Transmission Dynamics of HIV/AIDS with Abstinence from Behavioral Risks

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Abstract

A nonlinear mathematical model for abstinence from behavioral risks in the transmission dynamics of HIV/AIDS with condom usage and compliance was proposed and rigorously analyzed. The local stability of the model was established for both disease free and endemic equilibrium, showing that the disease will die out when the effective reproductive number (R_C) *is less than unity and persist whenever* R_C *exceeds unity. Numerical simulation was done to validate the analytical results.*

Keywords and Phrases: *HIV/AIDS Model, Behavioral Risks, Effective Reproductive Number, Disease Free Equilibrium, Endemic Equilibrium*

1.0 Introduction

One of the most dangerous viral diseases, whose cause is known and the principal routes of transmission understood is Acquired Immuno-Deficiency Syndrome (AIDS) (Hansasuta and Rowland-Jones, 2001).The etiological agent of AIDS is Human Immunodeficiency Virus (HIV), which is a global challenge not only to public health workers but the world at large, since it has reached epidemic proportions in all over the world (Safiel et al., 2012).

Despite tremendous effort by researchers and scientists, HIV remains incurable,although pharmaceutical interventions (such as the use of vaccines(i.e. prophylactic and therapeutic) and HAART) and non pharmaceutical interventions (e.g abstinence from behavioural risks,being faithful,condom usage,public highlightment etc) are measures in placed to halt it spread (Smith and Blower, 2004; Dworkin and Ehrhardt 2007; Elbasha and Gummel, 2006; Hussaini et al., 2010). HIV risk behaviour are pratices or factors that increases the rate of acquiring or

transmitting the virus. These may include having multiple sex partners, been homosexual, sharing of needles, blood transfussion, syringes or other equipments use to inject drugs or steroids (WebMD, 2014; CDC, 2015).

Subharan Africa and other developing countries across the globe continues to have high prevalences of HIV/AIDS emanating from limited acess to pharmaceutical intervention due to high cost,high rate of unprotected sex,resistance to sex education,associated stigma to HIV infectives and female condom users,mutiple sex partners etc, (Mukandavire et al., 2007; Hussaini et al., 2010). Thus, there is need to develop an effective strategy that is cheap and very to access in the prevention and control of HIV/AIDS infections, which is paramount in curbing its menance.

Mathematical models in epidemiology have continously play important roles in increasing our understanding on mechanisms that influences the spread of infectious diseases ,suggesting the qualitative impact of disease control measures and forecasting disease incidences for both short and long term (Tripathi et al., 2007; Seidu and Makinde, 2014). Thus arousing the interest of Applied Mathematician and Biologist to study the dynamics of HIV/AIDS (Al-Sheikh et al., 2011; Abdulraham et al.,2013). Several models for HIV/AIDS transmission dynamics existing in literarures are found in (Naresh et al., 2009; Mukandavire and Garira, 2007; Mukandavire et al., 2009; Mukandavire et al., 2010; Ibrahim et al., 2015) and the references cited there in.

In this paper, we extend the model presented in (Mukandavire et al., 2010) by incorporating condom usage, condom compliance, indulgence and abstinence from HIV/AIDS behavioural risks. This paper is organized as follows: Section 2 present the model formulation for HIV/AIDS transmission dynamics, Section 3 consist of equilibria and stability analysis. Numerical simulations results are presented and discuss in Section 4 and finally conclusion in Section 5.

2.0 Model Formulation

To construct a deterministic model for the transmission dynamics of HIV/AIDS in the presence of abstinence from behavioral risks, condom usage and condom compliance, the total population at time t denoted by $N(t)$ is stratified into four (4) mutually exclusive epidemiological compartments namely, susceptible individuals that indulge in behavioral risk $S(t)$,susceptible individuals that refrain from behavioral risk $R(t)$,HIV infected individuals $I(t)$ and individuals with full blown AIDS $A(t)$.

Basic Assumptions of the Model

i. Individuals recruited into the population through migration or birth are assume to be susceptible.

ii. Vertical transmission and migration of infectives are not considered or assumed to be negligible.

iii. Birth rate and death rate are not equal

iv. Condom efficacy and compliance are not hundred percent effective in the prevention of HIV, thus $\tau < 1$ and $\theta < 1$

v. The human population is homogenous and depend on time t.

vi. Abstinence from behavioral risk is assumed to be a perfect control measure against infections.

Thus the governing equations describing the dynamics of HIV/AIDS in the presence of the aforementioned important factors are presented below.

$$\frac{dS}{dt} = \pi + \alpha_2 R - \Gamma S - (\mu + \alpha_1)S \quad (1)$$

$$\frac{dR}{dt} = \alpha_1 S - (\mu + \alpha_2)R \quad (2)$$

$$\frac{dI}{dt} = \Gamma S - (\mu + \gamma)I \quad (3)$$

$$\frac{dA}{dt} = \gamma I - (\mu + \delta)A \quad (4)$$

where

$$\Gamma = \beta(1 - \tau\theta)(I + \eta A) \quad (5)$$

$$\frac{dN}{dt} = \pi - \mu N - \delta A \quad (6)$$

Table 1: Parameter Description and Hypothetical Values

Parameters	Symbols	Hypothetical Values	References
Recruitment rate	π	29	Mukandavire et al., 2010
Natural death rate	μ	0.02	Ibrahim et al.,2015
AIDS induced death rate	δ	0.333	Mukandavire et al., 2009**a**
Condom efficacy	τ	0.8	Karen and Susan,1999
Condom compliance	θ	(0 1)	Variable
Modification parameter	η	1.4	Mukandavire et al., 2009**b**
Progression rate to AIDS	γ	0.125	Mukandavire et al., 2007
Disease transmission coefficient	β	0.0005	Javidi and Nyamorady,2013
Rate of abstaining from behavioral risks	α_1	(0 1)	Variable
Progression rate from $R(t)$ to $S(t)$	α_2	(0 1)	Variable

Lemma1: The close set $\Omega = \left\{(S,R,I,A) \in \mathbb{R}^4_+ : S+R+I+A \le \frac{\pi}{\mu}\right\}$ is positively invariant and attracting with respect to the system $(1)-(4)$

Proof

From (6), we note that $\frac{dN}{dt} \le \pi - \mu N$ and establish that $N(t) \le N(0)e^{-\mu t} + \frac{\pi}{\mu}\left[1-e^{-\mu t}\right]$ by a standard comparism theorem (Lakshmikantham et al., 1989). $N(t)$, approaches $\frac{\pi}{\mu}$ as $t \to \infty$, thus the system (1)-(4) is positively-invariant and attracting in Ω.Thus the model is mathematically and epidemiologically meaningful in Ω (Hethcote,2000), and it is sufficient to consider solutions in Ω.

3.0 Equilibria and Stability Analysis of the Model

At equilibrium, we set the left hand side of (1)-(4) to zero, i.e

$\frac{dS}{dt} = \frac{dR}{dt} = \frac{dI}{dt} = \frac{dA}{dt} = 0$, so that (1)-(4) becomes

$$0 = \pi + \alpha_2 R - \Gamma S - K_1 S \quad (7)$$

$$0 = \alpha_1 S - K_2 R \quad (8)$$

$$0 = \Gamma S - K_3 I \quad (9)$$

$$0 = \gamma I - K_4 A \quad (10)$$

where

$$K_1 = \mu + \alpha_1, \quad K_2 = \mu + \alpha_2, \quad K_3 = \mu + \gamma, \quad K_4 = \mu + \delta$$

From (10),we get

$$A = \frac{\gamma I}{K_4} \quad (11)$$

From (9),

$$I = \frac{\Gamma S}{K_3} \quad (12)$$

From (8)

$$R = \frac{\alpha_1 S}{K_2} \quad (13)$$

Using (13) in (7) to have

$$\pi + \frac{\alpha_1\alpha_2 S}{K_2} - (\Gamma + K_1)S = 0$$

$$\left[K_2(\Gamma + K_1) - \alpha_1\alpha_2\right]S = K_2\pi$$

$$S = \frac{K_2\pi}{\left[K_2(\Gamma + K_1) - \alpha_1\alpha_2\right]} \qquad (14)$$

Substituting (11) into (5), to obtain

$$\Gamma = \beta(1-\tau\theta)\left[\frac{K_4 + \eta\gamma}{K_4}\right]I \qquad (15)$$

Using (15) into (12), to get

$$I = \frac{\beta(1-\tau\theta)\left[K_4 + \eta\gamma\right]SI}{K_3K_4}$$

$$\left\{\beta(1-\tau\theta)\left[K_4 + \eta\gamma\right]S - K_3K_4\right\}I = 0$$

This implies either

$$I = 0 \text{ or } S = \frac{K_3K_4}{\beta(1-\tau\theta)\left[K_4 + \eta\gamma\right]} \qquad (16)$$

3.1 Existence of Disease Free Equilibrium

Let ε_0 denote the disease free equilibrium, in the absence of infection, we have from (16), (11), (15) $I^* = 0$ $A^* = 0, \Gamma^* = 0$ respectively. From (14), we obtain

$$S^* = \frac{K_2\pi}{K_1K_2 - \alpha_1\alpha_2} \qquad (17)$$

Using (17) in (13), we have

$$R^* = \frac{\alpha_1\pi}{K_1K_2 - \alpha_1\alpha_2} \qquad (18)$$

$$\therefore \varepsilon_0 = \left(S^*, R^*, I^*, A^*\right) = \left(\frac{K_2\pi}{K_1K_2 - \alpha_1\alpha_2}, \frac{\alpha_1\pi}{K_1K_2 - \alpha_1\alpha_2}, 0, 0\right)$$

The stability of ε_0 can be explored by the method of Reproductive Number (R_C) which is determined by using the next generation method, on system (1) in the form of matrices F(non-negative) and V(non-singular) (Heffernan et al., 2005).Where F denote the new infection terms and V the transition term at ε_0. Therefore

$$F=\begin{bmatrix} L_1S^* & L_1\eta S^* \\ 0 & 0 \end{bmatrix}, \qquad \mathrm{V}=\begin{bmatrix} K_3 & 0 \\ -\gamma & K_4 \end{bmatrix}$$

$$R_C=\rho(FV^{-1})=\frac{L_1S^*\left[K_4+\eta\gamma\right]}{K_3K_4} \qquad (19a)$$

$$R_C=\frac{\beta K_2\pi(1-\tau\theta)\left[K_4+\eta\gamma\right]}{K_3K_4\left[K_1K_2-\alpha_1\alpha_2\right]} \qquad (19b)$$

where $L_1=\beta(1-\tau\theta)$

It is of great importance to know that $K_1K_2-\alpha_1\alpha_2=\mu(\mu+\alpha_1+\alpha_2)>0$ and $\tau\theta<1$ since $\theta<1$ and $\tau<1$, hence $R_C>0$.

Theorem 1: The disease free equilibrium of the system (1)-(4) is locally asymptotically stable if $R_C<1$ and unstable if $R_C>1$.

Proof

The Jacobian matrix of system (1)-(4) at ε_0 is given as

$$J(\varepsilon_0)=\begin{bmatrix} -K_1 & \alpha_2 & -L_1S^* & -L_1\eta S^* \\ \alpha_1 & -K_2 & 0 & 0 \\ 0 & 0 & L_1S^*-K_3 & -L_1\eta S^* \\ 0 & 0 & \gamma & -K_4 \end{bmatrix} \qquad (20)$$

The characteristic equation of (20) is given in the form

$$\lambda^4+a_3\lambda^3+a_2\lambda^2+a_1\lambda+a_0=0 \qquad (21)$$

Where the coefficients of the eigenvalues are expressed in terms of R_C with the aid of $(19a)$ as

$$a_3=\frac{\left[K_4+\eta\gamma\right]\left[(K_1K_2-\alpha_1\alpha_2)+K_4(K_1+K_2)\right]+K_3\eta\gamma(K_1+K_2)+K_3K_4\left[(K_4+\eta\gamma)+(K_1+K_2)\right](1-R_C)}{\left[K_4+\eta\gamma\right]},$$

$$a_2=\frac{\left[(K_1K_2-\alpha_1\alpha_2)+K_4(K_1+K_2)\right]+K_3\eta\gamma(K_1+K_2)+K_3K_4\left[K_4+\eta\gamma+K_1+K_2\right](1-R_C)}{\left[K_4+\eta\gamma\right]},$$

$$a_1=\frac{\left[K_4(K_4+\eta\gamma)+K_3\eta\gamma\right](K_1K_2-\alpha_1\alpha_2)+K_3K_4\left[(K_4+\eta\gamma)(K_1+K_2)+(K_1K_2-\alpha_1\alpha_2)\right](1-R_C)}{\left[K_4+\eta\gamma\right]},$$

$$a_0=K_3K_4\left[K_1K_2-\alpha_1\alpha_2\right](1-R_C)$$

Whenever $R_C < 1$, we note that $a_i > 0 \ \forall \ i = 0,..3$, then by Routh Hurwitz criterion, (21) will have all its roots (i.e. eigenvalues) to be negative. Hence the system is said to be locally asymptotically stable at ε_0 whenever $R_C < 1$ which completes the proof.

3.2 Existence of Endemic Equilibrium

Let ε_1 denote the endemic equilibrium, so that in the presence of infection $I^{**} \neq 0$, thus from (16), we have

$$S^{**} = \frac{K_3 K_4}{\beta(1-\tau\theta)\left[K_4 + \eta\gamma\right]} \qquad (21)$$

Substituting (21) into (13), to have

$$R^{**} = \frac{\alpha_1 K_3 K_4}{\beta(1-\tau\theta)K_2\left[K_4 + \eta\gamma\right]} \qquad (22)$$

Adding (7) and (9) to get

$$\pi + \alpha_2 R^{**} - K_1 S^{**} - K_3 I^{**} = 0 \qquad (23)$$

Substituting (21) and (22) into (23) to get,

$$\pi + \frac{\alpha_1\alpha_2 K_3 K_4}{\beta(1-\tau\theta)K_2\left[K_4 + \eta\gamma\right]} - \frac{K_1 K_3 K_4}{\beta(1-\tau\theta)\left[K_4 + \eta\gamma\right]} - K_3 I^{**} = 0$$

$$I^{**} = \frac{\beta(1-\tau\theta)\pi K_2\left[K_4 + \eta\gamma\right] - K_3 K_4\left[K_1 K_2 - \alpha_1\alpha_2\right]}{\beta(1-\tau\theta)K_2 K_3\left[K_4 + \eta\gamma\right]} \qquad (24)$$

Using (24) in (11) to obtain

$$A^{**} = \frac{\gamma\left\{\beta(1-\tau\theta)\pi K_2\left[K_4 + \eta\gamma\right] - K_3 K_4\left[K_1 K_2 - \alpha_1\alpha_2\right]\right\}}{\beta(1-\tau\theta)K_2 K_3 K_4\left[K_4 + \eta\gamma\right]} \qquad (25)$$

Expressing ε_1 in terms of R_C by substituting (19) into (21), (22), (23) and (24), respectively to obtain the following.

$$S^{**} = \frac{K_2\pi}{R_C\left[K_1 K_2 - \alpha_1\alpha_2\right]} \qquad (26)$$

$$R^{**} = \frac{\alpha_1\pi}{R_C\left[K_1 K_2 - \alpha_1\alpha_2\right]} \qquad (27)$$

$$I^{**} = \frac{\pi\left[R_C - 1\right]}{K_3 R_C} \qquad (28)$$

$$A^{**} = \frac{\gamma\pi\left[R_C - 1\right]}{K_3 K_4 R_C} \qquad (29)$$

Theorem 2: The endemic equilibrium of the system (1)-(4) is locally asymptotically stable if $R_C > 1$ and unstable if $R_C < 1$.

Proof

The Jacobian matrix of the system (1)-(4), evaluated at ε_1 is obtain as

$$J(\varepsilon_1)=\begin{bmatrix} -\beta^*(I^{**}+\eta A^{**})-K_1 & \alpha_2 & -\beta^* S & -\beta^* S^{**}\eta \\ \alpha_1 & -K_2 & 0 & 0 \\ \beta^*(I^{**}+\eta A^{**}) & 0 & \beta^* S^{**}-K_3 & \beta^* S^{**}\eta \\ 0 & 0 & \gamma & -K_4 \end{bmatrix} \quad (30)$$

by row elementary transform [12], (30) becomes,

$$\bar{J}(\varepsilon_1)=\begin{bmatrix} -J_1 & \alpha_2 & -\beta^* S^{**} & -\beta^* S^{**}\eta \\ \alpha_1 & \frac{-J_2}{J_1} & \frac{-\alpha_1\beta^* S^{**}}{J_1} & \frac{-\alpha_1\beta^*\eta S^{**}}{J_1} \\ 0 & 0 & \frac{-J_3}{J_2} & \frac{(K_1K_2-\alpha_1\alpha_2)\beta^*\eta S^{**}}{J_2} \\ 0 & 0 & 0 & \frac{-J_4}{J_3} \end{bmatrix}$$

$$\lambda_1=-J_1,\lambda_2=\frac{-J_2}{J_1},\lambda_3=\frac{-J_2}{J_1},\lambda_4=\frac{-J_4}{J_3}$$

where

$$\begin{aligned} L_1&=\beta(1-\tau\theta) \\ J_1&=L_1(I^{**}+\eta A^{**})+K_1 \\ J_2&=K_1J_1-\alpha_1\alpha_2 \\ J_3&=K_3J_2-L_1S^{**}(K_1K_2-\alpha_1\alpha_2) \\ J_4&=K_4J_3-L_1S^{**}\eta\gamma(K_1K_2-\alpha_1\alpha_2) \end{aligned} \quad (31)$$

Substituting (26), (28) and (29) appropriately into (31) to obtain

$$\begin{aligned} J_1&=\frac{L_1\pi(K_4+\eta\gamma)(R_C-1)}{K_3K_4R_C}+K_1 \\ J_2&=\frac{L_1\pi K_2(K_4+\eta\gamma)}{K_3K_4} \\ J_3&=\frac{L_1\pi K_2\left[K_4(R_C-1)+\eta\gamma R_C\right]}{K_4R_C} \\ J_4&=\frac{L_1\pi K_2(R_C-1)(K_4+\eta\gamma)}{R_C} \end{aligned} \quad (32)$$

Obviously, $J_2>0$ and whenever $R_C>1$, J_1, J_3 and J_4 are positive, hence $\lambda_i<0\forall i=1,...4$. Thus the system (1)-(4) is locally asymptotically stable whenever $R_C>1$, and this completes the proof.

4.0 Numerical Simulation and Discussion of Results

In this section, we present some numerical simulation for the set of parameters presented in Table 1, in order to study the dynamical behavioral of the model (1)-(4) and authenticate the above analytical findings.

Table 2: Effect of R_C on numbers of HIV/AIDS cases at steady state

θ	α_1	α_2	R_C	$I^{**}+A^{**}$	Remarks
0	0.9	0.1	0.6892	0	ε_0 stable (disease eradication)
0	0.8	0.1	0.7641	0	ε_0 stable (disease eradication)
0.2	0.8	0.2	1.0613	12.9682	ε_1 stable (no eradication)
0.2	0.8	0.4	1.6940	91.9753	ε_1 stable (no eradication)
0.4	0.8	0.4	1.3713	60.7908	ε_1 stable (no eradication)
0.6	0.6	0.6	1.5480	79.4783	ε_1 stable (no eradication)
0.6	0.4	0.6	1.8515	103.2540	ε_1 stable (no eradication)
0.8	0.2	0.6	2.4488	132.8285	ε_1 stable (no eradication)
0.9	0.2	0.8	1.7322	94.9038	ε_1 stable (no eradication)

Note: The Table is generated by using parameters value in Table 1 while varying the values of $\theta, \alpha_1, \alpha_2$.

It is clear to observe from the tabulated results that as HIV/AIDS cases increases, R_C increases. The qualitative dynamics of the model will change, since increase in either rate of abstinence from behavioral risks associated to HIV/AIDS α_1 or rate of condom compliance θ will reduces R_C, thus HIV/AIDS prevalence may decrease. Secondly, increase in rate of indulgence in HIV/AIDS associated risks behavior α_2 will increase R_C, hence disease burden may be triggered. Lastly, the results presented in this section validate the analytical results obtained in section 3, since $R_C < 1$, the disease can be eradicated and when $R_C > 1$, the disease will continue to persist.

5.0 Conclusion

In this study, a deterministic nonlinear model for the transmission dynamics of HIV/AIDS in the presence of condom usage and compliance, indulgence and abstinence from behavioral risk is constructed and painstakingly analyzed to obtain the following main results.

(1) The model (1)-(4) has a locally asymptotically stable disease free equilibrium whenever $R_C < 1$.

(2) The model endemic equilibrium is locally asymptotically stable whenever $R_C > 1$

(3) Increase in abstinence from HIV associated behavioral risks reduces R_C, thus reducing disease burden, conversely increase in indulgence in behavioral risks will increase R_C, hence promoting disease prevalence.

(4) Condom compliance may reduce HIV/AIDS cases when increased.

References

Abdulraham, S; bawa, M; Aliyu, Y.B. and Ajike, A.I(2013). Stability analysis of the transmission dynamics of HIV/AIDS in the presence of treatment and condom usage. International Journal of Science and Technology, 2(4),336-352.

Al-Sheikh, S., Musali, F. and Alsolami, M. (2011). Stability Analysis of an HIV/AIDS Epidemic Model with Screening. International Mathematical Forum, 6(66): 3251 – 3273.

CDC (7th December, 2015). www.cdc.gov/hiv/risk/estimates/riskbehaviours.html. (Accessed 5th April, 2016).

Dworkin,S.L. and Ehrhardt,A.A.(January,2007).Going Beyond "ABC" to include "GEM": Critical Reflections on Progress in the HIV/AIDS Epidemic. Am J Public Health 97(1):13-18. doi:10.2105/AJPH.2005.074591.

Elbasha, E.H and Gummel, A.B.(2006). Theoretical assessment of public health impact of imperfect prophylactic HIV-1 vaccines with therapentic benefits. Bullettin of mathematical biology, 68, 577-614. doi:10:1007/511538-005-9057-5.

Gummel, A.B; Mc Cluskey, C.C; and Van den Driessche, P.(2006). Mathematical study of a staged- progression HIV model with imperfect vaccines. Bullettin of Mathematical Biology, 68,2105-2128.

Hansasuta, P and Rowland-Jones, S.L. (2001). HIV-1 transmission and acute HIV-1 infection. Oxford Journal.

Heffernan, J. M., Smith, R. J. and Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. J. R.Soc. Interface, 2:281–293.

Hethcote, H. W. (2000). The mathematics of infectious diseases. *SIAM Re-view*. 42: 599-653.

Hussaini,N., Winter, N. and Gummel, A.B.(2010). Qualitative assessment of the role of public health education program on HIV transmission dyanamics. IMA Journal of mathematical medicine and Biology. To appear dio:10.1093/immammb/dqq2009.

Ibrahim, M.O., Akinyemi, S.T., Dago, M.M and Bakare, N.G. (2015). Mathematical Modelling of a Staged Progression HIV/AIDS with Control Measures. Journal of the Nigerian Association of Mathematical Physics. Volume 29,pp 163-166.

Javidi, M and Nyamorady N. (2013). Numerical Behaviour of a fractal order HIV/AIDS. World Journal of Modelling and Simulation. **9**(2):139-149.

Karen, R.D. and. Susan, C.W. (1999). The effectiveness of condoms in reducing heterosexual transmission of HIV. *Family Planning Perspectives*. **31**(6): 272-279.

Lakshmikantham, V., Leela, S. and Martynyuk, A. A. (1989). Stability Analysis of Nonlinear Systems. Marcel Dekker, Inc., New York and Basel.

Mukandavire, Z., Das,P., Chiyaka,C and Nyabadza, F. (2010).Global Analysis of an HIV/AIDS Epidemic Model.World Journal of Modelling and Simulation,**6**(3): pp 231-240.

Mukandavire, Z. and Garira, W.(2007). Effects of public health educational campaigns and the role of sex workers on the spread of HIV/AIDS among heterosexuals. Applied Mathematical Modelling. **33**:2084-2095.

Mukandavire, Z., Garira, W. and Chiyaka, C. (2007). Asymptotic properties of an HIV/AIDS model with a time delay, J. Math. Anal. Appl. 330: 916-933.

Mukandavire, Z., Garira, W. and Tchuenche, J.M.(2009**a**). Modelling effects of public health educational campaign on HIV/AIDS trasmission dynamics. Theoretical population Biology, **72**:346-365.

Mukandavire, Z., Gumel, A., et al. (2009**b**). Mathematical Analysis of a Model for 251 HIV Malaria Co-infection. Mathematical Biosciences and Engineering. **6**(2): 333–36.

Naresh, R., Tripathi, A. and Sharma, D. (2009).Modelling and analysis of the spread of AIDS Epidemic with immigration of HIV infectives. Mathematical and Computer Modelling 49:880-892.

Safiel, R., Massawe, E.S and Makinde, O.D. (2012).Modelling the Effect of Screening and Treatment on Transmission of HIV/AIDS Infection in a Population. American Journal of Mathematics and Statistics, 2(4): 75-88. doi: 10.5923/j.ajms.20120204.03.

Seidu, B. and Makinde, O.D.(2014). Optimal Control of HIV/AIDS in the Workplace in the Presence of Careless Individuals. Computational and Mathematical Methods in Medicine, Volume 2014, Article ID 831506, 19 pages. http://dx.doi.org/10.1155/2014/831506

Smith, R.J. and Blower, S.M.(2004). Could disease-modifying HIV vaccines cause population Level perversity? Lancet Infec. Dis.4, 636-639.

Tripathi, A., Naresh, R. and Sharma,D.(2007). Modelling the effect of screening of unaware infectives on the spread of HIV infection. Applied Mathematics and Computation, 184: 1053–1068.

WebMD (9th September, 2014). www.webmd.com/hiv-aids/high-risk-behaviors-for-hiv. (Accessed 5th April, 2016).