infections is unacceptably high and still exceeds the number of people starting ART each year [2].

As presented in casels *et al* (2008), ART reduces viral load and the probability of transmission. It also reduces HIV/AIDS-related mortality and, therefore, increases the life expectancy of infected individuals [3].

Many mathematical models of HIV/AIDS are available. For a survey, see Sani *et al* (2006) [5]. Stochastic models of HIV have been proposed and studied by researchers. For example, Peterson et al (1990) applied Monte-Carlo simulation technique in a population of intravenous drug users [6].

Greenhalgh and Hay (1997) studied a mathematical model of the spread of HIV/AIDS among injecting drug users [7]. Dalal *et al* (2007) examined a stochastic model of AIDS and condom use [8]. Dalal, *et al* (2008) also studied a stochastic model for internal HIV dynamics [9]. Ding *et al* (2009) carried out risk analysis for AIDS control based on a stochastic model with treatment rate [10]. Tuckwell and Le Corfec (1998) studied a stochastic model for early HIV-1 population dynamics [11]. Waema and Olowofeso (2005) studied a mathematical model for HIV transmission using generating function approach [12].

In this paper, the deterministic model proposed by Kimbir *et al* (2008) is the focus. We incorporate the term for mother-to-child transmission to obtain an extended deterministic model. We further formulate the stochastic counterpart of our extended model [13].

The plan of this paper is as follows. Introductory part is presented in section 1. The deterministic model is presented in section 2. The stochastic counterpart of the deterministic model is developed in section 3. Numerical simulations are carried out in section 4. Discussion and conclusive remarks are passed in sections 5 and 6 respectively.

Formulation of the Deterministic Model

Kimbir and his collaborators proposed their model based on the following assumptions. The population is partitioned into three compartments: the number of susceptible individuals $S(t)$, the number of infected individuals $I(t)$ and the number of people receiving ART $R(t)$. It is assumed that recruitment into the S-compartment occurs at the rate bN. Death occurs in all the compartments at the rate μ. In addition, there is AIDS-related death which occurs at the rate α_0 in I-compartment. Infected individuals are treated at the rate δ. Members of the R-compartment die due to AIDS at the rate α. With the availability of free diagnosis, we assume that people are now aware of their HIV status and consequently the infected individuals should now go for antiretroviral therapy and counseling. People that have developed full-blown AIDS symptoms cannot transmit. Based on the above assumptions, Kimbir *et al* (2008) formulated the following model [13].

$$\begin{aligned}\frac{dS}{dt} &= bN - B(t)S - \mu S,\\ \frac{dI}{dt} &= B(t)S - (\mu + \alpha_0 + \delta)I, \qquad (1)\\ \frac{dR}{dt} &= \delta I - (\mu + \alpha)R,\end{aligned}$$

where $N(t) = S(t) + I(t) + R(t)$.

The incidence rate is given by $B(t) = \frac{c\beta I + c'\beta' R}{N}$, where β is the probability of transmission by an individual in I-compartment, c is the number of sex partners for each member of the I-class, β' and c'.

The Extended Deterministic Model

Let ω represent the proportion neonates perinatally infected. We now incorporate this term for mother-to-child transmission to obtain an extended deterministic model in the sequel.

$$\begin{aligned}\frac{dS}{dt} &= b(1-\omega)N - B(t)S - \mu S,\\ \frac{dI}{dt} &= b\omega N + B(t)S - (\mu + \alpha_0 + \delta)I, \qquad (2)\\ \frac{dR}{dt} &= \delta I - (\mu + \alpha)R,\end{aligned}$$

where $N(t) = S(t) + I(t) + R(t)$.

All other terms and parameters are as in model (1)

Stochastic Model of HIV/AIDS Transmission Dynamics

In order to obtain the Corresponding stochastic differential equation (SDE) model for system (2), we need to identify the forgoing deterministic model as a birth, death and migration process. This SDE formulation assumes there is demographic variability in births, deaths and migrations.

Let X_1, X_2, X_3 denote random variables for the numbers of susceptible, infected individuals and infected individuals on antiretroviral drug. Let the incremental changes in susceptible, infected and infected individuals on ART during the small time period Δt be denoted by Δx_1, Δx_2 and Δx_3 respectively. Then we define the vector Δx as

$\Delta x(t) = (\Delta x_1(t), \Delta x_2(t), \Delta x_3(t))^T$,

Where $\Delta x_i(t) = x_i(t + \Delta t) - x_i(t)$ for $i = 1, 2$ and 3.

The mean and the covariance matrix of Δx have the forms $E(\Delta x) = \mu \Delta t$ and $V(\Delta x) = C\Delta t$, respectively, where C is a positive definite matrix. From the work done by Allen et al (2008), an explicit form for the matrix $B = \sqrt{C}$ exists for a given positive definite matrix C of order 2. Furthermore, the method of obtaining such a square root matrix is explained in Allen (2008). Then, the SDE model of the deterministic model of interest can be obtained in the form of $dx = \mu dt + Bdw$, where μ and $B = \sqrt{C}$ are defined as above. The variable $W = W(t)$ is a three dimensional wiener process. The notation

$dW = (dW_1, dW_2, dW_3)^T$ denotes the differential of the three dimensional Wiener process, because the wiener process is continuous but not differentiable. The incremental change in the wiener process satisfies.

$\Delta Wi(t) = Wi(t + \Delta t) - Wi(t) \sim N(0, \Delta t)$.

Therefore, in order to formulate the SDEs, the mean matrix $E(\Delta x)$ and the covariance matrix $V(\Delta x)$ need to be computed, so that the vector μ and the matrix B can be obtained.

The various transitions and probabilities for the system (1) are as follows.

Table 1: The compartmental changes in small time period Δt

Transition	Probability
$(\Delta X)_1 = [1, 0, 0]^T$	$p_1 = b(1-\omega)N\Delta t$
$(\Delta X)_2 = [-1, 1, 0]^T$	$p_2 = B(t)X_1\Delta t$
$(\Delta X)_3 = [-1, 0, 0]^T$	$p_3 = \mu X_1 \Delta t$
$(\Delta X)_4 = [0, -1, 0]^T$	$p_4 = (\mu + \alpha_0)X_2\Delta t$
$(\Delta X)_5 = [0, -1, 1]^T$	$p_5 = \delta X_2 \Delta t$
$(\Delta X)_6 = [0, 0, -1]^T$	$p_6 = (\mu + \alpha)X_3\Delta t$
$(\Delta X)_7 = [-1, 1, 0]^T$	$p_7 = b\omega N\Delta t$
$(\Delta X)_8 = [0, 0, 0]^T$	$1 - (p_1 + p_2 + p_3 + p_4 + p_5 + p_6 + p_7)$

The system (2) will be changed into the stochastic differential equations in the form;

$\Delta X(t) = F(t, X(t)dt + B(t, X(t))dw(t)$ (3)

Where, $E(\Delta X) = \sum_{i=1}^{7} p_i (\Delta X)_i$

$$= \begin{pmatrix} b(1-\omega)N - B(t)X_1 - \mu X_1 \\ b\omega N + B(t)X_1 - (\mu + \alpha_0 + \delta)X_2 \\ \delta X_2 - (\mu + \alpha)X_3 \end{pmatrix} \Delta t,$$

Covariance matrix $C = E((\Delta X)(\Delta X))^T$

$= \sum_{i=1}^{7} p_i (\Delta X)_i (\Delta X)_i^T$

$$= \begin{pmatrix} bN + B(t)X_1 & -b\omega N - B(t)X_1 & 0 \\ -b\omega N - B(t)X_1 & b\omega N + (B(t) + \mu)X_1 + (\mu + \alpha_0 + \delta)X_2 & 0 \\ 0 & 0 & (\mu + \alpha)X_3 \end{pmatrix} \Delta t$$

And $B = \sqrt{C}$.

Table 2: Data for HIV/AIDS model

Parameter	Value
b	0.0366
μ	0.0166

c	2
$c^{'}$	2
β	0.011-0.95 [1]
$\beta^{'}$	0.011-0.95 [1]
δ	Variable
α_0	0.001
α	0.0005
ω	Variable
S	50,000
I	2
R	0

Numerical Simulation

The control parameters for the model are transmission coefficient for members in R-class, $\beta^{'}$, the proportion of infected members on ART,δ and the proportion of neonates perinatally infected, ω. While we keep the values of other parameters in Table 2 fixed, we have allowed the values of β', δ and w to vary and investigated their effects on the spread of HIV/AIDS. The dynamics of the HIV/AIDS models (*2* and *3*) for various values of these control parameters are as shown in Figures $(1) - (7)$ below.

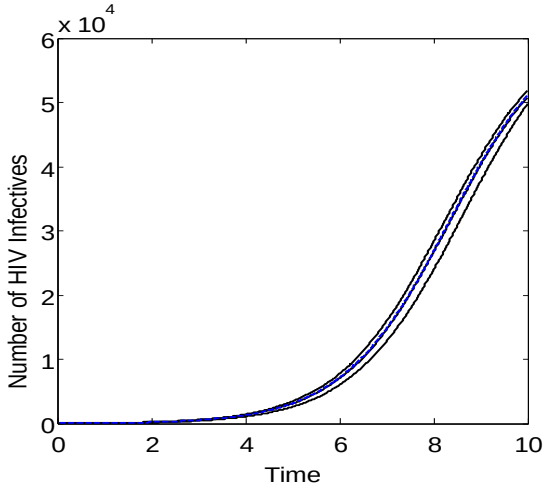

Figure 1: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2,c',=2,β=0.5,β'=0.5,δ=0,ω=0.01,p=0 (Without control)

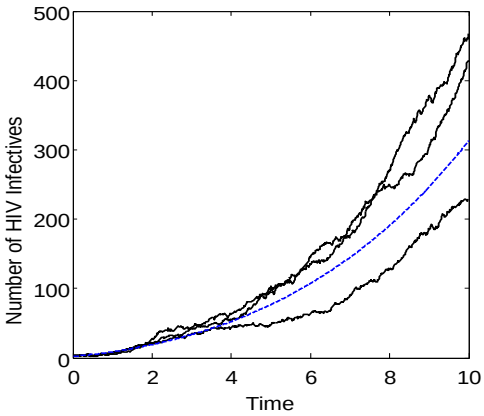

Figure 2: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2, c',=2,β=0.5,β'=0.011,δ=0.8,ω=0.001, p=0 (ART)

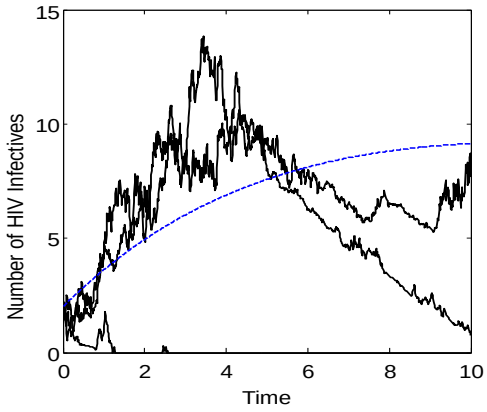

Figure 3: Sample paths of the stochastic model (black) and the trajectory of the deterministic model(blue) c=2, c',=2,β=0.5,β'=0.011,δ=1,ω=0.001,p=0 (ART)

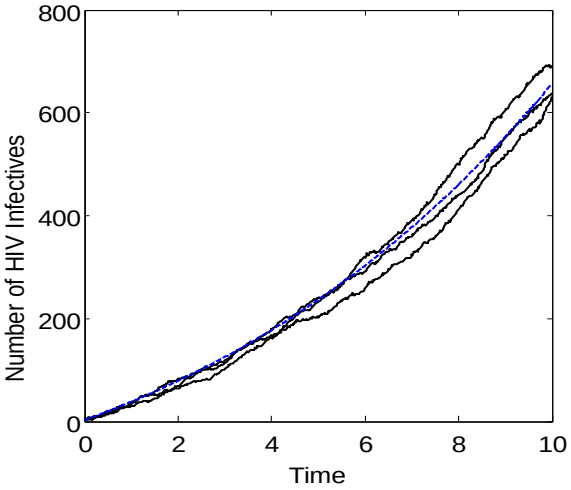

Figure 4: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2, β=0.5,,δ=0,ω=0.01,p=0.8 (Condom)

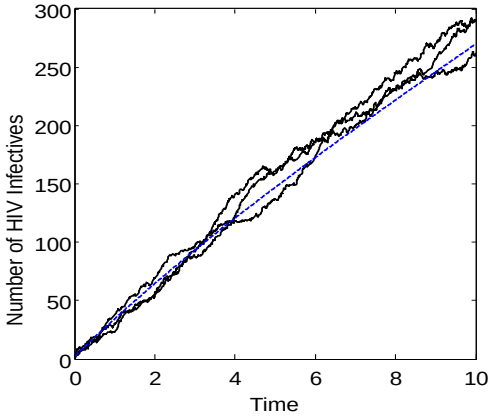

Figure 5: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2, β=0.5, δ=0, ω=0.01, p=1 (Condom)

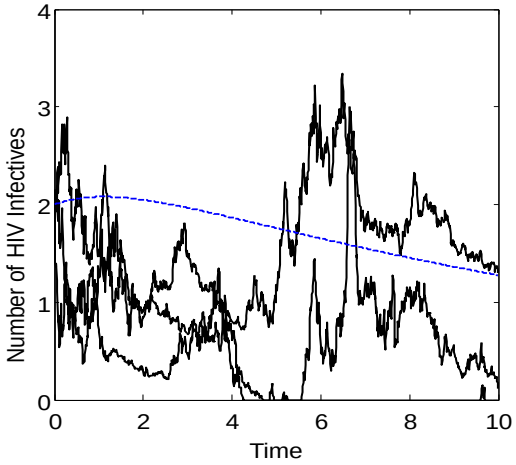

Figure 6: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2 c'=2, β'=0.011, β=0.5, δ=0.8, ω=0.001, p=0.8 (ART and Condom)

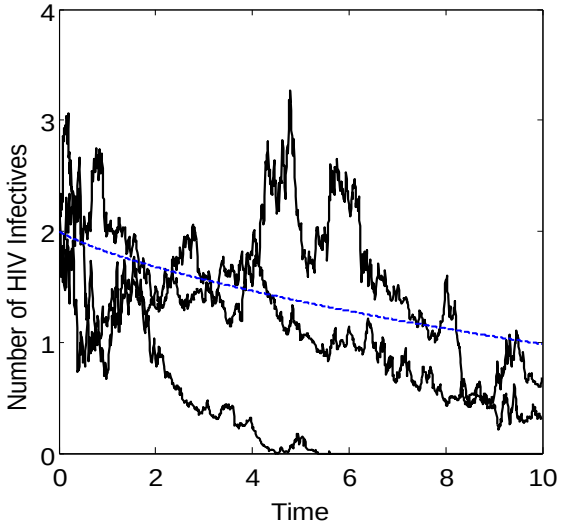

Figure 7: Sample paths of the stochastic model (black) and the trajectory of the deterministic model (blue) c=2 c'=2, β'=0.011,β=0.5, δ=1,ω=0.001,p=1 (ART and Condom)

The numerical results in this article are shown in Figures 1 through 7. Figure 1 shows a scenario where there is no control. In absence of any control measure, there is a very rapid growth in the number of cases. Figures 2 through 3show the results in a situation where there is effective ART. Figure 2 shows a slow upward trend in the number of cases while Figure 3 depicts a downward trend with all the realizations tending to the disease-free equilibrium state, though the trajectory of the deterministic model shows a much slower upward trend. Figures 4 through 5 show the results in a situation where there is condom use. The sample paths and the trajectory of the models show that condom only reduces the pace of growth of new cases. Figures 6 through 7show that there is control under effective combination of ART and condom use as a control strategy.

Conclusion

In this paper, deterministic and stochastic differential equation models for vertical and heterosexual transmission dynamics of HIV/AIDS in a population are formulated and investigated. The models are presented in sections 2 and 3. The models were solved numerically to investigate the effects of ART, condom use and both on the transmission dynamics and to also examine the model performance. The main results can be seen in Figures1 through 7. The results show that ART is a better control intervention than condom use, with the effective

combination of both the best. The model compete favorably well in the absence of any intervention. However, stochastic components ensue in the sample paths of the stochastic differential equation model, with a significant variability in the spread compared to the deterministic counterpart when there is effective control. The findings in this research point out that effective combination of ART and condom as a control strategy is sufficient for the control of HIV/AIDS.

References

[1]. Mukandavire, Z., Das, p., Chiyaka, C. and Nyabadza, Z.(2010). Global analysis of an HIV/AIDS epidemic model. World Journal of Modeling and Simulation, vol. 6 no. 3, pp. 231-240. ISSN1746-7233, England, UK.

[2]. Williams, B., Lima, V. and Gouws, E. (2011). Modeling the impact of antiretroviral therapy on the epidemics of HIV. Current HIV Research, Bentham Science Publishers Ltd.

[3]. www.Global2015.net/file/global2015hiv.pdf

[4]. Casels, S., clark, S. J. and Morris, M. (2008). Mathematical models for HIV transmission dynamics: Tools for social and behavioural science research. J. Acquir Immune Defic synd. Volume 47, Supplement 1. Lippincott Williams and Wilkins.

[5]. Sani, A. Kroese, D.P. and Pollet, P.K. (2006). Stochastic models for the spread of HIV in a mobile heterosexual population. Math. Biosci. Online: 1-27.

[6]. Peterson, D. Willard, K. Altmann, M. Gatewood, L. and Davidson, G.(1990). Monte Carlo simulation of HIV infection in an intravenous drug user community. Journal of Acquired Immune Deficiency Syndromes, 3 (11), pp.1086-1095.

[7]. Greenhalgh, D. and Hay. G. (1997). Mathematical modelling of the spread of HIV/AIDS amongst injecting drug users. IMA Journal of Mathematics Applied in Medicine and Biology, 14, pp.1138.

[8]. Dalal, N. Greenhalgh, D. and Mao, X. (2007). A Stochastic model of AIDS and condom use. Journal of Mathematical Analysis and Applications, 325, pp.36-53.

[9]. Dalal, N. Greenhalgh, D. and Mao, X. (2008). A Stochastic model for internal HIV dynamics. Journal of Mathematical Analysis and Applications, 341pp.1084-1101.

[10]. Ding, Y., Xu, M. and Hu, L. (2009). Risk analysis for AIDS control based on a stochastic model with treatment rate. Human and Ecological Risk Assessment, 15 (4), pp. 765-777.

[11]. Tuckwell, H. and Le Corfec, E. (1998). A Stochastic model for early HIV-1 population dynamics. Journal of Theoretical Biology, 195 (4), pp.451-463.

[12]. Waema, R. and Olowofeso, O. E. (2005). Mathematical modeling for human immunodeficiency virus (HIV) transmission using generating function approach. Kragujevac J. Sci. 27: 115-130.

[13]. Kimbir, A. R. and Oduwole, H. K. (2008). A mathematical model of HIV/AIDS transmission dynamics considering counseling and antiretroviral therapy. Journal of Modern Mathematics and Statistics, 2(5):166-169.