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Mathematical Analysis of Injectable PrEP and Vaginal Rings as HIV Prevention Strategies Among Female Sex Workers in Kenya

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Abstract

This paper formulated a deterministic mathematical model to study the use of injectable PrEP and vaginal rings as HIV prevention strategies among the Female Sex Workers (FSWs) in Kenya. The reproduction number, the disease-free equilibrium and endemic equilibrium points determined. The Next Generation Matrix (NGM) method was used to determine the Reproduction number. The Jacobian matrix method was used to analyze the local stability of the Disease Free Equilibrium which was locally stable and globally asymptotically stable when $R_0 < 1$. The analysis of the model found that low use of Vaginal rings and Injectable PrEP while engaging in sex resulted in contracting the HIV virus and consistent use of injectable PrEP and the vaginal rings reduced the risk of contracting of HIV virus and consequently reduced the AIDS population. This work recommends the use of both vaginal rings and Injectable PrEP as HIV/AIDS prevention strategies among the high risk populations such as female sex workers (FSWs). Therefore the Ministry of Health in Kenya (MoH-K) should make both Vaginal rings and Injectable PrEP available and affordable since it ranges from 1 to 8 US Dollars which is relatively expensive to the Female sex workers who rely on sex work as a source of livelihood. They should also educate these FSWs on the importance and proper use of using these preventive measures as a way of protecting themselves from HIV/AIDS.

Keywords: Injectable PrEp, Female Sex Workers(FSWs), Vaginal rings, Next generation matrix, Jacobian matrix.

1 Introduction

HIV/AIDS has been a life threatening disease globally and in Kenya since 1983. Female Sex Workers (FSWs) are categorized as vulnerable population and are highly susceptible to sexually transmitted infections (STIs). Moreover they are approximately 30 more times likely to be living with HIV than other women in the same reproductive age. In 2019, the HIV/AIDS prevalence was approximately 36% among the sex workers [21].
Due to vulnerability of the key populations, including; men who have sex with men, transgender and gender - diverse people, sex workers, people who inject drugs and people in prisons and other closed settings to HIV [21], WHO formulated new guidelines on HIV in 2022. The global median HIV prevalence among FSWs is 3.9% [8, 24] and in sub-Saharan Africa, the FSWs are at much higher risk of HIV with an estimate of about one in five sex workers expected to be living with HIV. The World Bank report on the global HIV epidemics among sex workersin 2013 indicated that the prevalence among sex workers varies globally reaches its highest at 36.9% in sub-Saharan Africa [6].

FSWs in Africa face many challenges like human right abuse and different forms of violence. This includes violence perpetrated by the police, their clients and by members of the wider community [5]. They also face sexual violence which includes rape, harassment, emotional abuse, humiliation, public insults, stigma and discrimination, as well as other violations such as refusal of clients to adhere to the agreed transaction fee or outright refusal to pay sex workers for services provided. Due to mobility and nature of their work, adherence to Anti-Retroviral Treatment (ARV) is very low among HIV-positive sex workers and often miss taking their anti-retrovirals (ARV) if they have been arrested and put in jail [14].
In Democratic Republic of Congo (DRC), the Congolese legislation criminalises female sex work, consider it to be anti-social and perceive it to be immoral by the general community according to [26].

Ethiopians consider sex work as an act of deviant behaviour and immoral. It is permitted on the basis that it would be impractical to abolish it instantly. This Ethiopian law has created an environment for sex workers to work freely and consequently also created unsafe working environments for sex workers because sex work is not legal or illegal in Ethiopia therefore difficult for sex workers rights to be respected [16]. In Ethiopia, the AIDS related services are accessed for free from governmental clinics and limited access from private clinics due to stigma and discrimination of the sex workers.

Malawi has an estimated sex worker population of about 20000. Due to stigma associated with sex work, majority of these sex workers remain hidden. The female sex workers have an HIV prevalence of about 73% associated with low condom use and lack of accessibility and availability of AIDS related services [9].
In Nigeria according to [23] considers sex work as illegal and the offender can be either a man or woman aiding and abetting prostitution. It recommends two year imprisonment for partially or wholly participating in prostitution.

Uganda has experienced increasing HIV incidences for approximately the last 30 years and the only country in East Africa with increasing HIV prevalence in the last five years. Also the sex workers are a at risk population in Uganda with HIV prevalence of 33% with HIV prevalence higher in women than their male counterparts according to [7]. Criminalisation of sex work in Uganda is likely to increase HIV risk among the sex workers

due to stigma, discrimination and police abuse and exploitation of the sex workers.

In Kenya, HIV prevalence among Female Sex Workers (FSWs) in 2021 was estimated as 29.3% compared to 4.3% in the general female population according to [19]. Also sex workers are frequently regarded as easy targets for harassment and violence, and are considered immoral and deserving of punishment according to traditional cultures and the Kenyan constitution. Since then, several prevention measures have been recommended which includes use of both male and female condoms, lubricants and pre-exposure prophylaxis (PrEP). PrEP prevents HIV from getting into the body and replicating itself which only happens when there are high levels of PrEP in the body hence must be used correctly.

In 2018, the National AIDS and Sexually Transmitted Infections formulated national guidelines to include female sex workers in the HIV prevention response in Kenya according to [2]. They found that there was increased uptake of treatment and prevention services and also increase in harassment and discrimination among the sex workers requiring further attention. This implies that interventions can inform replication and scale-up of such interventions in Kenya. The interventions also expected increased use of condoms and Pre-Exposure prophylaxis (PrEP).

Pre-exposure prophylaxis is a relatively new intervention being offered to FSWs and other populations with high risk of HIV infection according to [3]. Also [22] found that Pre and Post-Exposure prophylaxis (PrEP and PEP) can reduce the risk of HIV acquistion but is under utilized by the vulnerable populations which includes sex workers. Also knowledge and awareness about them was low but high willingness to use them. Their findings underlined the importance of locating PrEP and PEP within sex worker-friendly health services and conducting outreach promotion of these biomedical HIV prevention methods for Kenyan sex workers. [20], found that there was need to awareness and understanding of PrEP as an HIV prevention tool in combination with other safer sex methods.

The dapivirine vaginal ring is made from flexible silicon that is easy to bend and insert which works in a similar manner to the contraceptive ring and used by women at high risk of HIV infection who include adolescent girls and adolescent girls, women who sell sex, or women whose partners have HIV and not on treatment according to [25]. The ring is inserted in the vagina and slowly releases the antiretroviral drug dapivirine to prevent HIV and usually replaced after 28 days. the World Health Organization (WHO) in January 2021, conditionally approved and recommended the vaginal ring to be offered to women at high risk of HIV infection. WHO recommended the rings to be offered in combination of other HIV prevention measures like condoms. Since then Kenya, South Africa, Uganda, Zambia and Zimbabwe have approved the dapivirine vaginal ring.
This work analyses how the use of injectable PrEP and vaginal rings combined will affect the HIV infection dynamics among FSWs in Kenya. The deterministic model was developed based on the following assumptions:

- The rate of infection is the same for all the exposed classes.
- The exposed classes (E_1) and (E_2) are not infectious.

- The exposed classes have same rate of becoming infectious
- Susceptible population is exposed through heterosexual contact with infected males.

2 Methodology

2.1 Model formulation

In this section, a deterministic mathematical model is formulated to study the effects of the use of injectable PrEP and vaginal rings preventive measures on the HIV AIDS dynamics among the FSWs in Kenya. Figure 1 shows the diagrammatic representation of the deterministic model, where the population was divided into five compartments which includes; Susceptible population (S), two exposed populations (E_1) and (E_2), where (E_1) is the population that becomes infected with HIV while using vaginal rings, (E_2) becomes infected with HIV while using injectable PrEP, the HIV infected population (I) and the AIDS population (A).

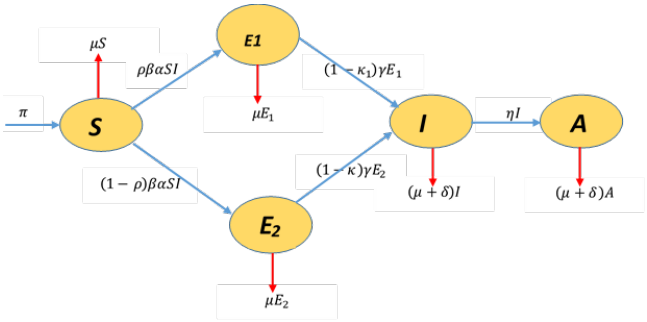

Figure 1: Model Flow Chart

The state variables and parameters used in the model are described in tables 1 and 2 respectively.

State variable	Description	Initial value	Source
S	Susceptible population	129,271	[18]
E_1	Population Exposed while using Vaginal rings	50,000	Estimated
E_2	Population Eeposed while using injectable PrEP	120,000	Estimated
I	HIV infected population	85,000	Estimated
A	AIDS infected population	70,000	Estimated

Table 1: Description of state variables and their possible values

Parameter	Description	value	Source
π	Recruitment rate	0.8500	[12]
α	Modifying factor for transmission rate β	0.5	Assumed
ρ	Proportion of the Susceptible population which becomes exposed without using either preventive measure	0.723	Assummed
β	HIV infection rate from infected males	0.3425	[15]
κ	Rate of using injectable PrEP	0.56	[17]
κ_1	Rate of using vaginal rings	0.82	Assumed
γ	Rate at which the exposed population becomes HIV infected	0.293	[1]
η	Rate at which the infected population develops full AIDS	0.66	[10]
μ	Natural death rate	0.0539	[11]
δ	HIV/AIDS induced death rate	0.016	[12]

Table 2: Description of model parameters and their estimated values

where $0 < \pi, \alpha, \rho, \beta, \kappa, \kappa_1, \gamma, \eta, \mu, \delta < 1$.

2.2 Qualitative Analysis

2.2.1 The Invariant region

With reference to [13], to investigate the boundedness of the model then the total population is given by; $N = S + E_1 + E_2 + I + A$ and the sum of the time derivatives is given by;

$$\left.\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE_1}{dt} + \frac{dE_2}{dt} + \frac{dI}{dt} + \frac{dA}{dt} \\ \frac{dN}{dt} &= \pi - \mu(S + E_1 + E_2 + I + A) - \delta(I + A) \end{aligned}\right\} \tag{1}$$

When $\delta = 0$ it implies that there is no disease mortality rate and hence the sum of the time derivative of the total population $\frac{dN}{dt}$ becomes;

$$\pi - \mu N - \delta(I + A) \leq \pi - \mu N \tag{2}$$

Therefore it implies that;

$$\frac{dN}{dt} \leq \pi - \mu N \tag{3}$$

Integrating both sides of equation (3) with respect to t and taking limits as $t \to \infty$

we obtain the limit of N(t) as;

$$\lim_{t \to \infty} N(t) \leq \frac{\pi}{\mu} \tag{4}$$

Equation (4) implies that the biologically feasible region for our system is;

$$\Lambda = \{S, E_1, E_2, I, A \in \mathbb{R}^5 | 0 < N(t) \leq \frac{\pi}{\mu}\} \tag{5}$$

And the model is therefore bounded and positively invariant hence well posed.

2.2.2 Governing Equations

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \rho\beta\alpha SI - (1-\rho)\beta\alpha SI - \mu S \\
\frac{dE_1}{dt} &= \rho\beta\alpha SI - (1-\kappa_1)\gamma E_1 - \mu E_1 \\
\frac{dE_2}{dt} &= (1-\rho)\beta\alpha SI - (1-\kappa)\gamma E_2 - \mu E_2 \\
\frac{dI}{dt} &= (1-\kappa_1)\gamma E_1 + (1-\kappa)\gamma E_2 - (\mu+\eta+\delta)I \\
\frac{dA}{dt} &= \eta I - (\mu+\delta)A
\end{aligned}\right\} \tag{6}$$

2.2.3 Equilibrium Points

The equilibrium points represent both Disease Free Equilibrium point (DFE) and the Endemic Equilibrium point (EEP). The DFE represent the state at which there is no disease in the population. It is obtained by equating the governing equations to zero and also E_1, E_2, I and A equal to zero.

$$\pi - \rho\beta\alpha SI - (1-\rho)\beta\alpha SI - \mu S = 0$$

Solving this gives; S $= \pi\frac{}{\mu}$

Thus the, DFE = (S^0,$E_1{}^0$,$E_2{}^0$,I^0,A^0) = $(\frac{\pi}{\mu}, 0, 0, 0, 0)$

Endemic Equilibrium Point is the point at which the disease persists and continues to spread within the population. It is obtained by the system;

$$\left.\begin{aligned}
\pi - \rho\beta\alpha S^*I^* - (1-\rho)\beta\alpha S^*I^* - \mu S^* &= 0 \\
\rho\beta\alpha S^*I^* - (1-\kappa_1)\gamma E_1^* - \mu E_1^* &= 0 \\
(1-\rho)\beta\alpha S^*I^* - (1-\kappa)\gamma E_2^* - \mu E_2^* &= 0 \\
(1-\kappa_1)\gamma E_1^* + (1-\kappa)\gamma E_2^* - (\mu+\eta+\delta)I^* &= 0 \\
\eta I^* - (\mu+\delta)A^* &= 0
\end{aligned}\right\} \tag{7}$$

The EEP is therefore given by $(S^*, E_1^*, E_2^*, I^*, A^*)$

It is obtained by solving equations system (7) simulatnoueosly to get;

$$\left.\begin{aligned} S^* &= \frac{\pi}{\beta\alpha I^* + \mu} \\ E_1^* &= \frac{\rho\beta\pi\alpha I^*}{(\beta\alpha I^* + \mu)((1-\kappa_1)\gamma + \mu)} \\ E_2^* &= \frac{(1-\rho)\beta\pi\alpha I^*}{((\beta\alpha I^* + \mu)((1-\kappa)\gamma + \mu)} \\ I^* &= I^* \\ A^* &= \frac{\eta I^*}{\mu + \delta} \end{aligned}\right\} \tag{8}$$

System (8) gives the EEP implicitly in terms of I^*.

2.2.4 Reproduction Number

Reproduction number refers to the number of new infection that an infected individual can cause when put in susceptible population in his entire infectious period [12]. It is determined using the next generation matrix method where matrices F and V are obtained from the system of governing differential equations. Matrix F represents the rate of appearance of new infections while matrix V represents negated exits from the infected classes.

The infected classes in our system include E_1,E_2, I and A. The classes E_1 and E_2 are not infectious therefore I is the sole infectious class;

$$F = \begin{bmatrix} 0 & 0 & \rho\beta\alpha S & 0 \\ 0 & 0 & (1-\rho)\beta\alpha S & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{9}$$

and

$$V = \begin{bmatrix} (1-\kappa_1)\gamma + \mu & 0 & 0 & 0 \\ 0 & (1-\kappa)\gamma + \mu & 0 & 0 \\ (1-\kappa_1)\gamma & (1-\kappa)\gamma & \mu + \eta + \delta & 0 \\ 0 & 0 & -\eta & \mu + \delta \end{bmatrix} \tag{10}$$

The inverse of equation (10) is given by;

$$V^{-1} = \begin{bmatrix} \frac{-1}{\gamma\kappa_1 - \gamma - \mu} & 0 & 0 & 0 \\ 0 & \frac{-1}{\gamma\kappa - \gamma - \mu} & 0 & 0 \\ \frac{(-1+\kappa_1)\gamma}{(\gamma\kappa_1 - \gamma - \mu)(\mu+\eta+\delta)} & \frac{(-1+\kappa)\gamma}{(\gamma\kappa - \gamma - \mu)(\mu+\eta+\delta)} & \frac{1}{\mu+\eta+\delta} & 0 \\ \frac{(-1+\kappa_1)\eta\gamma}{(\gamma\kappa_1 - \gamma - \mu)(\mu+\eta+\delta)(\mu+\delta)} & \frac{(-1+\kappa)\eta\gamma}{(\gamma\kappa - \gamma - \mu)(\mu+\eta+\delta)(\mu+\delta)} & \frac{\eta}{(\mu+\delta)(\eta+\mu+\delta)} & \frac{1}{\mu+\delta} \end{bmatrix} \tag{11}$$

Using equations (9) and (11) we obtain;

$$FV^{-1} = \begin{bmatrix} \frac{(-1-\kappa_1)\rho\alpha\beta\pi\gamma}{\mu(\gamma\kappa_1-\gamma-\mu)(\mu+\eta+\delta} & \frac{(-1-\kappa)\rho\alpha\beta\pi\gamma}{\mu(\gamma\kappa-\gamma-\mu)(\mu+\eta+\delta} & \frac{\rho\alpha\beta\pi}{\mu(\mu+\eta+\delta)} & 0 \\ \frac{(1-\kappa_1)(1-\rho)\beta\alpha\pi\gamma}{\mu(\gamma\kappa_1-\gamma-\mu)(\mu+\eta+\delta} & \frac{(-1-\kappa)(1-\rho)\beta\alpha\pi\gamma}{\mu(\gamma\kappa-\gamma-\mu)(\mu+\eta+\delta} & \frac{(1-\rho)\beta\alpha\pi}{\mu(\mu+\eta+\delta} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{12}$$

The eigenvalues of equation (12) are;

$$\lambda_1 = \lambda_2 = \lambda_3 = 0, \lambda_4 = \frac{\beta\alpha\gamma\pi[\rho(-1+\kappa_1)(\gamma\kappa-\gamma-\mu)+(-1+\kappa)(1-\rho)(\gamma\kappa_1-\gamma-\mu)]}{\mu(\mu+\eta+\delta)(\gamma\kappa_1-\gamma-\mu)(\gamma\kappa-\gamma-\mu)} \tag{13}$$

Hence $R_0 = Max(\lambda_1, \lambda_2, \lambda_3, \lambda_4) = \frac{\beta\alpha\gamma\pi[\rho(-1+\kappa_1)(\gamma\kappa-\gamma-\mu)+(-1+\kappa)(1-\rho)(\gamma\kappa_1-\gamma-\mu)]}{\mu(\mu+\eta+\delta)(\gamma\kappa_1-\gamma-\mu)(\gamma\kappa-\gamma-\mu)}$

Therefore the expression for R_0 simplifies to;

$$R_0 = \frac{\beta\alpha\pi}{\mu(\mu+\eta+\delta)}\left[\frac{\rho(1-\kappa_1)\gamma}{(1-\kappa_1)\gamma+\mu} + \frac{(1-\rho)(1-\kappa)\gamma}{(1-\kappa)\gamma+\mu}\right] \tag{14}$$

Class A doesn't appear in equation (14) above because individuals in this class do not contribute to new infections. The average time duration of being infectious of an individual is given by the term $\frac{1}{\mu+\eta+\delta}$ and $\frac{\beta\alpha\pi}{\mu}$ represents the transmission potential of single infected individual when placed in an entire susceptible population.

The term $\frac{\rho(1-\kappa_1)\gamma}{(1-\kappa_1)\gamma+\mu} + \frac{(1-\rho)(1-\kappa)\gamma}{(1-\kappa)\gamma+\mu}$ is the probability that new exposed individuals enters class I.

2.3 Local Stability Analysis of DFE

The local stability analysis of the DFE is computed using the Jacobian matrix method and the Routh - Hurwitz criterion. The Jacobian matrix of system (6) is given by;

$$J = \begin{bmatrix} -\beta\alpha\rho I - (1-\rho)\beta\alpha I - \mu & 0 & 0 & -\beta\alpha\rho S - (1-\rho)\beta\alpha S & 0 \\ \beta\alpha\rho I & -(1-\kappa_1)\gamma - \mu & 0 & \beta\alpha\rho S & 0 \\ (1-\rho)\beta\alpha I & 0 & -(1-\kappa)\gamma - \mu & (1-\rho)\beta\alpha S & 0 \\ 0 & (1-\kappa_1)\gamma & (1-\kappa)\gamma & -(\mu+\eta+\delta & 0 \\ 0 & 0 & 0 & \eta & -(\mu+\delta) \end{bmatrix} \tag{15}$$

Theorem 1: The disease-free equilibrium of the system (1) is locally asymptotically stable if $R_0 < 1$.
Proof

At DFE the Jacobian matrix equation (9) becomes;

$$J = \begin{bmatrix} -\mu & 0 & 0 & \beta\alpha\rho S^0 - (1-\rho)\beta\alpha S^0 & 0 \\ 0 & -(1-\kappa_1)\gamma - \mu & 0 & \beta\alpha\rho S^0 & 0 \\ 0 & 0 & -(1-\kappa)\gamma - \mu & (1-\rho)\beta\alpha S^0 & 0 \\ 0 & 1-\kappa_1)\gamma & (1-\kappa)\gamma & -(\mu+\eta+\delta) & 0 \\ 0 & 0 & 0 & \eta & -(\mu+\delta) \end{bmatrix} \tag{16}$$

The eigenvalues are obtained by solving the eqaution;

$$\begin{vmatrix} -\mu-\lambda & 0 & 0 & \beta\alpha\rho S^0-(1-\rho)\beta\alpha S^0 & 0 \\ 0 & [-(1-\kappa_1)\gamma-\mu]-\lambda & 0 & \beta\alpha\rho S^0 & 0 \\ 0 & 0 & [-(1-\kappa)\gamma-\mu]-\lambda & (1-\rho)\beta\alpha S^0 & 0 \\ 0 & 1-\kappa_1)\gamma & (1-\kappa)\gamma & [-(\mu+\eta+\delta)]-\lambda & 0 \\ 0 & 0 & 0 & \eta & [-(\mu+\delta)]-\lambda \end{vmatrix} = 0 \quad (17)$$

The first and the fifth eigenvalues of equation (17) are;

$$\left.\begin{aligned} \lambda_1 &= -\mu \\ \lambda_5 &= -\mu-\delta \end{aligned}\right\} \quad (18)$$

The other three eigenvalues are obtained by solving the polynomial;

$$\begin{vmatrix} [-(1-\kappa_1)\gamma-\mu]-\lambda & 0 & \beta\alpha\rho S^0 \\ 0 & [-(1-\kappa)\gamma-\mu]-\lambda & (1-\rho)\beta\alpha S^0 \\ 1-\kappa_1)\gamma & (1-\kappa)\gamma & [-(\mu+\eta+\delta)]-\lambda \end{vmatrix} = 0 \quad (19)$$

Using first row cofactor expansion, the associated characteristic equation of equation (19) is given by;

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0 \quad (20)$$

Expanding and grouping like terms in powers of λ we obtain the coefficients as;

$$\begin{aligned} A_1 &= [(1-\kappa_1)\gamma+\mu] + [(1-\kappa)\gamma+\mu] + (\mu+\eta+\delta) \\ A_2 &= [(1-\kappa_1)\gamma+\mu]\{[(1-\kappa)\gamma+\mu] + (\mu+\eta+\delta)\} + \{(1-\kappa)\gamma+\mu](\mu+\eta+\delta) - (1-\kappa)(1-\rho)\beta\gamma\alpha S^0\} + \beta\alpha\rho S^0 \\ A_3 &= [(1-\kappa_1)\gamma+\mu][(1-\kappa)\gamma+\mu](\mu+\eta+\delta)\left(1-\frac{\beta\alpha\pi}{\mu(\mu+\eta+\delta)}\left[\frac{\rho(1-\kappa_1)\gamma}{(1-\kappa_1)\gamma+\mu}+\frac{(1-\rho)(1-\kappa)\gamma}{(1-\kappa)\gamma+\mu}\right]\right) \end{aligned}$$

(21)

A_3 simpifies to $A_3 = [(1-\kappa_1)\gamma+\mu][(1-\kappa)\gamma+\mu](\mu+\eta+\delta)\,(1-R_0)$

Using Routh-Hurwitz Criterion of a polynomial of order 3, using equation (21) then $A_1 > 0$ since all the parameters are positive and less than 1.
$A_3 > 0$ implies that $R_0 < 1$ and that $A_1A_2 - A_3 > 0$ is also satisfied. All eugenvalues have negative real part and hence the DFE is locally asymptotically stable.

2.4 Global Stability Analysis of DFE

In this section, the global asymptotic stability of the disease free equilibrium is analyzed using the theorem by Castillo Chavez et al [4] is used to prove the global stabillity of DFE.

Theorem 2: The system (6) can be written as;

$$\frac{dX}{dt} = F(X,Z), \frac{dz}{dt} \quad = G(X,Z) \Big\} \tag{22}$$

where $X = S$ represents the disease free population and $Z = (E_1, E_2, I, A)$ represents the infected classes. The Disease Free Equilibrium of system (6) is given by, DFE = (S^0,$E_1{}^0$,$E_2{}^0$,I^0,A^0) = $(\frac{\pi}{\mu}, 0, 0, 0, 0)$ is globally asymptotically stable if $R_0 < 1$ and locally asymptotically stable if assumptions (H1) and (H2) are satisfied;

$H1$: for $dXdt = F(X, 0)$, the DFE is globally asymptotically stable.

$H2$: $G(X,Z) = AZ - \tilde{G}(X,Z)$, $\tilde{G}(X,Z) \geq 0 for$(X,Z) $\in \mathbb{R}^5$ is an M- matrix (all off-diagonal elements are non-negative) and $\mathbb{R}^5$ is the region where the model makes biological sense.

Proof: In our case $F(X, 0) = \pi - \mu S, Z = (E_1, E_2, I, A)$, then;

$$A = \begin{bmatrix} -(1-\kappa_1)\gamma - \mu & 0 & \rho\beta\alpha S^0 & 0 \\ 0 & -(1-\kappa)\gamma - \mu & (1-\rho)\beta\alpha S^0 & 0 \\ (1-\kappa_1)\gamma & (1-\kappa)\gamma & -(\mu+\eta+\delta) & 0 \\ 0 & 0 & \eta & -(\mu+\delta) \end{bmatrix} \tag{23}$$

$$AZ = \begin{bmatrix} (-(1-\kappa_1)\gamma - \mu)E_1 + \rho\beta\alpha S^0 I \\ (-(1-\kappa)\gamma - \mu)E_2 + (1-\rho)\beta\alpha S^0 I \\ (1-\kappa_1)\gamma E_1 + (1-\kappa)\gamma E_2 - (\mu+\eta+\delta)I \\ \eta I - (\mu+\delta)A \end{bmatrix} \tag{24}$$

where;

$$Z = \begin{bmatrix} E_1 \\ E_2 \\ I \\ A \end{bmatrix} \tag{25}$$

$$G(X,Z) = \begin{bmatrix} \rho\beta\alpha SI + (-(1-\kappa_1)\gamma - \mu)E_1 \\ (1-\rho)\beta\alpha SI + (-(1-\kappa)\gamma - \mu)E_2 + \\ (1-\kappa_1)\gamma E_1 + (1-\kappa)\gamma E_2 - (\mu+\eta+\delta)I \\ \eta I - (\mu+\delta)A \end{bmatrix} \tag{26}$$

Using the second condition, we then obtain;
$\tilde{G}(X,Z) = AZ - G(X,Z)$

$$\tilde{G}(X,Z) = \begin{bmatrix} \rho\beta\alpha(S^0 - S)I \\ (1-\rho)\beta\alpha(S^0 - S)I \\ 0 \\ 0 \end{bmatrix} \geq 0$$

It follows that $\tilde{G}(X,Z) \geq 0$ if $S^0 \geq S$. Conditions $H1$ and $H2$ are satisfied and thus the DFE is globally asymptotically stable for $R_0 < 1$.

3 Numerical Simulation and Discussion

In this section, we present the numerical simulations showing how E_1, E_2, I and A populations are affected by use of vaginal rings and injectable PrEP as preventive measures while engaging in sex.

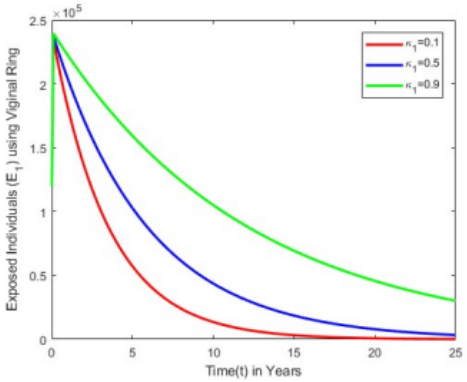

Figure 2: Variation of the Exposed (E_1) population population while using Vaginal rings with time

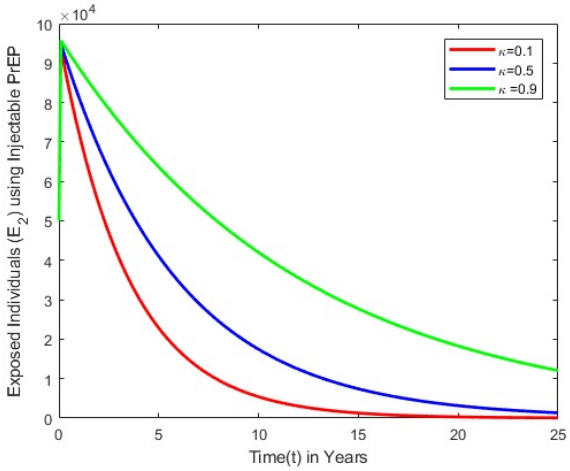

Figure 3: Variation of the Exposed (E_2) population while using Injectable PrEP with time

Figure 2 and Figure 3 respectively shows the impact of using vaginal rings and injectable PrEP with different adherence levels (κ and κ_1). When the use is low ($\kappa = \kappa_1 = 0.1$), FSWs engage in unprotected sex, which shortens the time it takes to contract HIV if exposed to the virus. On the other hand, when the use is high ($\kappa = \kappa_1 = 0.9$), consistent use of vaginal rings and injectable PrEP provides better protection, resulting in a longer time before contracting HIV, even if exposed.

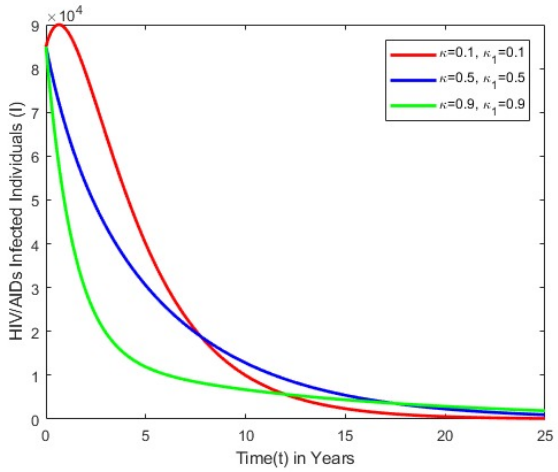

Figure 4: Variation of the HIV/AIDS Infections (I) while using Vaginal rings and Injectable PrEP with time

Figure 4 shows that low usage of both preventive strategies; vaginal rings and injectable PrEP results in an initial increase in the HIV-infected population and a rapid progression to full AIDS. In contrast, widespread adoption of these preventive strategies reduces HIV/AIDS infections and significantly delays the progression to full AIDS among those infected.

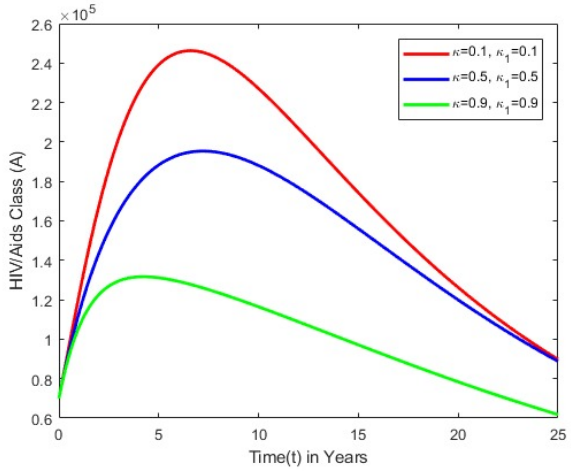

Figure 5: Variation of the HIV/AIDS cases (A) while using Vaginal rings and Injectable PrEP with time

Figure 5 shows that proper and consistent use of vaginal rings and injectable PrEP reduces the AIDS population

drastically.

4 Conclusion

In this paper, we examined the transmission dynamics of HIV/AIDS with prevention strategies among female sex workers (FSWs) in Kenya. These strategies includes: use of vaginal rings and injectable PrEP. The basic reproduction number is computed using the next generation matrix approach. The analysis shows that the disease free equilibrium point is both locally and globally asymptotically stable whenever $R_0 < 1$. From the numerical simulation, it is shown that the use of both vaginal rings and injectable PrEP are effective in reducing HIV/AIDS exposure and infections among high-risk populations, specifically female sex workers. Based on our findings, we recommend that the Ministry of Health in Kenya (MoH-K) ought to guarantee the availability and affordability of both injectable PrEP and vaginal rings to FSWs. Currently, the cost ranges from 1 to 8 US dollars, which is relatively expensive for female sex workers (FSWs) who depend on sex work as their primary source of income. Additionally, the MoH-K should have a targeted education campaigns to inform these FSWs on the importance of using vaginal rings and injectable PrEP to protect themselves from HIV/AIDS exposure and infection.

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