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Analysis of a Mathematical Model Incorporating Dual Protection and ART Adherence for a High Risk HIV Population

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Abstract. In this paper, a mathematical model for dual protection, incorporating PrEP and Condom use, and ART adherence is formulated, based on a system of ordinary differential equations and analyzed. The results obtained from stability analysis indicate that provided the basic reproductive number is less than unity, the disease free equilibrium point is both locally and globally asymptotically stable, while provided the basic reproductive number is greater than unity, the endemic equilibrium point exists and is locally asymptotically stable. Sensitivity analysis is undertaken to establish the most sensitive model parameter. The most sensitive parameter to the value of R_0 is β_1, the mean contact rate with undiagnosed infectives. This implies that in order to control the spread of HIV in a high risk population, efforts should be geared towards reducing the undiagnosed by testing and enrolling them on ART treatment. This in turn lowers their infectivity as well as chances of progressing to the AIDS class.

1. Introduction

Numerous efforts have been made in an attempt to control the spread of HIV, with the aim of reducing its effects. According to the UNAIDS fact sheet 2019, at least 1.7 million new HIV infections were reported by the end of the year 2018 [13].

Scientific as well as public health interventions such as testing and counseling, circumcision, use of PrEP (Pre-Exposure Prophylaxis), PeP (Post-Exposure Prophylaxis), condom use, and antiretroviral therapy have been proposed and utilized. Consistent use of condoms can result to 80% reduction in HIV incidence among the heterosexual population [2], while the effectiveness of condom

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use for men who have sex with men is 70% [3]. Proper use (correctly and consistently) as well as quality concerns have been directly attributed to the success of this approach.

In 2012, the U.S Food and Drug Administration (FDA) approved the use of Truvada for PrEP as an oral pill taken once a day [14]. Numerous efficacy trials(by iPrEX, Partners PrEP, TDF2, e.t.c) have since been conducted to ascertain the potential of PrEP to prevent HIV infection. The iPrEX trial demonstrated that PrEP has the potential of reducing the risk of HIV infection among transgender women, bisexual men, as well as men who have sex with men [8]. Two major studies; Partners PrEP, and TDF2 demonstrated the effectiveness of PrEP among heterosexual men and women. Out of all these studies, none displayed a 100% effectiveness [11]. Adherence has been found to be directly correlated with the effectiveness of PrEP [11]. In the absence of adherence, which guarantees efficacy, PrEP failures have been characterized by; system failures, people failures, Doctor failures, drug failures, as well as assay failures [9]. These failures expose PrEP users to the risk of HIV infection hence the need for additional protection whenever PrEP has been utilized.

The nature of storage, date of manufacture, religious as well as socio-cultural beliefs also influence how each HIV prevention venture is utilized. The challenges experienced when various approaches are employed in an attempt to control the spread of HIV infection in a high risk population form the basis for the need to use dual protection in order to achieve maximum protection. A combination prevention approach as proposed by [6], based on proven efficacy interventions, provides one with the best opportunity to curb the spread of HIV among the high risk population.

In this study, we propose a mathematical model of dual protection against HIV infection by the use of condom and PrEP, and adherence to ART treatment, while focusing on the high risk population collectively. Earlier studies have either narrowed down to a particular category of persons at high risk of infection [3], or have used a combination of prevention techniques where one technique acts as a supplement to the other [4], [5]. The study will focus on the impact of dual protection on reducing the number of new infections, and that of ART adherence in ensuring those who are infected remain less infectious.

2. Model Formulation and Description

The population is subdivided into the classes; susceptible, infected, and Aids individuals. The susceptible class has been further subdivided into two compartments on the basis of degree of risk of infection. These include susceptible individuals at high risk of infection, denoted by (S_H), and those at low risk, denoted by (S_L). The high risk population incorporates mainly commercial sex workers, men who have sex with men (MSM), and HIV-Discordant couples [8]. The infected class is subdivided into two compartments; those who are unaware of their HIV status (I), and those who have been diagnosed and consequently enrolled for treatment (T_D). The individuals who are unaware of their HIV status may progress to the T_D compartment after successful HIV

awareness campaigns that will persuade them to get tested, or when they develop HIV symptoms and consequently enroll for ART treatment. If ART treatment fails, the individual progresses to the AIDS compartment. This happens when there is lack of adherence to ART, which allows the virus to multiply, thus increasing the plasma viral load. This results in weakening of the immune system and hence the AIDS symptoms begin to manifest. The AIDS compartment comprises of those who posses full blown symptoms, and are mostly bedridden, they thus do not significantly contribute to the spread of the disease. Exit from the AIDS class is through natural death.Thus, considering a population of size $N(t)$, at a time t,

$$N(t) = S_H(t) + S_L(t) + I(t) + T_D(t) + A(t). \tag{1}$$

The following interventions have been incorporated in the model;

(a) $0 \leq \phi_1 \leq 1$ - measures PrEP effectiveness, including its awareness and proper use as a means to prevent susceptible individuals from being infected. Thus, $(1 - \phi_1)$ measures PrEP failure.

(b) $0 \leq \phi_2 \leq 1$- measures condom effectiveness as a result of proper use, following adequate awareness campaigns and availability. Thus, $(1 - \phi_2)$ measures condom failure.

(c) $0 \leq \phi_3 \leq 1$ - measures the efficacy of ART treatment, including uptake with proper adherence, with the aim of reducing the plasma viral load and reconstructing the individual's immune system hence making them less infectious.

Movement of individuals from the susceptible to infected and then to the AIDS classes is illustrated by the compartmental model shown in Figure 1.

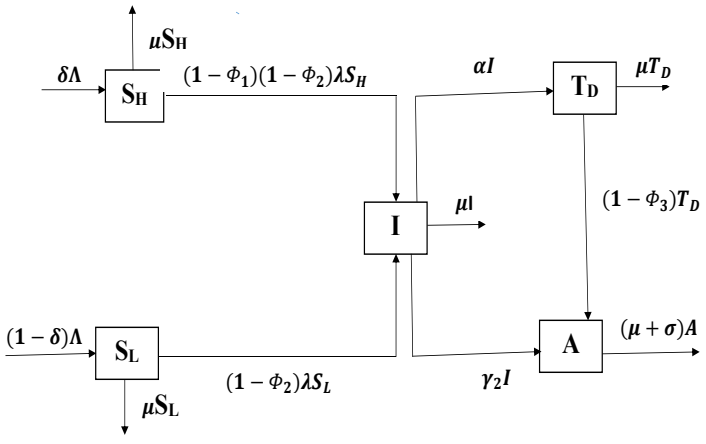

FIGURE 1. Compartmental Model.

The following symbols will be used to represent various phenomena as described in Table 1.

Symbol	Description
Λ	constant rate of recruitment of susceptible upon becoming sexually active.
δ	proportion of susceptible individuals at high risk of infection.
$(1-\delta)$	proportion of susceptible population at low risk of HIV infection.
λ	rate of acquisition of an infection by susceptibles.It is given by; $\lambda = (\frac{\beta_1 I + \beta_2 T_D}{N})$, where β_1, and β_2 are the mean contact rates for the susceptible individuals with I and T_D respectively.
μ	natural removal rate by death.
σ	aids induced mortality.
α	represents the proportion of infected individuals who upon being tested and found to be HIV positive,they enroll for ART treatment.
γ_2	represents the proportion of infected individuals who do not get tested hence remain undiagnosed until they begin to exhibit AIDS symptoms.

TABLE 1. Table showing symbols and their description

From the dynamics described above, the following system of ordinary differential equations is formulated.

$$\begin{aligned}
\frac{dS_H}{dt} &= \delta\Lambda - (1-\phi_1)(1-\phi_2)\lambda S_H - \mu S_H \\
\frac{dS_L}{dt} &= (1-\delta)\Lambda - (1-\phi_2)\lambda S_L - \mu S_L \\
\frac{dI}{dt} &= (1-\phi_1)(1-\phi_2)\lambda S_H + (1-\phi_2)\lambda S_L - \alpha I - \gamma_2 I - \mu I \\
\frac{dT_D}{dt} &= \alpha I - (\gamma_3 + \mu) T_D \\
\frac{dA}{dt} &= \gamma_2 I + \gamma_3 T_D - (\mu + \sigma) A. \qquad (2)
\end{aligned}$$

3. Model Analysis

It can be shown that the solutions for the system of ordinary differential equations (2) are all positive and bounded for all $t > 0$, with positive initial conditions in the feasible region $\Gamma = \left\{ (S_H(t), S_L(t), I(t), (T_D(t), A(t)) \in \mathbb{R}^5_+ : N(t) \leq \frac{\Lambda}{\mu} \right\}$. It therefore suffices to study the dynamics of the system (2) in this region.

The mathematical model developed in (2) has two unique equilibrium points, that is, the Disease Free Equilibrium (D.F.E), and the Endemic Equilibrium (E.E). The D.F.E is obtained by setting

$I = T_D = A = 0$ in (2) to yield

$$E_0 = \left(\frac{\delta\Lambda}{\mu}, \frac{(1-\delta)\Lambda}{\mu}, 0, 0, 0 \right). \tag{3}$$

The basic reproduction number (R_0) of the system (2), computed using the next generation matrix approach [10] is given by

$$R_0 = \frac{\beta_3}{Q_1} + \frac{\alpha\beta_4}{Q_1 Q_2}. \tag{4}$$

By [10, Theorem 2], the following result is thus established.

Theorem 3.1. *The Disease Free Equilibrium of the model (2), $E_0 = \left(\frac{\delta\Lambda}{\mu}, \frac{(1-\delta)\Lambda}{\mu}, 0, 0, 0 \right)$, is locally asymptotically stable whenever $R_0 < 1$ and unstable otherwise.*

Proof. The proof follows immediately from the computation of R_0 above and Theorem 2 of Van den Driessche and Watmough [10]. □

Mathematically, Theorem (3.1) implies that whenever there is a small perturbation on the system, the system returns to the disease free equilibrium. Epidemiologically, this implies that when a few HIV infectious individuals are introduced in a population that is fully susceptible to HIV infection, the disease dies out whenever $R_0 < 1$, otherwise, the disease will spread. It is therefore necessary to show that eliminating HIV in a population is independent of the size of the initial sub-population by proving the global asymptotic stability of the disease free equilibrium.

Theorem 3.2. *The Disease Free Equilibrium $E_0 = \left(\frac{\delta\Lambda}{\mu}, \frac{(1-\delta)\Lambda}{\mu}, 0, 0, 0 \right)$ of the system (2) is globally asymptotically stable whenever $R_0 < 1$.*

Proof. Castillo Chavez's theorem [1] is used to analyze the global asymptotic stability of the mathematical model (2) such that $E_0 = (\mathbf{X}^*, \mathbf{0})$,
$\mathbf{X} = (S_H, S_L)$ and $\mathbf{Z} = (I, T_D, A)$.
Now;

$$F(\mathbf{X},\mathbf{0}) = \begin{pmatrix} \delta\Lambda - \mu S_H \\ (1-\delta)\Lambda - \mu S_L \end{pmatrix} \text{ and } G(\mathbf{X},\mathbf{Z}) = P\mathbf{Z} - \tilde{G}(\mathbf{X},\mathbf{Z}).$$

Matrix P is given by

$$\begin{pmatrix} \frac{h_1\beta_1 S_H}{N} + \frac{(1-\phi_2)\beta_1 S_L}{N} - (\alpha + \gamma_2 + \mu) & \frac{h_1\beta_2 S_H}{N} + \frac{(1-\phi_2)\beta_2 S_L}{N} & 0 \\ \alpha & -(\gamma_3 + \mu) & 0 \\ \gamma_2 & \gamma_3 & -(\sigma + \mu) \end{pmatrix},$$

where $h_1 = (1-\phi_1)(1-\phi_2)$, and $P\mathbf{Z}$ is given by

$$\begin{pmatrix} \frac{h_1\beta_1 I S_H}{N} + \frac{(1-\phi_2)\beta_1 I S_L}{N} - (\alpha + \gamma_2 + \mu) + \frac{h_1\beta_2 T_D S_H}{N} + \frac{(1-\phi_2)\beta_2 T_D S_L}{N} \\ \alpha I - (\gamma_3 + \mu) T_D \\ \gamma_2 I + \gamma_3 T_D - (\sigma + \mu) A \end{pmatrix}.$$

Moreover, $G(\mathbf{X},\mathbf{Z})$ is given by

$$\begin{pmatrix} (1-\phi_1)(1-\phi_2)\left(\frac{\beta_1 I+\beta_2 T_D}{N}\right)S_H+(1-\phi_2)\left(\frac{\beta_1 I+\beta_2 T_D}{N}\right)S_L-(\alpha+\gamma_2+\mu)I \\ \alpha I-(\gamma_3+\mu)T_D \\ \gamma_2 I+\gamma_3 T_D-(\sigma+\mu)A \end{pmatrix},$$

and therefore $\tilde{G}(\mathbf{X},\mathbf{Z}) = P\mathbf{Z} - G(\mathbf{X},\mathbf{Z}) = \begin{pmatrix} \tilde{G}_1(\mathbf{X},\mathbf{Z}) \\ \tilde{G}_2(\mathbf{X},\mathbf{Z}) \\ \tilde{G}_3(\mathbf{X},\mathbf{Z}) \end{pmatrix} = \begin{pmatrix} \mathbf{0} \\ \mathbf{0} \\ \mathbf{0} \end{pmatrix}$. Hence conditions H_1 and H_2 are satisfied. Also from Theorem (3.1), E_0 is locally asymptotically stable whenever $R_0 < 1$. Therefore following Castillo Chavez's theorem, E_0 is globally asymptotically stable whenever $R_0 < 1$, as desired. □

This implies that with a large perturbation of the disease free equilibrium, solutions of the model represented by the system (3.2) converge to D.F.E whenever $R_0 < 1$. Epidemiologically, this implies that if a sufficiently large number of HIV infected individuals are introduced in a population that is fully susceptible to HIV infection, the disease will die out whenever $R_0 < 1$.

3.1. **Existence of the Endemic Steady State.**

Theorem 3.3. *An endemic equilibrium point $E_1 = (S_H^{**}, S_L^{**}, I^{**}, T_D^{**}, A^{**})$, of the system (2) exists whenever $R_0 > 1$.*

Proof. Equating the right hand side of each equation in the system (2) to zero and simplifying yields;

$$\delta\Lambda-(1-\phi_1)(1-\phi_2)\left(\frac{\beta_1 I^{**}+\beta_2 T_D^{**}}{N}\right)S_H^{**}-\mu S_H^{**}=0, \tag{5}$$

$$(1-\delta)\Lambda-(1-\phi_2)\left(\frac{\beta_1 I^{**}+\beta_2 T_D^{**}}{N}\right)S_L^{**}-\mu S_L^{**}=0, \tag{6}$$

$$(1-\phi_1)(1-\phi_2)\left(\frac{\beta_1 I^{**}+\beta_2 T_D^{**}}{N}\right)S_H^{**}+$$

$$(1-\phi_2)\left(\frac{\beta_1 I^{**}+\beta_2 T_D^{**}}{N}\right)S_L^{**}-(\alpha+\gamma_2+\mu)I^{**}=0, \tag{7}$$

$$\alpha I^{**}-(\gamma_3+\mu)T_D^{**}=0, \tag{8}$$

$$\gamma_2 I^{**}+\gamma_3 T_D^{**}-(\mu+\sigma)A^{**}=0. \tag{9}$$

From equation (8), $T_D^{**} = \frac{\alpha}{Q_2} I^{**}$.

Substituting for T_D^{**t} in equation (9) and simplifying gives $A^{**} = \left(\frac{\gamma_2}{Q_3} + \frac{\alpha\gamma_3}{Q_2 Q_3}\right) I^{**}$.

Using equation (5) and substituting T_D^{**} gives

$$\delta\Lambda N - (1-\phi_1)(1-\phi_2)\left(\beta_1 + \frac{\beta_2\alpha}{Q_2}\right) I^{**}S_H^{**} - \mu N S_H^{**} = 0$$

$$\Rightarrow S_H^{**} = \frac{\delta\Lambda N}{a_1 I^{**} + \mu N},$$

where $a_1 = (1-\phi_1)(1-\phi_2)\left(\beta_1 + \frac{\beta_2\alpha}{Q_2}\right)$. In a similar manner, S_L^{**} is expressed as $S_L^{**} = \frac{(1-\delta)\Lambda N}{a_2 I^* + \mu N}$, where $a_2 = (1-\phi_2)\left(\beta_1 + \frac{\beta_2\alpha}{Q_2}\right)$.
Using equation (7) and substituting for S_H^{**} and S_L^{**},we obtain

$$\frac{a_1 I^{**}\delta\Lambda}{a_1 I^{**} + \mu N^{**}} + \frac{a_2 I^{**}(1-\delta)\Lambda}{a_2 I^{**} + \mu N^{**}} - Q_1 I^{**} = 0 \tag{10}$$

Thus from equation (10),

$$\left(\frac{a_1\delta\Lambda}{a_1 I^{**} + \mu N^{**}} + \frac{a_2(1-\delta)\Lambda}{a_2 I^{**} + \mu N^{**}} - Q_1\right) I^{**} = 0. \tag{11}$$

From equation (11), $I^{**} = 0$ corresponds to the disease free equilibrium point of the system (2), denoted by (E_0). The other solution of (11) when $I^{**} \neq 0$ corresponds to the endemic equilibrium point of the system such that,

$$\frac{a_1\delta\Lambda}{a_1 I^{**} + \mu N^{**}} + \frac{a_2(1-\delta)\Lambda}{a_2 I^{**} + \mu N^{**}} - Q_1 = 0. \tag{12}$$

Multiplying through by $(a_1 I^{**} + \mu N^{**})(a_2 I^{**} + \mu N^{**})$ yields

$$C I^{**2} + D I^{**} + E = 0. \tag{13}$$

where: $C = -Q_1 a_1 a_2$, $D = (a_1 a_2 \delta\Lambda + a_1 a_2(1-\delta)\Lambda) - (Q_1 a_1 \mu N + Q_1 a_2 \mu N)$, and $E = a_1\delta\Lambda\mu N + a_2(1-\delta)\Lambda\mu N - Q_1\mu N\mu N$.
The endemic equilibrium of the system exists if the roots of equation (13) are real and positive. Descarte's rule of signs is used to check the possible number of real roots of the polynomial. The number of positive real roots is equal to the number of sign changes in the coefficients of the terms of a polynomial [15]. Considering that all the parameters used are positive, the sign of C is negative. The sign of E is then checked as follows;

$$\begin{aligned} E &= a_1\delta\Lambda\mu N + a_2(1-\delta)\Lambda\mu N - Q_1\mu N\mu N \\ &= (1-\phi_1)(1-\phi_2)\left(\beta_1 + \frac{\beta_2\alpha}{Q_2}\right)\delta\Lambda\mu N + \\ &\quad (1-\phi_2)\left(\beta_1 + \frac{\beta_2\alpha}{Q_2}\right)(1-\delta)\Lambda\mu N - Q_1\mu N\mu N \\ &= (1-\phi_1)(1-\phi_2)(\beta_1 + \beta_2\alpha)\delta\Lambda\mu N + \\ &\quad (1-\phi_2)(\beta_1 + \beta_2\alpha)(1-\delta)\Lambda\mu N - Q_1 Q_2\mu N\mu N \end{aligned}$$

Using $R_0 = \frac{\beta_3}{Q_1} + \frac{\alpha\beta_4}{Q_1Q_2}$and the limiting value of $N = \frac{\Lambda}{\mu}$, we obtain $E = (R_0 - 1)\Lambda^2$. Thus $E > 0$ iff $R_0 > 1$. Since C is negative, and E is positive, we see that there is at least one sign change regardless of the sign of D. This implies that equation (13) has at least one positive real root. Hence an endemic equilibrium point of the system (2) exists whenever $R_0 > 1$. □

3.2. **Local Stability of the Endemic Equilibrium.** At the endemic equilibrium, there is persistence of HIV infection in the population.

Theorem 3.4. *The endemic equilibrium point* $E_1 = (S_H^{**}, S_L^{**}, I^{**}, T_D^{**}, A^{**})$ *of system* (2) *is locally asymptotically stable if* $R_0 > 1$.

Proof. The Jacobian matrix of the system (2) evaluated at endemic equilibrium is

$$J(E_1) = \begin{pmatrix} -b_1 & 0 & -b_2 & -b_3 & 0 \\ 0 & -b_4 & -b_5 & -b_6 & 0 \\ b_7 & b_8 & b_9 - Q_1 & b_{10} & 0 \\ 0 & 0 & \alpha & -Q_2 & 0 \\ 0 & 0 & \gamma_2 & \gamma_3 & -Q_3 \end{pmatrix}$$

where

$b_1 = \frac{(1-\phi_1)(1-\phi_2)(\beta_1Q_2+\beta_2\alpha)\mu I^{**}+Q_2\mu\Lambda}{Q_2\Lambda}$, $b_2 = \frac{(1-\phi_1)(1-\phi_2)\beta_1a_1\delta\Lambda I^{**}}{a_1I^{**}+\Lambda}$, $b_3 = \frac{(1-\phi_1)(1-\phi_2)\beta_2a_1\delta\Lambda I^{**}}{a_1I^{**}+\Lambda}$

$b_4 = \frac{(1-\phi_2)(\beta_1Q_2+\beta_2\alpha)\mu I^{**}+Q_2\mu\Lambda}{Q_2\Lambda}$, $b_5 = \frac{(1-\phi_2)(1-\delta)\Lambda\beta_1a_2I^{**}}{a_2I^{**}+\Lambda}$, $b_6 = \frac{(1-\phi_2)(1-\delta)\Lambda\beta_2a_2I^{**}}{a_2I^{**}+\Lambda}$

$b_7 = \frac{(1-\phi_1)(1-\phi_2)(\beta_1Q_2+\beta_2\alpha)\mu I^{**}+Q_2\mu\Lambda}{Q_2\Lambda}$, $b_8 = \frac{(1-\phi_2)(\beta_1Q_2+\beta_2\alpha)\mu I^{**}+Q_2\mu\Lambda}{Q_2\Lambda}$

$b_9 = \frac{(1-\phi_1)(1-\phi_2)\beta_1a_1\delta\Lambda I^{**}}{a_1I^{**}+\Lambda} + \frac{(1-\phi_2)(1-\delta)\Lambda\beta_1a_2I^{**}}{a_2I^{**}+\Lambda}$, $b_{10} = \frac{(1-\phi_1)(1-\phi_2)\beta_2a_1\delta\Lambda I^{**}}{a_1I^{**}+\Lambda} + \frac{(1-\phi_2)(1-\delta)\Lambda\beta_2a_2I^{**}}{a_2I^{**}+\Lambda}$

Clearly, $-Q_3$ is an eigenvalue of the Jacobian matrix $J(E_1)$. The other eigenvalues can be computed by finding the solution to the equation

$$P(\lambda) = \begin{vmatrix} \lambda + b_1 & 0 & -b_2 & -b_3 \\ 0 & \lambda + b_4 & -b_5 & -b_6 \\ b_7 & b_8 & \lambda - (b_9 + Q_1) & b_{10} \\ 0 & 0 & \alpha & \lambda + Q_2 \end{vmatrix} = 0$$

The characteristic equation of $J(E_1)$is then given by;

$$P(\lambda) = \lambda^4 + c_0\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0 \tag{14}$$

where;

$c_0 = b_1 + b_4 - b_9 - Q_1 + Q_2$

$c_1 = b_1b_4 + b_2b_7 + b_5b_8 - b_1b_9 - b_4b_9 - \alpha b_{10} - b_1Q_1 - b_4Q_1 + b_1Q_2 + b_4Q_2 - b_9Q_2 - Q_1Q_2$

$c_2 = -\alpha b_3b_7 + b_2b_4b_7 + b_1b_5b_8 - \alpha b_6b_8 - b_1b_4b_9 - alphab_1b_{10} - alphab_4b_{10} - b_1b_4Q_1 + b_1b_4Q_2 + b_2b_7Q_2 + b_5b_8Q_2 - b_1b_9Q_2 - b_4b_9Q_2 - b_1Q_1Q_2 - b_4Q_1Q_2$

$c_3 = -\alpha b_3b_4b_7 - \alpha b_1b_6b_8 - \alpha b_1b_4b_{10} + b_2b_4b_7Q_2 + b_1b_5b_8Q_2 - b_1b_4b_9Q_2 - b_1b_4Q_1Q_2$

The number of negative zeros of equation (14) depends on the signs of c_0, c_1, c_2 and c_3. Descarte's

Rule of Signs is applied to study the number of negative real roots of the polynomial$P(\lambda_1)$ comprising of the coefficients c_0, c_1, c_2 and c_3 given by;

$$P(\lambda_1) = c_0\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0 \tag{15}$$

Descarte's rule of signs states that the number of negative real zeros of $P(\lambda)$ is either equal to the variations in sign of $P(-\lambda)$ or less than this by an even number [15]. The possibilities of negative real zeros of $P(\lambda)$, is as summarized in Table 2. The maximum number of variations of signs in $P(-\lambda)$ is 3, hence the characteristic polynomial (15) has three negative roots. Thus $P(-\lambda) = \lambda^4 - c_0\lambda^3 + c_1\lambda^2 - c_2\lambda + c_3 = 0$ has negative roots.Therefore, given that cases 1-8 in Table 1 are satisfied, model (2) is locally asymptotically stable if $R_0 > 1$. □

Table 2. The Zeros of the characteristic equation (14)

Cases	c_0	c_1	c_2	c_3	$R_0 > 1$	Sign Change	No. of - Roots
1	+	−	−	+	$R_0 > 1$	2	2,0
2	+	−	+	+	$R_0 > 1$	2	2,0
3	−	−	+	−	$R_0 > 1$	2	2,0
4	+	+	−	−	$R_0 > 1$	1	0
5	−	−	+	+	$R_0 > 1$	1	0
6	+	+	+	−	$R_0 > 1$	1	0
7	−	+	−	+	$R_0 > 1$	3	3,1
8	−	−	−	−	$R_0 > 1$	0	0

This implies that for a small pertubation of the E_1, solutions of the mathematical model represented by the system (2) always converge to E_1, whenever $R_0 > 1$. Epidemiologically, it implies that if a few HIV infected individuals are introduces in a fully susceptible population, the disease will persist provided $R_0 > 1$.

4. Sensitivity Analysis

In mathematical modeling, Sensitivity refers to the degree to which a given input parameter in a mathematical model influences its output. Sensitive parameters are thus those that cause a significant impact on the disease transmission dynamics. Sensitivity analysis will aid in identifying the parameters which greatly impact on the value of the basic reproductive number R_0, and hence ought to be targeted when coming up with intervention strategies. The sensitivity of model parameters is calculated using the normalized forward sensitivity index. The normalized forward sensitivity index of the basic reproductive number is given by $S_w^{R_0} = \frac{\partial R_0}{\partial w} \times \frac{w}{R_0}$, where w is the

parameter whose sensitivity is to be determined [7]. R_0 is given by

$$\begin{aligned} R_0 &= \frac{(1-\phi_1)(1-\phi_2)\beta_1\delta + (1-\phi_2)(1-\delta)\beta_1}{\alpha+\gamma_2+\mu} \\ &\quad + \frac{\alpha(1-\phi_1)(1-\phi_2)\beta_2\delta + (1-\phi_2)(1-\delta)\beta_2}{(\alpha+\gamma_2+\mu)(\gamma_3+\mu)}. \end{aligned} \tag{16}$$

$$\begin{aligned} \text{For}\beta_1, S_{\beta_1}^{R_0} &= \frac{\beta_1(\gamma_3+\mu)}{\beta_1(\gamma_3+\mu)+\alpha\beta_2}. \\ \text{For}\beta_2, S_{\beta_2}^{R_0} &= \frac{\alpha\beta_2}{\beta_1(\gamma_3+\mu)+\alpha\beta_2}. \\ \text{For } \alpha, S_{\alpha}^{R_0} &= \frac{[\beta_2(\alpha+\gamma_2+\mu)-(\beta_1(\gamma_3+\mu)+\alpha\beta_2)]\alpha}{(\alpha+\gamma_2+\mu)(\beta_1(\gamma_3+\mu)+\alpha\beta_2)}. \\ \text{For}\gamma_2, S_{\gamma_2}^{R_0} &= (\alpha\gamma_2+\gamma_2^2+\mu\gamma_2)\ln|\alpha+\gamma_2+\mu|. \\ \text{For}\gamma_3, S_{\gamma_3}^{R_0} &= \frac{-\alpha\beta_2\gamma_3}{(\beta_1(\gamma_3+\mu)^2+\alpha\beta_2(\gamma_3+\mu)}. \\ \text{For } \delta, S_{\delta}^{R_0} &= \frac{-\phi_1\delta}{1-\delta\phi_1} \\ \text{For } \mu, S_{\mu}^{R_0} &= \frac{[(\alpha+\gamma_2+\mu)(\gamma_3+\mu)\beta_1+((\gamma_3+\mu)\beta_1+\alpha\beta_2)(\alpha+\gamma_2+\gamma_3+2\mu)]\mu}{(\alpha+\gamma_2+\mu)(\gamma_3+\mu)((\beta_1(\gamma_3+\mu)+\alpha\beta_2))}. \end{aligned}$$

Based on the sensitivity indices in Table 3, the most sensitive parameter to the value of R_0 is β_1,

TABLE 3. Sensitivity Indices for the Model Parameters

Parameter	Description	Sensitivity Index
δ	Proportion of high risk sussceptibles	−0.36986
ϕ_1	PreP effectiveness	−0.041095
ϕ_2	Condom effectiveness	−0.11111
γ_3	ART Failure	−0.27182
β_1	Mean contact rate with I	0.72345
β_2	Mean contact rate with T_D	0.27654
α	Progression from I to T_D	−0.40225
γ_2	Progression from I to A	−0.05123
μ	Natural mortality rate	−0.02969

the mean contact rate with undiagnosed infectives. This implies that in order to control the spread of HIV in a high risk population, efforts should be geared towards reducing the number of those who are undiagnosed by testing them and enrolling them on ART treatment. This in turn lowers their infectivity as well as chances of progressing to the AIDS class.

5. Conclusion

In this study, a mathematical model is formulated, based on a system of ordinary differential equations, incorporating the impact of dual protection and ART adherence in preventing the spread of HIV among persons at high risk of infection.

Stability analysis of the model was done and depicted that when $R_0 < 1$, the disease free equilibrium is both locally and globally asymptotically stable. The Endemic Equilibrium of the mathematical model exists and was shown to be locally asymptotically stable whenever $R_0 > 1$, implying that there is persistence of HIV infection in the population provided that R_0 is greater than unity. Sensitivity analysis was conducted, depicting that the most sensitive parameter is β_1, the mean contact rate with the un-diagnosed infectives. Therefore, in order to control the spread of HIV among the high risk population, efforts ought to be channeled towards the undiagnosed population by frequently testing and enrolling them on ART treatment

which guarantees low viral load within the infected individual, making them less infective. Thus Dual protection and ART adherence are essential in the fight against the spread of HIV among the high risk population.

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