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Research Article

Mathematical Study of a Staged Progression of HIV Model with Imperfect Vaccine and Condom Use

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ABSTRACT: *The model, a staged progression of HIV is formulated and used to investigate the potential impact of an imperfect vaccine and condom use. The condom is assumed to prevent the transmission of HIV during sexual intercourse if properly used. It is also noted that condoms have their rate of failures. The vaccines on the other hand being also imperfect, has its own characteristics such as protection against infection, causing bypass of primary stage infection , capable of reducing viral load on the infected (especially individuals in the fully blown AIDS infection stage on vaccine could be brought to asymptomatic stage). The model which incorporates individual in the AIDS stage, is rigorously analyzed to give insight into its qualitative features. Using a comparison theorem, the model with mass action incidence is shown to have a locally-asymptotically stable disease free equilibrium whenever a certain threshold, known as the vaccination/condom reproduction number,* R_{VC} *, is less than unity. The model has a unique endemic equilibrium whenever this threshold exceeds unity. The epidemiological implication of these results are that imperfect vaccine with condom use, can eliminate HIV in a given community if it can reduce the reproduction number to a value less than unity, but the disease will persist if otherwise.*

Keywords: *HIV/AIDS, Staged progression, Vaccination/Condom reproductive number, local stability.*

I. INTRODUCTION

Human Immuno-Deficiency Virus, HIV, has been rated as one of the world's most dreaded disease. Since when it appeared in 1980s, it has claimed over 20 million lives and has been estimated that 36 to 40 million people are living with it. HIV pandemic has continued to inflict major socio-economic burden on many developing nations [1], [2], [3]. It has been opined by many people that curtailing the Global spread requires an effective vaccine [4], [5]. HIV predisposes one to other secondary infections. According to [6]; HIV infection accelerates the activation of Tuberculosis (TB) infection while TB accelerates the HIV development to AIDS and hence the risk of death and the development of other opportunistic infections. Owing to the Global HIV vaccine enterprise and the related initiatives, several candidate-HIV vaccines are currently undergoing clinical trials [7], [8]. Just like many other drugs developed, these vaccines are expected to be imperfect. That is it may be effective to some, but not for others and/or may offer protection that wares with time [9]. The vaccine may also offer some therapeutic benefits such as altering the clinical course of the disease [10], [11], [12], [9].

It is a fact that several authors have evaluated the potential community-wide impact of such a vaccine using mathematical models, [13], [14], [15] and [16]. However, some of these models failed to incorporate some other known important aspect of HIV disease such as its staged-progression nature, where HIV-infected individuals pass through sequential stages:- being highly infective during the primary infection stage (first few weeks of infection), having low infectivity during the asymptomatic (lasting many years) and becoming more infective in the Aids stage.

This stage progression models were considered in [17], [18] and [19] but they did not incorporate the use of vaccines. Some of these authors failed to recorganize that infected individuals in the Aids stage can also infect others [9] Individuals in the Aid stage are also sexually active and do engage in risky sexual behaviours such as having multiple sexual partners or

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involved in inconsistent use of condoms [20]. [9] actually incorporated all these facts and even included the possibility of vaccine induced by pass of primary infection stage but did not incorporate the impact of condom use in their model. This study therefore compliments and extends the aforementioned studies by incorporating the effect of condom use on the imperfect vaccine model.

Condoms are sheaths that trap the sperm when a man climaxes. Wearing them greatly reduce the chances of pregnancy. They also provide some protection against sexually transmitted infections (STIs) including HIV. But this protection is not 100% in all cases. In fact, it is estimated that condoms are about 98% effective if correctly used. Other human factors can also contribute to lower rate of its effectiveness.

A survey carried out by the Netherlands Institute of Social Sexological Research (NISSR), Utredit, assessed the extent to which condom are used effectively in commercial heterosexual intercourse on 127 female prostitutes and 91 male clients. They found out that 49% of the prostitute and 16% of their clients experienced condom breakages during the sexual intercourse within a space of 6 months. They however attributed those breakages to human factors such as rough and prolonged intercourse, incorrect handling of the condoms or use of insufficient lubricants. They also noted that 13% of the clients and 36% of the prostitutes have experienced condom slipping off, before or after ejaculation. This shows that both vaccines and condoms have their own individual failure rates.

II. MODEL FORMULATION AND BASIC PROPERTIES

Following [17] and [9] the population being studied is assumed to be a small high risk subset of a larger population. It is further assumed that this larger population is relatively free of HIV and provides a constant source of uninfected individuals entering the high-risk population. The model monitors the temporary dynamics of the high-risk population which is subdivided into subpopulation of unvaccinated ($S_u^{(t)}$) and vaccinated ($S_v^{(t)}$) susceptible individuals. The subdivisions in the HIV infected individuals are the highly infectious primary stage ($I_1^{(t)}$), low infections secondary stage ($I_2^{(t)}$) and highly infectious Aids stage ($I_3^{(t)}$). The total high-risk population is N(t). That is:

$$N(t) = (S_u^{(t)}) + (S_v^{(t)}) + (I_1^{(t)}) + (I_2^{(t)}) + (I_3^{(t)}).$$

The compartmental diagram in Fig. 1 below illustrates the flow of individual as they face the possibility of acquiring specific disease infection stages.

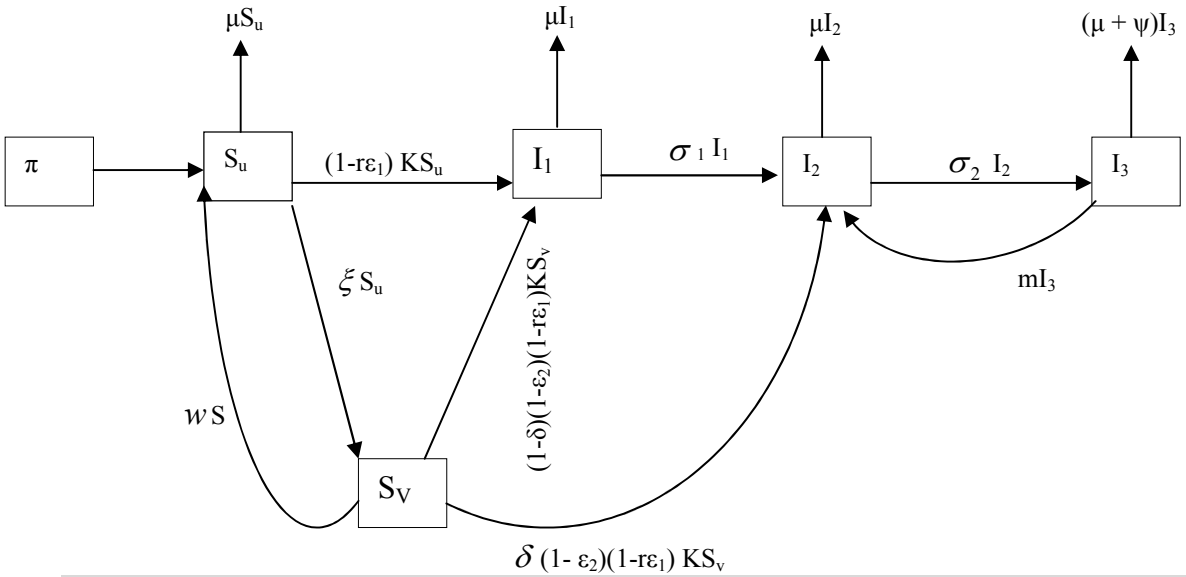

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Figure 1: Flowchart of the transmission dynamics of HIV subject to imperfect vaccines and condom use.

The force of infection on unvaccinated and vaccine-waned susceptible individual is given as: K = $\beta(N)(I_1 + n_2 I_2 + n_3 I_3)$

The unvaccinated susceptible subpopulation ($S_u^{(t)}$) is increased by the daily recruitment of uninfected sexually-active individuals from the larger population (at the rate π) and by waning of vaccine-induced immunity of the vaccine sub-population. This susceptible sub-population is however decreased by vaccination of some of them (at per capital rate, ξ). Also this susceptible sub-population which is further divided into two sub-groups:- the proportion that use condom (r) and those that do not use condom (1-r), are decreased via horizontal transfer to infected individuals in any of the three infected classes. Note that r is the average number of the susceptible sub-group that use condom divided by the total number of the susceptible sub-group. We assume that r is approximately-the same for both vaccinated and unvaccinated sub-groups. We note that condom use is not 100% effective in controlling HIV transmission as earlier mentioned. Thus, if we assume that 0< ε_1 <l accounts for its rate of efficiency, then 1- ε_1 accounts for its inefficiency.

Let C(N) be the average number of contacts sufficient to transmit infection in a unit time for infected individual in the population (of size N), without or with failed condoms. Then the number of new infection in a unit time is $\frac{C(N)SI}{N}$, where S and I represent the population of susceptible and infected individuals respectively. It is convenient to define the transmission level per contact $\beta(N)$ $as \frac{C(N)}{N}$) with $\beta(N) > 0$ for N > 0.

The terms $\beta(N)$ I_1, $\beta(N)$ I_2 and $\beta(N)$ I_3 are the force of infection from the primary, secondary and Aids stages respectively. That is, the average number of contacts with infected individuals in these stages per unit time. The parameters $O \leq n_2 \leq I$ accounts for the assumed infectivity of infected individuals in the secondary infection stage (due to their lower viral load). Similarly, a modification parameter $O \leq n_3 \leq I$ accounts for the transmission rate from individuals in the Aids stage which is believed to have similar number of effective contacts as individual in the primary infection stage because of their high viral load. Thus, the term $\beta(N)$ (I_1+n_2I_2+n_3I_3) gives the total force of infection and the number of new cases per unit time from the unvaccinated susceptible individuals. The susceptible sub-population is further decreased by natural death (at per capital rate, μ). The parameter μ, also includes the rate at which individuals leave the high-risk population due to migration or other reasons not related to HIV infection.

The rate of change S_u is therefore:

$$\frac{dS_u}{dt} = \Pi + wS_v - \beta(N)(1-r)(I_1 + n_2 I_2 + n_3 I_3) S_u$$

$$- \beta(N)\, r(1-\varepsilon_1)((I_1 + n_2 I_2 + n_3 I_3) S_u - \xi S_U - \mu S_u$$

$$= \pi + w S_v - \beta(N)(I_1 + n_2 I_2 + n_3 I_3)(1 - r + r(1-\varepsilon_1)) S_u - (\xi + \mu) S_u$$

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$$= \Pi + wS_V - \beta(\mathrm{N})(I_1 + n_2 I_2 + n_3 I_3)(1 - r\varepsilon_1)S_u - (\xi + \mu)Su \qquad (1)$$

w is the rate of waning of the vaccine on the vaccinated sub-group.

The sub-population of vaccinated susceptible individuals ($S_v^{(t)}$) is generated by the vaccination of susceptible individuals (at the per capital rate, ξ) and diminishes by vaccine waning (at per capital rate w) and infection of vaccinated individual with failed vaccines and failed condom; or failed vaccines without any condom use. Let $O \leq \varepsilon_2 \leq I$ account for the efficacy of the vaccine-induced protection (since no vaccine offers 100% protection all the time). Since a vaccine is assumed to lead to reduction in viral load in a break-through infections [16], it is further assumed that a proportion (δ) of vaccinated infected individuals move straight to secondary infection stage by passing the primary infection stage [9]. However, the authors noted that there was no conclusive biological evidence to this and assigned $\delta = 0$ if the bypass is not possible. Thus $\delta \geq 0$. With the above assumptions and the facts on the condom use we have the rate of change of S_v as:

$$\frac{dS_v}{dt} = \xi S_u - \beta(N)\left[(1-r)(1-\delta)(1-\varepsilon_2) + r(1-\delta)(1-\varepsilon_2)(1-\varepsilon_1)\right]\left(I_1 + n_2 I_2 + n_3 I_3\right)S_v - wS_v - \mu S_v$$

$$= \xi S_u - \beta(N)(1-\delta)(1-\varepsilon_2)(1-r\varepsilon_1)\ \left(I_1 + n_2 I_2 + n_3 I_3\right)S_v - (w+\mu)S_v \qquad (2)$$

For subpopulation of individuals in the infected I_1, I_2, I_3 stages, condoms use is invalid since these groups are already infected and therefore cannot be protected from HIV infection any more. Thus individual in the primary infection stage ($I_{(t)}$) is generated by the infection of the susceptible and failed vaccinated individual without and with failed condom and decreased by progression to secondary infection stage (at per capital rate σ_1) and natural death (at per capital rate μ). The rate of change of I_1 is

$$\frac{dI_1}{dt} = (1 - r\varepsilon_1)\ \beta(N)(I_1 + n_2 I_2 + n_3 I_3)\left[S_u + (1-\varepsilon_2)S_v\right] - \sigma_1 I_1 - \mu I_1 \qquad (3)$$

The sub-population of individuals at the secondary stage ($I_2^{(t)}$) is generated by the infection of some vaccinated susceptible individuals (proportion δ), the progression of individuals in the primary infection stage (at per capital rate σ_1) and via vaccine-induced conversion of individual in the Aids stage to the asymptomatic stage (at per capital rate m). It is worthy to note that in the absence of cure for HIV, the current anti-HIV therapeutic treatment are aimed at reducing viral load to level consistent with the secondary stage of infection, there by elongating the life span of those treated individuals [21]. The secondary infection class is diminished by progression to Aids stage (at per capital rate σ_2) and natural death (at per capital rat μ). Thus:

$$\frac{dI_2}{dt} = \delta(1-\varepsilon_2)\ (1-r\varepsilon_1)\beta(N)(I_1 + n_2 I_2 + n_3 I_3)S_v + \sigma_1 I_1 + mI_3 - \sigma_2 I_2 - \mu I_2 \ldots\ldots (4)$$

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The sub-population of individuals in the Aids stage of infection $I_3^{(t)}$ is generated by progression to Aids of individuals in the secondary stage (at per capital rate σ_2). It is diminished by vaccine-induced therapeutic effect (at per capital rate m), natural death (at per capital rate μ) and disease-induced death (at per capital rate ψ), to give

$$\frac{dI_3}{dt} = \sigma_2 I_2 - mI_3 - \mu I_3 - \psi I_3 \qquad (5)$$

Equations (1)-(5), representing the transmission dynamics of HIV with imperfect vaccine and condom use, were well fathomed since for r = 0, which is the rate of condom use, reduces the equation to the derivations of [9] which discussed the transmission with imperfect vaccine only.
By assumption, all the parameters of the models are assumed non-negative with natural death rate, μ, being positive, i.e μ > 0. Also $I_1 + n_2I_2 + n_3I_3 > 0$ at time t = o, since, the equation (1)-(5) monitors human population and that all the stated variables are non-negative. Thus adding equation (1) – (5) gives:

$$\frac{dN}{dt} = \pi - \mu N - \psi I_3$$

Consequently, in the absence of HIV infection, $N \rightarrow \frac{\pi}{\mu}$ as $t \rightarrow \infty$. Then $\frac{\pi}{\mu}$ is an upper bound of N(t) provided that $N(0) \leq \frac{\pi}{\mu}$. Thus the feasible region is

$$D = \left(S_u, S_v, I_1, I_2, I_3\right) \varepsilon\ R^5 : S_u + S_v + I_1 + I_2 + I_3 \leq \frac{\pi}{\mu}\ \text{) is a positive variant.}$$

Consequently, in the region D, the usual existence, uniqueness and continuation results hold for the system (1)-(5).

III. STABILITY ANALYSIS OF DISEASE-FREE EQUILIBRIUM

Local Stability

The model has a diseases-free (DFE) obtained by setting the right-hand side of (1) – (5) to zero and solving for S_u and S_V.

$$\sum\nolimits_0 : (S_u^*,\ S_v^*,\ I_1^*,\ I_2^*, I_3^*) = \left(\frac{\pi(w+\mu)}{\mu(w+\xi+\mu)}, \frac{\pi\xi}{\mu(w+\xi+\mu)}, 0, 0, 0\right)$$

Following [22], and [9], the next generation matrixes, for the system (1) – (5) are as follows:
Adopting the notations as used by the authors above, the non-negative Matrix, V, for the new infection terms and the remaining transfer terms respectively are given by:

$$F = \begin{bmatrix} B(N^*)Z^* & B(N^*)Z^*n_2 & B(N^*)Z^*n_3 \\ B(N^*)\delta(1-\varepsilon_2)(1-r\varepsilon_1)S_v^* & B(N^*)\delta(1-\varepsilon_2)(1-r\varepsilon_1)n_2S_v^* & B(N^*)\delta(1-\varepsilon_2)(1-r\varepsilon_1)n_3S_v^* \\ 0 & 0 & 0 \end{bmatrix}$$

(7)

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International Journal of Scientific and Allied Research
[IJSAR]

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http://www.mdcjournals.org/ijsar-jmas...html

$$V = \begin{bmatrix} \sigma_1 + \mu & 0 & 0 \\ -\sigma_1 & \sigma_2 + \mu & -m \\ 0 & -\sigma_2 & m + \mu + \psi \end{bmatrix} \qquad (8)$$

With $N^* = S_u^* + S_v^* + I_1^* + I_2^* I_3^* = \frac{\pi}{\mu}$

and $Z^* = \left[(1 - r\varepsilon_1)(S_u^* + (1-\delta)(1-\varepsilon_2)S_v^*\right]$

The vaccination and condom use reproduction number denoted by R_{VC} is then given by:
$R_{vc} = \rho\left(FV^{-1}\right)$, where ρ denotes the spectrum radius (dominant eigenvalue).

It follows that detV = $(\sigma_1 + \mu)[(\sigma_2 + \mu)(m + \mu + \psi) - \sigma_2 m]$

$= (\sigma_1 + \mu)[(\sigma_2 + \mu)(\mu + \psi) + \mu m]$

To derive V^{-1}, we get first the co-factor, C^+, of V. That is:

$a_{11} = (\sigma_2 + \mu)(m + \mu + \psi) - \sigma_2 m = (\sigma_2 + \mu)(\mu + \psi) + \mu m$

$a_{12} = \sigma_1 (m + \mu + \psi)$

$a_{13} = \sigma_1 \sigma_2$

$a_{21} = 0$

$a_{22} = (\sigma_1 + \mu)(m + \mu + \psi)$

$a_{23} = (\sigma_1 + \mu)\sigma_2$

$a_{31} = 0$

$a_{32} = (\sigma_1 + \mu)m$

$a_{33} = (\sigma_1 + \mu)(\sigma_2 + \mu)$

$$C = \begin{pmatrix} (\sigma_2 + \mu)(\mu + \psi) & \sigma_1 (m + \mu + \psi) & \sigma_1 \sigma_2 \\ 0 & (\sigma_1 + \mu)(m + \mu + \psi) & (\sigma_1 + \mu)\sigma_2 \\ 0 & (\sigma_1 + \mu)m & (\sigma_1 + \mu)(\sigma_2 + \mu) \end{pmatrix}$$

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$$C^T = \begin{pmatrix} (\sigma_2+\mu)(\mu+\psi)+\mu\ m & 0 & 0 \\ \sigma_1(m+\mu+\psi) & (\sigma_1+\mu)(m+\mu+\psi) & (\sigma_1+\mu)m \\ \sigma_1\sigma_2 & (\sigma_1+\mu)\sigma_2 & (\sigma_1+\mu)(\sigma_2+\mu) \end{pmatrix}$$

$$\therefore\ V^{-1} = \frac{1}{\det V}\begin{pmatrix} (\sigma_2+\mu)(\mu+\psi)+\mu\ m & 0 & 0 \\ \sigma_1(m+\mu+\psi) & (\sigma_1+\mu)(m+\mu+\psi) & (\sigma_1+\mu)m \\ \sigma_1\sigma_2 & (\sigma_1+\mu)\sigma_2 & (\sigma_1+\mu)(\sigma_2+\mu) \end{pmatrix}$$

Let $\beta^* = \beta(N^*)Z^* = \beta(N^*)(1-r\varepsilon_1)(S_u^* + (1-\delta)(1-\varepsilon_2)S_v^*$

and $\zeta^* = \beta(N^*)\delta(1-\varepsilon_2)((1-r\varepsilon_1)S_v^*$ in (7), the matrix, F, of new infection terms to have:

$$F = \begin{pmatrix} \beta^* & \beta^*_{n_2} & \beta^*_{n_3} \\ \zeta^* & \zeta^*_{n_2} & \zeta^*_{n_3} \\ 0 & 0 & 0 \end{pmatrix}$$

Then, FV^{-1} is given as: $\frac{1}{\det V}\begin{bmatrix} A & B & C \\ D & E & F \\ 0 & 0 & 0 \end{bmatrix}$, For

$\mathbf{A} = B^*\left[(\sigma_2+\mu)(\mu+\psi)+\mu m\right]+Bn_2^*\left[\sigma_1(m+\mu+\psi)\right]+B^*n_3\sigma_1\sigma_2$

$\mathbf{B} = B^*n_2(\sigma_1+\mu)(m+\mu+\psi)+B^*n_3(\sigma_1\mu)\sigma_2$

$\mathbf{C} = B^*n_2(\sigma_1+\mu)m+B^*n_3(\sigma_1+\mu)(\sigma_2+\mu)$

$\mathbf{D} = \zeta^*\left[(\sigma_2+\mu)(\mu+\psi)+\mu m\right]+\zeta^*n_2\left[\sigma_1(m+\mu+\psi)\right]+\zeta^*n_3[\sigma_1\sigma_2]$

$\mathbf{E} = \zeta^*n_2(\sigma_1+\mu)(m+\mu+\psi)+\zeta^*n_3(\sigma_1+\mu)\sigma_2$

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F= $\zeta^* n_2(\sigma_1+\mu)m+\zeta^* n_3(\sigma_1+\mu)(\sigma_2+\mu)$

Thus ρFV^{-1}, the vaccination and condom use reproduction number where ρ is the dominant eigenvalue which we denote as R_{VC} is then;

$$R_{VC}=\frac{1}{\det v}\{\ B^*[(\sigma_2+\mu)(\mu+\psi)+\mu m]+B^* n_2[\sigma_1(m+\mu+\psi)]+B^* n_3\sigma_1\sigma_2+$$

$$\zeta^* n_2(\sigma_1+\mu)(m+\mu+\psi)+\zeta^* n_3(\sigma_1+\mu)\sigma_2\ \}$$

$$=\left(\beta^*[\frac{1}{\sigma_1+\mu}+\frac{n_2\sigma_1(m+\mu+\psi)}{\det V}+\frac{n_3\sigma_1\sigma_2}{\det V}]+\frac{\zeta^*}{\det V}[n_2(\sigma_1+\mu)(m+\mu+\psi)+n_3\sigma_2(\sigma_1+\mu)]\right)$$

By using the theorem 2 of [22] we have the following results.

Lemma: The disease-free equilibrium ε_0 of (1) – (5) given by (6) is locally asymptotically stable if $R_{VC}<1$ and unstable if $R_{VC}>1$.

In the absence of condom use, but with vaccination, $\varepsilon_1=0$. The vaccination and condom use reproduction number reduces to only vaccination reproduction number, R_V, i.e

$$R_v = K_1\frac{1}{\sigma_1+\mu}+\frac{n_2\sigma_1(m+\mu+\psi)}{\det V}+\frac{n_3\sigma_1\sigma_2}{\det V}+\frac{K_2}{\det V}(n_2(m+\mu+\psi)+n_3\sigma_2)(\sigma_1+\mu)$$

Where $K_1 = \beta(N^*)(S_u^*+(1-\delta)(1-\varepsilon_2)S_v^*)$

$K_2 = \beta(N^*)\delta(1-\varepsilon_2)S_v^*$

With condom use only as the only HIV preventive measure, we have that ξ=m = $S_v^*=0$. Then the condom reproductive number, R_c, will give:

$$R_C = \beta(N^*)(1-r\varepsilon_1)S_u^*(\frac{1}{\sigma_1+\mu}+\frac{n_2\sigma_1}{(\sigma_1+\mu)(\sigma_2+\mu)}+\frac{n_3\sigma_1\sigma_2}{(\sigma_1+\mu)(\mu+\psi)(\sigma_2+\mu)})$$

Without any HIV preventive measure, that is, no condom or vaccination, here ξ = ε_1 = m = 0 and of course $S_v^*=0$. The basic reproduction number, R_0, will then be:-

$$R_0 = \beta(N^*)S_u^*(\frac{1}{\sigma_1+\mu}+\frac{n_2\sigma_1}{(\sigma_1+\mu)(\sigma_2+\mu)}+\frac{n_3\sigma_1\sigma_2}{(\sigma_1+\mu)(\mu+\psi)(\sigma_2+\mu)})$$

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