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On Simulation of the Effect of Compatibility Test on the Transmission Dynamics of HIV in a Heterosexual Population

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Abstract
Tests to determine genotypes, HIV status of partners are nowadays conducted to ascertain their compatibility for marriage or other forms of sexual relationships. The objective of this article is to simulate the effect of compatibility test compliance parameter on the transmission dynamics of HIV. A mathematical model of HIV incorporating compatibility test compliance parameter is presented. A unique disease-free equilibrium state was determined, indicating possibility of control of HBV disease. The model was solved numerically using Runge-Kutta method of order four to determine the sensitivity of the model solution to this parameter. The results of the numerical simulations of the model show that effective compatibility compliance as a control strategy can eradicate HIV disease. Finally, these findings strongly suggest that high level of compatibility compliance is crucial to the success of HIV disease control.

Key words: HIV, mathematical model, disease-free equilibrium, endemic equilibrium

1. Introduction

The human immune-deficiency virus (HIV) together with the associated acquired immune deficiency syndrome (AIDS) is a pandemic (Latora *et al*, 2006; Mukandavire *et al*, 2010; Williams *et al*, 2011). AIDS is an illness that damages a person's ability to fight off disease, leaving the body open to attack from ordinary innocuous infections and some forms of cancers. HIV disrupts the functioning of the immune system. A weakened immune system allows the development of a number of different infections and cancers, and it is these diseases which cause illness and death in people with AIDS. HIV also infects and causes direct damage to other types of cells (Pinsky and Douglas, 2009).

Two-third of all HIV infected people live in Sub-Saharan Africa; Nigeria ranking third in the highest burden apart from South Africa and India (Eze, 2009). This calls for more concerted control effort to arrest this ugly trend.
Heterosexual contacts are most responsible for HIV infections compared to homosexual, drug injection and mother- to-child routes of transmission (Casels *et al*, 2008; Williams *et al*, 2011).
Several intervention methods are available. These range from sex abstinence, use of condoms, education and use of antiretroviral drugs and counseling. Condoms used correctly can reduce the likelihood of HIV transmission to an extremely low level. Combinations of antiretroviral medications that have been available since 1996 are slowing, stopping, and even reversing the progression of HIV disease. Antiretroviral drugs are allowing many HIV infected people (who otherwise would become ill) to live active, healthy lives with few or no symptoms. These drugs are not a cure for HIV; the medications now available must be continued indefinitely to prevent progression of the disease (Pinsky and Douglas, 2009). To prevent HIV transmission through sexual contact, nowadays, HIV tests are conducted to determine compatibility of partners. This has helped in preventing HIV transmission and it is worthwhile studying the effects.

Two types of HIV are currently recognized: HIV-1 and HIV-2. The classification is based on differences in genetic structure. HIV-2 is the less common type and is found primarily in Western Africa. Both types of virus are transmitted in the same way and cause the same illnesses. However, it appears that HIV-2 is more difficult to transmit and that time from infection toillness is longer. In addition, a number of different sub-types or strains of HIV-1 have been classified. These subtypes (also known as "clades") are distinguished by smaller variations in their genetic composition. The sub-types are identified by letter. They are unevenly distributed geographically. Sub-type B is found mostly in the Americas, Japan, Australia, the Caribbean, and Europe. Sub-types A and D are most common in sub-Saharan Africa (Pinsky and Douglas, 2009).

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.
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.
The plan of this paper is as follows. Introductory part is presented in section 1. Section 2 is devoted to model formulation. Section 3 is devoted to numerical simulation. Discussion is done in section 4. Conclusive remarks are passed in sections 5.

2. Model Formulation

For a review of pioneer statistical and mathematical models of HIV/AIDS, the reader is referred to Schwager *et al* (1989). 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. Greenhalgh and Hay (1997) studied a mathematical model of the spread of HIV/AIDS among injecting drug users. Dalal *et al* (2007) examined a stochastic model of AIDS and condom use. Dalal, *et al* (2008) also studied a stochastic model for internal HIV dynamics. Ding *et al* (2009) carried out risk analysis for AIDS control based on a stochastic model with treatment rate. Tuckwell and Le Corfec (1998) studied a stochastic model for early HIV-1 population dynamics. Waema and Olowofeso (2005) studied a mathematical model for HIV transmission using generating function approach. A mathematical model to study the impact of antiretroviral therapy and counseling was formulated and analyzed by Kimbir and Oduwole, (2008). A Mathematical model of the dynamics of HIV transmission and control in a population considering a campaign of mandatory HIV status before sexual partnership was formulated and analysed by Abu. and Okutachi (2011). A stochastic differential equation model of SIS epidemic was formulated by *Gray et al* (2011).

2.1 The Variables and Parameters of the Model

The variables and parameters are defined as follows.

$S_1 = the\ number\ of\ susceptible\ females,$

$S_2 = the\ number\ of\ susceptible\ males,$

$I_1 = the\ number\ of\ infected\ females,$

$I_2 = the\ number\ of\ infected\ males,$

$A = the\ number\ of\ AIDS\ patients,$

$\xi_1, \xi_2 =$ recruitment rates into female and males,

$\beta_1, \beta_2 :=$ transmission coefficients for females and males respectively,

$\mu = natural\ mortality\ rate,$

$\mu_1 = disease - induced\ mortality\ rate$

$\sigma_1, \sigma_2 :=$ progression rates from infected female and male populations to AIDS status respectively,

$c_1, c_2 = average\ numbers\ of\ sex\ patners\ in\ female\ and\ male\ populations.$

$\alpha_1, \alpha_2 =$ compatibility compliance parameters

2.1 **The Model Equations**

We propose our model as follows. Our model is a two-sex version of the model proposed by Abu and Okutachi (2011).

$$\frac{dS_1}{dt} = \xi_1 - c_1\beta_1 S_1 I_2 - (\mu + \alpha_1)S_1 \quad (1)$$

$$\frac{dS_2}{dt} = \xi_2 - c_2\beta_2 S_2 I_1 - (\mu + \alpha_2)S_2 \quad (2)$$

$$\frac{dI_1}{dt} = c_1\beta_1 S_1 I_2 - (\mu + \sigma_1)I_1 \quad (3)$$

$$\frac{dI_2}{dt} = c_2\beta_2 S_2 I_1 - (\mu + \sigma_2)I_2 \quad (4)$$

$$\frac{dA}{dt} = \sigma_1 I_1 + \sigma_2 I_2 - (\mu + \mu_1)A \quad (5)$$

3. Numerical Simulation

We use the following parameter values for our simulations to obtain the ensuing Figures.

Table 1

Variable/parameter	value	Source
$S_1(0)$	5000	assumed
$S_2(0)$	5000	assumed
$I_1(0)$	3	assumed
$I_2(0)$	3	assumed
$A(0)$	0	assumed
ξ_1	2	assumed
ξ_2	2	assumed
β_1	0.5-0.7	CDC (2013)
β_2	0.2	CDC (2013)
μ	0.0189	estimated
σ_1,	0.0667	estimated
σ_2	0.0667	estimated
μ_1	0.05	estimated
c_1	2	assumed
c_2	2	assumed
α_1	0-1	variable
α_2	0-1	variable

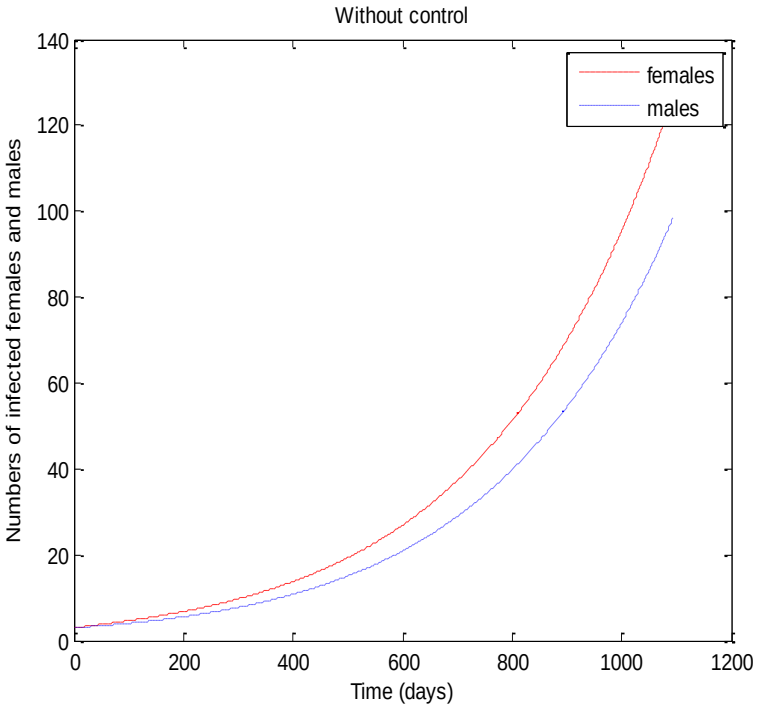

Figure 1: *Graph showing the numbers of infected males and females without control*

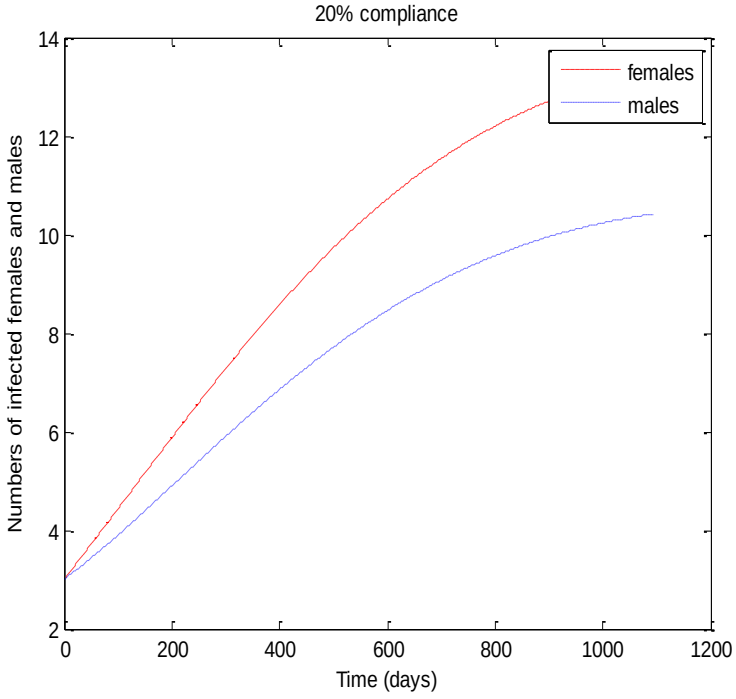

Figure 2: *Graph showing the numbers of infected males and females with* 20% *compliance*

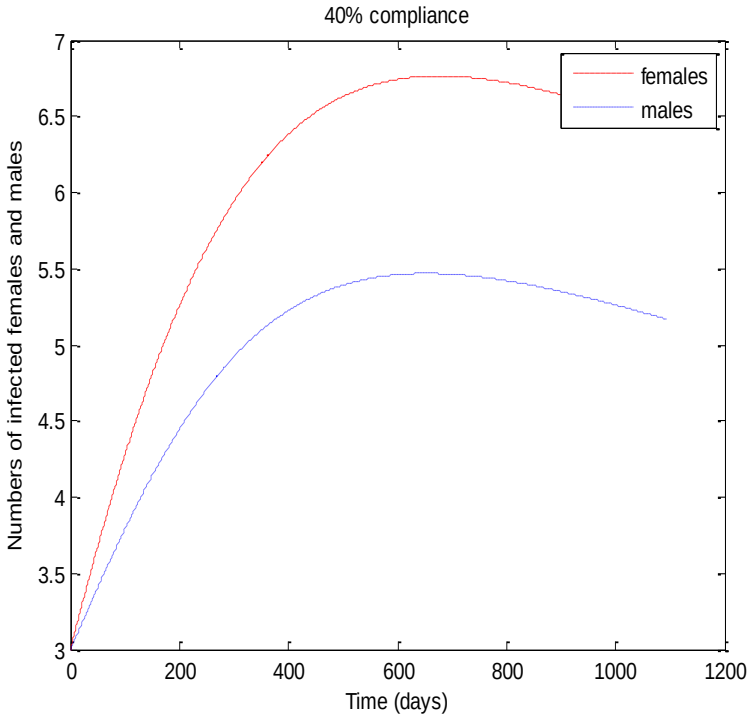

Figure 3: Graph showing the numbers of infected males and females with 40% compliance

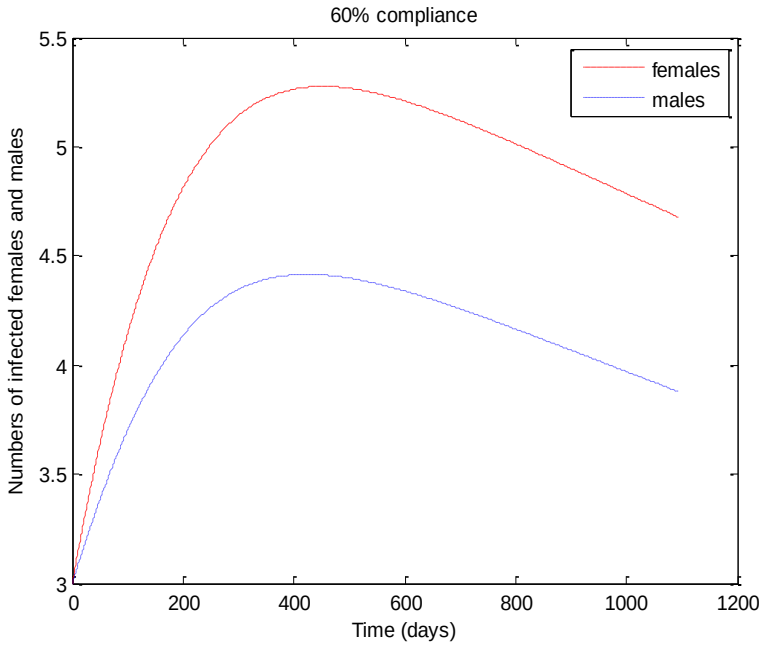

Figure 3: Graph showing the numbers of infected males and females with 60% compliance

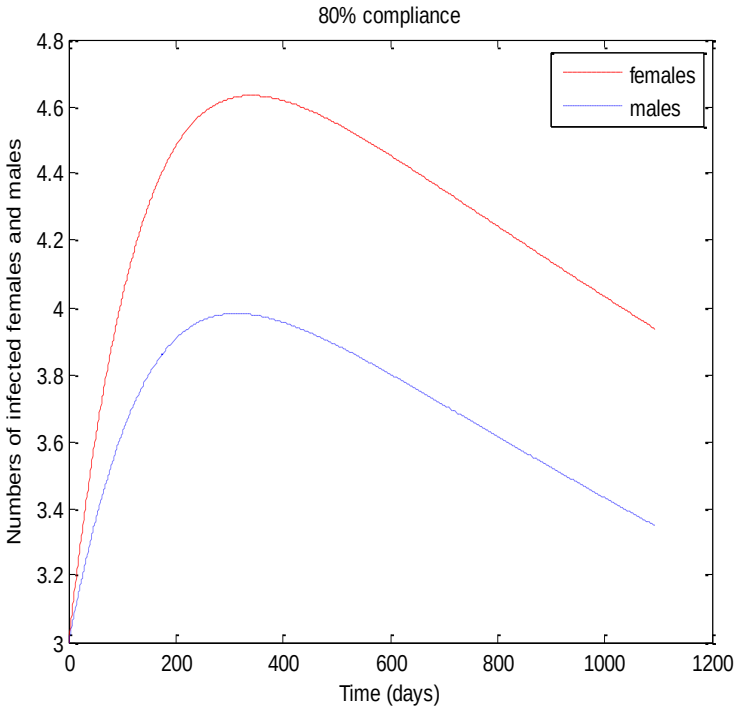

Figure 4: *Graph showing the numbers of infected males and females with* 80% *compliance*

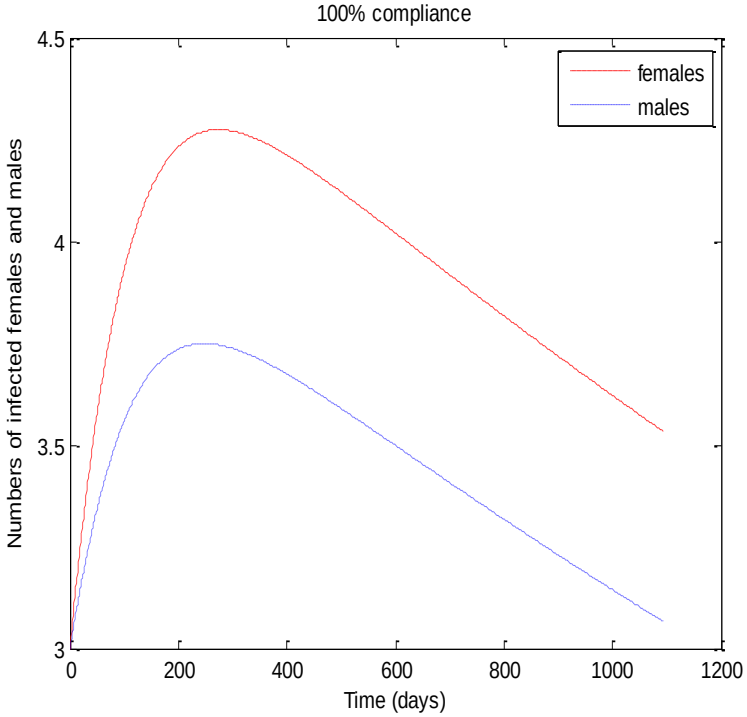

Figure 5: *Graph showing the numbers of infected males and females with* 100% *compliance*

4. Discussion of Results

In this article a mathematical model for HIV transmission in a heterosexual population, considering the effect of compatibility test is presented and numerically explored. The main aim is to assess the feasibility of control and eradication of HIV infection for different values of compliance parameter. The results of the numerical simulations can be seen in Figures 1 through 5. Figure 1 shows the numbers of infected males and females in absence of any intervention. This shows that HIV cases can increase rapidly in absence of any control. Figures 2 through 5 show that the number of HIV cases decreases with the level of compliance, guarantying eradication when compliance of 80% and above is in place.

Conclusion

In this article, we developed a mathematical model for HIV transmission in a heterosexual population, incorporating compatibility test. The model can be seen in section 2.1. The model was solved numerically using Runge-Kutta method of order four. The solutions for varying values of compatibility compliance parameter can be seen in Figures 1 through 5. The findings in this study reveal that effective compatibility compliance is crucial for eradication of HIV disease.

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