Original Research Article

A MATHEMATICAL DELAY MODEL FOR CONTROL OF THE SPREAD OF HIV/AIDS IN A HOMOGENEOUS POPULATION

Abstract

In this paper, we have considered a Delay Differential Equations model describing HIV/AIDS transmission incorporating time lags τ_1, and τ_2 and use of Prophylaxis . The formulated model has been analysed in which the Stationary points have been shown to be asymptotically Stable. Numerical Simulations have been carried out to determine the effects of time lags and Prophylaxis use. It has been shown that when the Reproduction number $R_\tau < 1$, HIV is controlled in the population and vice versa. Numerical simulations were carried out to determine the impact of time delay and Prophylaxis use on the control of HIV. It was observed that optimal use of Prophylaxis and time delay within 3 days resulted in $R_\tau < 1$, hence resulting in predominance in the uninfected (Susceptibles), coupled with the diminishing of the Exposed, Infected and AIDS individuals. The findings imply that prophylaxis use and time delay are key parameters in regulation of R_τ and therefore aids in the control of the spread of HIV/AIDS.

Keywords: Control; HIV/AIDS; Prophylaxis;Delay differential equations; Stability
2010 Mathematics Subject Classification: 53C25; 83C05; 57N16

1 Introduction

Human Immunodefiency Virus/Acquired Immunodefficiency Syndrome (HIV/AIDS) remains a significant public health challenge worldwide. From the onset of HIV/AIDS, there has been

an increase of mathematical models especially ODEs models describing the dynamics of the disease[1, 2, 3]. These models provide an understanding of the transmission dynamics of HIV/AIDS. A number of studies have been undertaken on delay models [4] and reference therein, but these studies did not incorporate post exposure prophylaxis use and it is effect on transmission of HIV transmission. The combination of these parameters and variable are key in studying the regulation of transmission of HIV/AIDS.
Numerous epidemiological models use (SIR) framework,for example Abueldahab *et al* [3]. Even though such models illustrate transmission dynamics of the disease, they may be inaccurate and limited to the extent that they presume individuals become contagious as soon as they enter the Infected compartment. As such, these models may be practically unsuitable in modelling diseases like HIV/AIDS where exposed people take some time to be contagious. There is a need to study both delays and effects of prophylaxis in the effort to control or eradicate HIV/AIDS in the society. Wasike *et al* [5] studied a SIR DDE model for the spread of HIV/AIDS. In their study, they considered a framework for the spread of HIV among people by mature voluntarily sex age group enrolment at a constant value. Their study included two delays, that is, time lag to become infective and the other to become full blown. In their findings, τ_1 is proportionate to the survival of the infected in the population i.e short τ_1 leads to quick elimination of the infected persons from the population and vice versa which increases the force of infection. Though the model is rich in transmission dynamics with two delays, it is limited in various ways. First, the model evidently lacks the exposure class and therefore does not account for the exposure period, making it less suitable for diseases like HIV/AIDS where such period is significant in the spread of the disease. By not including the exposed class, the SIR model may misvalue the rate of new infections, leading to inaccurate predictions. Secondly, the model only describes the dynamics of HIV/AIDs spread, but does not have parameters for disease control. We improved on the aforementioned model to have exposure class and prophylaxis use, and categorised the first delay to capture the omitted parameters in the preceding model. Therefore in our study, a SEIA compartmental model was used. The exposed class in the SEIA model gave an allowance for intervention against the transmission of the disease even if there was an interaction with the infective due to availability of prophylaxis treatment.The period when an individual is exposed but not yet infectious is significant for the spread or control of transmission of any disease [6]

2 Model Formulation

We formulate a framework in this section which categorizes the persons under study into the following compartments; Susceptible persons, Exposed persons, Infected persons, and AIDS persons. We start by stating the underlying assumptions and description of the model, then proceed to formulate the model which defines the changes in transmission of HIV/AIDS. We compute the reproduction number (R_τ) and equilibrium points, then finaliz by performing stability analysis of the stationary states in the interest of studying the long term behaviour of the solutions of the model.

2.1 Assumptions

We make the following assumptions in the model.

(i) The recruitment into the study population is sexually mature persons by birth who are recruited into susceptible class.

(ii) After a person gets infected they remain infected pending symptoms or signs of having AIDS or dies

(iii) An individual that has clinically tested and is infected will be categorized as being infective.

(iv) An individual who is exposed and uses prophylaxis within the required time will not be infected but becomes susceptible again at a rate of ϕ which ranges between $(0-1)$.

(v) Individuals who are exposed and do not use prophylaxis will transit into HIV positive at a rate of θ

(vi) The first time lag τ_1 is between $(0-3)$ days

(vii) The second time lag τ_2 is (10) years.

(viii) All who transit back to Susceptibles after exposure used Prophylaxis.

(ix) All who transit to infective did not use Prophylaxis.

(x) Prophylaxis implies PEP.

(xii) The natural death/mortality is the same from all the classes.

Table 1: Description and Defination of variables and parameters.

Variable	Description
$S(t)$	The vulnerable group of uninfected persons at a time t
$E(t)$	These are individuals who have interacted with the infected individuals and are clinically not infected at a time t.
$I(t)$	The equivalent group of HIV positive persons at a time t who are clinically tested and confirmed to be positive.
$A(t)$	The equivalent group of HIV positive people who are in AIDS phase, in this phase the patient's body's defense mechanism is totally damaged rendering it feeble to fight opportunistic infections symptoms at a time t.
Parameter	**Definition**
ϱ	The transmission probability.
Δ	Recruitment rate of susceptibles into a population.
κ	The natural deaths which are not AIDS related .
σ_c	The number of sexual partners in a given year.
τ_1	The delay between being exposed and getting infected.
τ_2	The delay between being HIV positive and having AIDS.
ϕ	The rate at which exposed individuals are transiting to the susceptible compartment.
θ	The degree of transiting to the Infected compartment by exposed persons.
α	The degree of transiting to the AIDS compartment by infected individuals.
δ	The degree of exiting population due to AIDS related reasons by AIDS individuals.

2.2 Description of the Model

Figure (1) is an illustrative diagram with four compartments. The first compartment represents susceptibles who get enrolled in this compartment at birth at rate Δ. This compartment can either reduce due to natural mortality, or after sexual intercourse with HIV positive individuals from the compartment of infectives causing them to transit to exposed class. The second compartment comprises of exposed individuals. These individuals enter this compartment from susceptible compartment at a rate $\varrho\sigma_c S\frac{I}{P}$ as a result of their interractions with the infectives. With the use of prophylaxis within the limits of time lag τ_1, the exposed individuals may not get infected. Such individuals transit back to the susceptible compartment at the rate ϕ. Otherwise, the exposed individuals will transit to the third compartment of infected people at the rate θ or exit the compartment through natural mortality . The infected persons exit their compartment through natural mortality or enter the AIDS fourth compartment after time lag τ_2 at rate α. People from AIDS compartment leave as a result of natural mortality or succumb to the disease at rate δ. At any given time, the total population that is sexually active is the sum of individuals in all compartments given by $P(t)$

Those dynamics described above can thus be represented mathematically by the diagram and equations below;

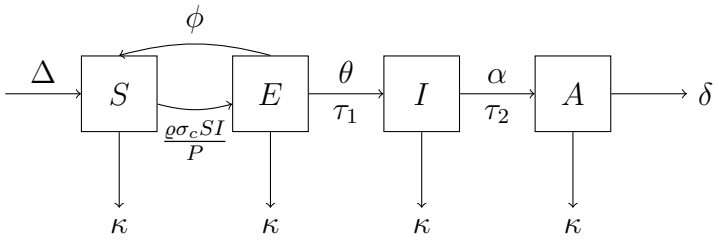

Figure 1: SEIA compartmental model

2.3 The Model's equations

From the illustrative diagram, the system equations below govern the transmission changes;

$$\begin{aligned}
\dot{S}(t) &= \Delta - \kappa S(t) + \phi E(t) - \frac{\varrho\sigma_c SI}{P(t)}, \\
\dot{E}(t) &= \frac{\varrho\sigma_c SI}{P(t)} - (\kappa + \phi + \theta)E, \\
\dot{I}(t) &= \theta E(t-\tau_1) - (\kappa + \alpha)I, \\
\dot{A}(t) &= \alpha I(t-\tau_2) - (\delta + \kappa)A(t),
\end{aligned} \tag{2.1}$$

In order analyse the system of equations above, we begin by introducing some notes that will be useful in the subsequent sections.

Let $\tau = max(\tau_1, \tau_2)$, then we define a Banach space $\mathcal{B}([-\tau, 0], \mathbb{R}^4_+)$ consisting of all continuous functions $g(s) = (S(s), E(s), I(s), A(s)), s \in [-\tau, 0]$ and map the interval $[-\tau, 0]$

into positive real Euclidean space $\mathbb{R}^4_+$. We endow this space with the norm $||g|| = \sup_{-\tau \leq s \leq 0} |g(s)|$, where $|g(s)|$ is the Euclidean norm in $\mathbb{R}^4_+$ which guarantees the continuity and boundedness of functions over the delay interval $[-\tau, 0]$. This space is key in taking care of the past characteristics of the system, as well as the present. System (2.1) is appended with initial conditions

$$S_0(t) \geq 0,\ E_0(t) \geq 0,\ I_0(t) \geq 0,\ A_0(t) \geq 0 \text{ for } t \geq 0. \tag{2.2}$$

2.4 Basic Properties of the model

In epidemic models, the total number of persons or inhabitants is naturally definite or finite; therefore solutions for model (2.1) must not go beyond an entire population size. Therefore the study of boundedness and positivity conditions of the solutions of the above model ensures that the population remains within genuine physical boundaries.

2.4.1 Positivity

Positivity is the property that ensures that solutions $S(t), E(t), I(t)$ and $A(t)$ stay positive $\forall\ t \geq 0$ if the initial functions in (2.2) are non-negative $\forall\ t \geq 0$.

Suppose that the initial conditions in (2.2) holds, then every solution of system (2.1) remains positive for all $t \geq 0$

Proof. We need to prove that $S(t), E(t), I(t)$ and $A(t)$ are all positive $\forall\ t \geq 0$.
From the first equation of system (2.1), it follows from comparison principle for ODEs that

$$\frac{dS}{dt} > -(\kappa + \frac{\varrho \sigma_c I}{P(t)})S(t),$$

and consequently, we have

$$S(t) > S_0(t) e^{-\int_0^t (\kappa + \frac{\varrho \sigma_c I}{P(t)}) d\ell} \geq 0 \tag{2.3}$$

Similarly, one can show that $E(t), I(t)$ and $A(t)$ are positive. Hence all solutions of system (2.1) remains positive $\forall\ t \geq 0$. □

We define the region Γ as

$$\Gamma = \{(S, E, I, A) \in R^2_+ \times \mathcal{B}([-\tau, 0], \mathbb{R}^2_+) : S + E + I + A \leq P\}$$

2.4.2 Boundedness

A function $g(t)$ is bounded if there exists a positive real number M such that $|g(t)| \leq M$, see [7]. All the solutions of system (2.1) with initial conditions (2.2) are bounded in the feasible region Γ.

Proof. Adding all the equations in system (2.1), we obtain

$$\dot{P}(t) = \Delta - \kappa P(t) - dA(t) + \theta(E(t-\tau_1) - E(t)) + \alpha(I(t-\tau_2) - I(t)), \tag{2.4}$$

For an increasing population, we must have that

$$\theta(E(t-\tau_1) - E(t)) > 0 \text{ and } \alpha(I(t-\tau_2) - I(t)) > 0 \tag{2.5}$$

Thus, equation (2.4), becomes

$$\dot{P}(t) \leq \Delta - \kappa P(t),$$

from which we obtain

$$P(t) \leq \frac{\Delta}{\kappa} + \left[P(0) - \frac{\Delta}{\kappa}\right] e^{-\kappa t}.$$

It follows that,

$$0 \leq \lim_{t\to\infty} \sup P(t) \leq \frac{\Delta}{\kappa}. \tag{2.6}$$

Therefore, $P(t)$ is bounded and all the possible solution sets of the system stay in the region Γ. Hence, the region $\Gamma = \{(S, E, I, A) \in R_+^2 \times \mathcal{B}([-\tau, 0], \mathbb{R}_+^2) : S + E + I + A \leq P \leq \frac{\Delta}{\kappa}\}$ is positively invariant. □

2.4.3 Existence of Equilibrium Points

Here, we investigate the existence of the equilibrium points of the model system (2.1).

Let $F^* = (S^*, E^*, I^*, A^*)$ be an equilibrium point of system (2.1), i.e.

$$\begin{aligned}
\Delta - \kappa S^*(t) + \phi E^*(t) - \varrho\sigma_c \frac{S^*(t)I^*(t)}{P^*(t)} &= 0,\\
\varrho\sigma_c \frac{S^*(t)I^*(t)}{P^*(t)} - (\kappa + \phi + \theta)E^*(t) &= 0,\\
\theta E^*(t-\tau_1) - (\kappa + \alpha)I^*(t) &= 0,\\
\alpha I^*(t-\tau_2) - (\delta + \kappa)A^*(t) &= 0,
\end{aligned} \tag{2.7}$$

from which we have

$$\begin{aligned}
S^* = \frac{(P^*\Delta)(\kappa + \phi + \theta)}{P^*\kappa(\kappa + \phi + \theta) + \varrho\sigma_c(\kappa + \theta)I^*} \quad , \quad E^* = \frac{\varrho\sigma_c\Delta I^*}{P^*\kappa(\kappa + \phi + \theta) + \varrho\sigma_c(\kappa + \theta)I^*}\\
A^* = \frac{\alpha e^{-\lambda\tau_2} I^*}{\delta + \kappa}
\end{aligned} \tag{2.8}$$

Using equation (2.8) in the third equation of system (2.7) with $P^* \approx \frac{\Delta}{\kappa}$, we obtain

$$\left[(\kappa + \theta)(\kappa + \alpha)\varrho\sigma_c I^* + \Delta(\kappa + \phi + \theta)(\kappa + \alpha)(1 - R_\tau)\right] I^* = 0 \tag{2.9}$$

where

$$R_\tau = \frac{\varrho\sigma_c\theta e^{-\lambda\tau_1}}{(\kappa + \phi + \theta)(\kappa + \alpha)} \tag{2.10}$$

is the reproduction number. The reproduction number can also be recovered using the method used in [9]. If there is no delay then (2.10) reduces to

$$R_0 = \frac{\varrho\sigma_c\theta}{(\kappa+\phi+\theta)(\kappa+\alpha)} \tag{2.11}$$

The following theorem gives the number of equilibrium points for system (2.1).

i. The system (2.1) always has a disease free equilibrium point $F_0 = \left(\frac{\Delta}{\kappa}, 0, 0, 0\right)$

ii. If $R_\tau > 1$, there exists a unique positive endemic equilibrium point $F^* = (S^*, E^*, I^*, A^*)$ where

$$\begin{aligned} S^* &= \frac{\Delta}{\kappa R_\tau} \quad , \quad E^* = \frac{\Delta(R_\tau - 1)}{(\kappa+\theta)R_\tau} \\ A^* &= \frac{\alpha\Delta(\kappa+\phi+\theta)(R_\tau-1)e^{-\lambda\tau_2}}{\varrho\sigma_c(\kappa+\theta)(\delta+\kappa)} \qquad I^* = \frac{\Delta(\kappa+\phi+\theta)(R_\tau-1)}{\varrho\sigma_c(\kappa+\theta)} \end{aligned} \tag{2.12}$$

Proof. i. $I^* = 0$ is always a root of equation (2.9). Thus, from (2.8), we have that the disease free equilibrium point of system (2.1) is $F_0 = \left(\frac{\Delta}{\kappa}, 0, 0, 0\right)$.

ii. From (2.9), we have that $I^* = \frac{\Delta(\kappa+\phi+\theta)(R_\tau-1)}{\varrho\sigma_c(\kappa+\theta)}$ is a root and I^* is positive if $R_\tau > 1$. Thus, a unique positive endemic equilibrium point F^* exists and is given by (2.12) □

2.5 Local Stability Analysis of Uninfected Steady State F_0

The stability analysis of this stationary point is performed through the Jacobian matrix of the system (2.1).

If $R_\tau \leq 1$, the uninfected steady state F_0 of system (2.1) is locally asymptotically stable for every time delay $\tau_1 \geq 0$.

Proof. To determine the stability of the uninfected equilibrium point, we need to compute the Jacobian matrix (J) of the system (2.1) at the point F_0. The Jacobian matrix of the system is given by

$$J_{F_0} = \begin{pmatrix} -\kappa & \phi & -\varrho\sigma_c & 0 \\ 0 & -(\phi+\kappa+\theta) & \varrho\sigma_c & 0 \\ 0 & \theta e^{-\lambda\tau_1} & -(\kappa+\alpha) & 0 \\ 0 & 0 & \alpha e^{-\lambda\tau_2} & -(\delta+\kappa) \end{pmatrix} \tag{2.13}$$

The characteristic equation $Q(\lambda) = det(J_{F_0} - \lambda I) = 0$ is given by

$$Q(\lambda,\tau_1) = (\lambda+\kappa)(\delta+\kappa+\lambda)[\lambda^2+(2\kappa+\phi+\theta+\alpha)\lambda+(\kappa+\alpha)(\kappa+\phi+\theta)-\varrho\sigma_c\theta e^{-\lambda\tau_1}] = 0 \tag{2.14}$$

In the absence of delay, all the roots of equation (2.14) have negative real parts provided $R_0 < 1$. When $\tau_1 \neq 0$, then two of the roots of equation (2.14) are given by $\lambda = -\kappa$ and $\lambda = -(\kappa + \delta)$. The other roots can be obtained from

$$\lambda^2 + (2\kappa + \phi + \theta + \alpha)\lambda + (\kappa + \alpha)(\kappa + \phi + \theta) - \varrho\sigma_c\theta e^{-\lambda\tau_1} = 0$$

This equation can be written in the form

$$G(\lambda) + H(\lambda, \tau_1)e^{-\lambda\tau_1} = 0 \tag{2.15}$$

where $G(\lambda) = \lambda^2 + (2\kappa + \phi + \theta + \alpha)\lambda + (\kappa + \alpha)(\kappa + \phi + \theta)$ and $H(\lambda, \tau_1) = -\varrho\sigma_c\theta$. The roots of equation (2.15) will lie to the left of the complex plane provided all zeros of $G(\lambda)$ have negative real parts and $|G(0)| \geq |H(0)|$ for all $\tau_1 > 0$, see [[5], Lemma 3.2]. All the coefficients of $G(\lambda)$ are positive hence all its roots have negative real parts. For the second condition, we see that $R_0 \leq 1$, where R_0 is as defined in (2.11). Therefore, all the roots of equation (2.14) lie to the left of the complex plane provided $R_0 \leq 1$.

□

2.6 Global stability of the uninfected Equilibrium

The uninfected state is globally attracting when $R_\tau < 1$. We Use the method used in [8] to show that the disease free equilibrium point F_0 is globally stable.

The uninfected equilibrium $F_0 = (\frac{\Delta}{\kappa}, 0, 0, 0)$ of (2.1) is a globally attracting if $R_\tau < 1$ and assumptions (L_l) and (L_2) below fulfilled.

1 (L_l): For $\frac{dS}{dt} = f_1(S, 0);\ S_*$ is globally attracting.

2 (L_2): $y(S, I) = MI - \widehat{y}(S, I),\ \widehat{y}(S, I) \geq 0$ for $S, I \in \Gamma$

Proof. $S \in R^1$ and $y(S, I) \in R^3$ denotes the elements of uninfected and infected(inclusive of Exposed, Infected and AIDS) individuals respectively
$S = S(t),\ y(S, I) = (E(t), I(t), A(t))$

$$\begin{aligned} f_1(S, I) &= \Delta - \kappa S + \phi E(t) - \varrho\sigma_c S(t)\frac{I(t)}{P(t)} \\ f_1(S, 0) &= \Delta - \kappa S = 0, \\ \lim_{t\to\infty} S_*(t) &= \frac{\Delta}{\kappa} \end{aligned} \tag{2.16}$$

Clearly, $S_* = (\frac{\Delta}{\kappa})$ is globally asymptotically stable equilibrium of $f_1(S, 0)$, which satisfies the first condition (L_1)

$$y(S, I) = \left\{ \begin{array}{l} f_2 = \varrho\sigma_c\frac{S(t)I(t)}{P(t)} - (\kappa + \phi + \theta)E(t), \\ f_3 = \theta E(t - \tau_1) - (\alpha + \kappa)I(t), \\ f_4 = \alpha I(t - \tau_2) - (\delta + \kappa)A(t), \end{array} \right\}$$

From (L_2) : $y(S, I) = MI - \widehat{y}(S, I)$; where $M = \partial_y(S_0, 0)$ is the matrix of infected subsystem $y(S, I)$ after linearization around the uninfected equilibrium

$$MI = \left\{ \begin{array}{c} \varrho\sigma_c I - (\kappa+\theta+\phi)E \\ \theta E e^{-(\lambda\tau_1)} - (\kappa+\alpha)I \\ \alpha I e^{-(\lambda\tau_2)} - (\delta+\kappa)A \end{array} \right\};$$

But;

$$y(SI) = MI - \widehat{y}(S,I), \tag{2.17}$$

Rearranging equation (2.17) we get;

$$\widehat{y}(S,I) = \left\{ \begin{array}{c} \varrho\sigma_c I(t) - (\kappa+\theta+\phi)E(t) \\ \theta E(t)e^{-(\lambda\tau_1)} - (\kappa+\alpha)I(t) \\ \alpha I(t)e^{-(\lambda\tau_2)} - (\delta+\kappa)A(t) \end{array} \right\} - \left\{ \begin{array}{c} \varrho\sigma_c \frac{S(t)I(t)}{P(t)} - (\kappa+\phi+\theta)E(t) \\ \theta E(t)e^{-(\lambda\tau_1)} - (\alpha+\kappa)I(t) \\ \alpha E(t)e^{-(\lambda\tau_2)} - (\delta+\kappa)A(t) \end{array} \right\},$$

$$= \begin{pmatrix} \varrho\sigma_c I(1-S/P) \\ 0 \\ 0 \end{pmatrix}.$$

Therefore, $\widehat{y}(S,I) \geq 0$ since $0 \leq S \leq P$, which satisfies the second condition (L_2). Because both assumptions are satisfied, the uninfected equilibrium F_0 is globally asymptotically stable provided $R_\tau < 1$. □

2.7 Local Stability analysis of Infected Equilibrium

For $\tau_1 > 0$, the Infected steady state F^* of system (2.1) is locally asymptotically stable if $R_\tau^2 > R_\tau$. In the absence of delay, the point F^* is locally asymptotically stable if $R_\tau \geq R_0$.

Proof. We linearize the system at the point F^* to get

$$J_{F^*} = \begin{bmatrix} -(\kappa + \frac{\kappa\varrho\sigma_c I^*}{\Delta}) & \phi & \frac{-\kappa\varrho\sigma_c S^*}{\Delta} & 0 \\ \frac{\kappa\varrho\sigma_c I^*}{\Delta} & -(\phi+\kappa+\theta) & \frac{\kappa\varrho\sigma_c S^*}{\Delta} & 0 \\ 0 & \theta e^{-\lambda\tau_1} & -(\kappa+\alpha) & 0 \\ 0 & 0 & \alpha e^{-\lambda\tau_2} & -(\delta+\kappa) \end{bmatrix} \tag{2.18}$$

The Characteristic equation becomes

$$\begin{aligned} R(\lambda,\tau_1) \quad = \quad & \{\lambda+\delta+\kappa\}\left\{\lambda^3 + [3\kappa+\alpha+\theta+\phi+\kappa\varrho\sigma_c\Delta^{-1}I^*]\lambda^2 + \right. \\ & [(2\kappa+\alpha+\theta)\kappa\varrho\sigma_c\Delta^{-1}I^* + \kappa(2\kappa+\alpha+\theta+\phi) + (\kappa+\theta+\phi)(\kappa+\alpha)]\lambda \\ & +(\kappa+\alpha)(\kappa+\theta)\kappa\varrho\sigma_c\Delta^{-1}I^* + \kappa(\kappa+\theta+\phi)(\kappa+\alpha) - \\ & \left.[\theta\kappa\varrho\sigma_c\Delta^{-1}S^*\lambda + \theta\kappa^2\varrho\sigma_c\Delta^{-1}S^*]e^{-\lambda\tau_1}\right\} = 0 \end{aligned} \tag{2.19}$$

One of the roots of (2.19) is given by $\lambda = -(\delta+\kappa)$. The other roots are obtained from

$$D(\lambda) + E(\lambda)e^{-\lambda\tau_1} = 0 \tag{2.20}$$

where $D(\lambda) = \lambda^3 + [3\kappa + \alpha + \theta + \phi + \kappa\varrho\sigma_c\Delta^{-1}I^*]\lambda^2 + [(2\kappa + \alpha + \theta)\kappa\varrho\sigma_c\Delta^{-1}I^* + \kappa(2\kappa + \alpha + \theta + \phi) + (\kappa + \theta + \phi)(\kappa + \alpha)]\lambda + (\kappa + \alpha)(\kappa + \theta)\kappa\varrho\sigma_c\Delta^{-1}I^* + \kappa(\kappa + \theta + \phi)(\kappa + \alpha)$ and $E(\lambda) = -[\theta\kappa\varrho\sigma_c\Delta^{-1}S^*\lambda + \theta\kappa^2\varrho\sigma_c\Delta^{-1}S^*]e^{-\lambda\tau_1}$. In the absence of delay, we see that all the roots of equation (2.19) have negative real parts provided $R_\tau \geq R_0$. When $\tau_1 \neq 0$, the roots of equation (2.20) will lie to the left of the complex plane provided all zeros of $D(\lambda)$ have negative real parts and $|D(0)| \geq |E(0)|$ for all $\tau_1 > 0$. All the coefficients of $D(\lambda)$ are positive and hence all its roots have negative real parts. The second stability condition implies that $R_\tau^2 \geq R_0$. Therefore, for every $\tau_1 > 0$, the characteristic equation (2.19) has all its roots having negative real parts provided $R_\tau^2 \geq R_0$.

□

3 Numerical Simulations

In this chapter, we present findings of the python simulations and discussion of the results. To visualise the dynamical behaviour of the model (2.1), it was assimilated mathematically by way of Python. Parameters and variables were estimated based on available epidemiological data and reasonable assumptions about disease progression in Kenya as given in the table below;

Table 2: Table of data

Parameter/Variable	Initial Data	Source
ϕ, rate of moving back to exposed class	between 0-1	Estimate
θ, rate of progression to infected class	0.125	Estimate
α, rate of progression to AIDS class	0.125	Estimate
κ, mortality rate	1/66	Estimate
δ, AIDS related death	0.36 per year	Estimate
σ_c, contact rate	3 per year	[5]
ϱ, Probability of getting infected	0.44	[5]
Δ Recruitment rate into the Population	1000000	Estimate
S(t) Susceptibles	4000000	Estimate
E(t)Exposed	2000000	Estimate
I(t)Infected	1300000	[10]
A(t)AIDS	50000	Estimate
τ_1, incubation delay	[0,3) days	[10]
τ_2, second delay	10 years	[10]
t-time	100 years	Estimate

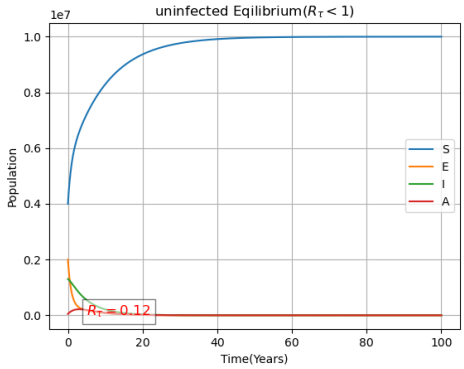

Figure 2: graph at uninfected equilibrium point

3.1 (i)Uninfected Steady State (F_0)

When $R_\tau < 1$, HIV/AIDS is controlled in the population. i.e susceptibles individuals are predominant and converge at F_0 while the Exposed, infective and AIDS individuals will converge to zero or are eliminated from the population as it is shown in figure 2. This is in agreement with qualitative analysis in literature for instance, see [5, 11]. If $R_\tau > 1$ we will have infected equilibrium point.

3.2 (ii)Infected steady State (F^*)

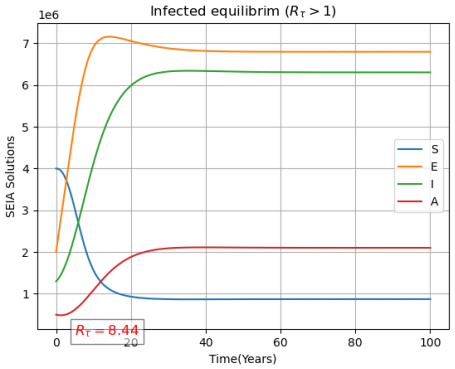

Figure 3: graph at infected equilibrium point

When the $R_\tau = 8.44$, as exhibited in figure 3, the SEIA system is locally stable at Infected steady state. If there is no use of prophylaxis, we have the disease being persistent in the population ,the exposed being predominant and coexisting closely with infected and the full

blown cases. The susceptibles therefore drops below the AIDS individuals. This findings are inconsistent with the findings of Wasike *et al* [5]. For them, a short τ_1 leads to infectives dying out faster from a population except for prophylaxis unlike in our case where infectives become predominant and suceptibles diminish. However, the findings are consistent with the ones in [12, 13].

3.3 (iii) Variation of τ_1 and ϕ

In this section we show results and findings when τ_1 is varied between $0 - 3$ days and ϕ varied between $0 - 1$;

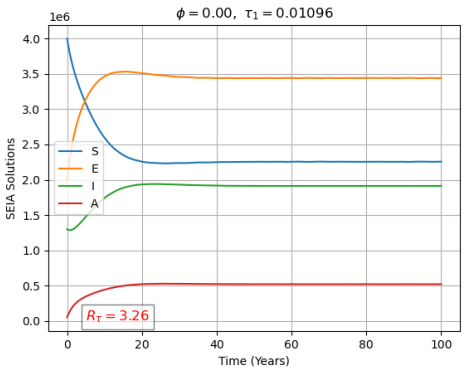

Figure 4: graph when $\phi = 0$ and $\tau_1 = 0.01096$

When there are no mitigation measures taken into consideration, there will be high exposure leading to exposed individuals being predominant in the population and the susceptible individuals diminishing as they become highly exposed as shown in figure 4. Again, if there are no interventions, they will be converted to infectives and eventually full blown AIDS. This will lead to the whole population being wiped out. It is worth noting that this is an ideal situation. Even with the coming up of pre-exposure prophylaxis, if sensitization and awareness of both PEP and PrEP are not carried out, the population can be wiped out. This findings are partly in agreement with the findings in [5] for prolonged τ_1, where we have susceptibles diminish while the infectives and AIDS individuals increase.

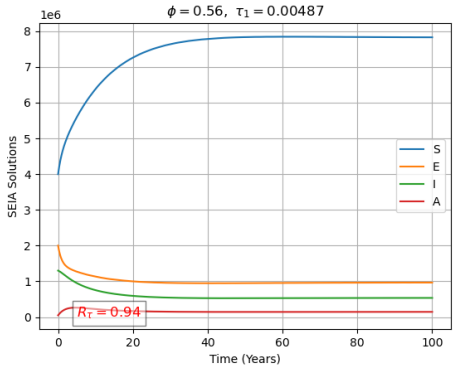

Figure 5: graph for $\phi = 0.56$ and $\tau_1 = 0.00487$

As seen in figure 5, if the delay τ_1 is reduced and there is use of Prophylaxis, there is a resultant significant rise of susceptibles accompanied by a drop of those infected, exposed , and AIDS Persons. Even though we still have exposed, infected and AIDS coexisting together in the population, their numbers have significantly dropped as a result of use of prophylaxis with minimal delay in use

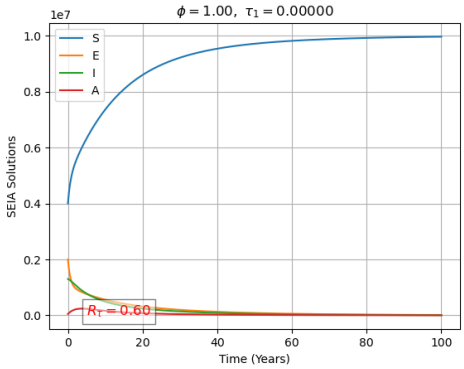

Figure 6: graph for $\phi = 1$ and $\tau_1 = 0$

When prophylaxis use rate is at 1 and the delay τ_1 is at zero i.e no delay in using prophylaxis, the susceptible individuals will be predominant while the exposed, infectives and AIDS individuals will reduce significantly converging to zero. In this case we have rate of conversion back to susceptibles being optimum. From figure 6 it is evident after about 40 years we will have HIV/AIDS controlled in a population as shown in figure 6. The graph is almost similar to the graph in 2 at uninfected equilibrium point. This implies that if prophylaxis is used with no delay, HIV/AIDS can be controlled in a population, and this can be achieved through effective awareness and sensitization of the key parameters.

4 Conclusion

In this study, we developed and analyzed a non linear mathematical model for the spread of HIV/AIDS. We determined basic reproduction number (R_τ), uninfected and infected equilibrium. The qualitative findings were in agreement with numerical simulations i.e when $R_\tau < 1$ the disease was controlled in the population and vice versa. We also established that ϕ and τ_1 were key parameters in regulation of the value of R_τ. To maintain $R_\tau < 1$, ϕ needs to be at 1 and $\tau_1 \leq 3$ days. Our findings highlight the importance of prophylaxis use and reduction of initial delay after exposure before starting the use of the prophylaxis. We assumed that the use of prophylaxis was between 0 and 1. The assumption made the model uncomplicated and the primary dynamics demonstrated. However, these assumptions might restrict the model's applicability to situations in reality. Future work will focus on extending the model to include more realistic aspects such as real data on prophylaxis use.

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