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Male Circumcision: A Means to Reduce HIV Transmission between Truckers and Female Sex Workers in Kenya

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Abstract

Kenya records over 1.5 million cases of HIV-infected people with a prevalence of 4.8% among adults in 2019, ranking Kenya as the seventh-largest HIV population in the world. A recent study showed that 55.9% of Kenyan truckers pay for sex in while 46.6% had a regular partner along their trucking route in addition to a wife or girlfriend at home. The complexity in the sexual network of Truckers, which can be a conduit for the widespread of HIV, necessitated the need to better understand the dynamics of transmission of HIV/AIDS between truckers and female sex workers. In this study, a model is formulated for HIV/AIDS dynamics along the Northern corridor highway in Kenya. The reproduction number, disease-free equilibrium and endemic equilibrium points were determined and their stabilities were also determined using the next-generation matrix method. The disease-free equilibrium is stable when $R_{0u} < 1, R_{0c} < 1$ and $R_{0f} < 1$ while the endemic equilibrium point is stable when $R_{0u} > 1, R_{0c} > 1$ and $R_{0f} > 1$. It is found that circumcision can be used as an intervention to minimize the infection of HIV among truckers and female sex workers.

Keywords: HIV/AIDS dynamics, truckers, female sex workers (FSW), male circumcision.
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1. Introduction

HIV/AIDS is a major global public health issue and in 2018 an estimated 37.9 million people were living with HIV. The global HIV prevalence of adults was 0.8%. In 2018, there were roughly 1.7 million new HIV infections and 770,000 people died of AIDS-related illnesses according to Case et. al [1]. The majority of the people living with HIV were located in low- and middle-income countries, with an estimated 68% of these living

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in sub-Saharan Africa. Among this group, 20.6 million are living in Eastern and Southern Africa which recorded 800,000 new HIV infections in 2018 [1]. In 2018, 25.7 million people living with HIV in the African Region. The African Region also accounted for about two-thirds of the global total new HIV infections. In 1996, 10.5% of Kenyans were living with HIV, although the prevalence level almost declined to a half-standing at 5.9% by 2015 due to scaling up of HIV treatment and care according to Maina et. al [2]. In 2016, 64% of people living with HIV were on treatment and 51% of these were virally suppressed [3]. In 2018, about 1.1 million people were infected with HIV in the African region. Kenya has the seventh-largest HIV epidemic in the world with 1.5 million people living with HIV and 21,000 people dying from AIDS-related illnesses yearly [4].

In sub-Saharan Africa, truckers and sex workers who provide services to the truckers at major transport stopovers, are at higher risk of contracting HIV compared to other cohorts working in other settings [5]. In East Africa, HIV rates remain high along the border posts and major highways [6]. A study in 1991 among truck drivers in Kenya found that 18% were HIV-positive; 61% of the sample reported having visited female sex workers (FSWs) and only 32% had ever used condoms [7]. According to Kelvin et. al [8] and Kelvin et. al [6], a study among 3,805 truckers in Kenya found that 55.9% had paid for sex in the past 6 months and 46.6% had a regular partner along their trucking route in addition to a wife or girlfriend at home. Due to complex sexual networks, truckers can be a conduit for the spread of HIV across partner types (commercial, regular partners on their trucking route, wives or girlfriends at home) as well as across national and international borders according to International Labor Organization (2005). More specifically HIV/AIDS epidemic in vulnerable groups which involves sex workers and truckers require special attention to reduce its spread. Despite approximately 20 years of the epidemic in the East African region, these vulnerable populations which include truckers and sex workers were largely ignored according to Hawken [9], Voeten et. al [10], Ghani and Aral [11]. Localized programs had been put in place to cater for the needs of highway-based sex workers and their clients. In Kenya, sex workers have the highest reported HIV prevalence of any group. The most recent data from 2011 showed that 29.3% of female sex workers were living with HIV [12]. In 2015, a study of female sex workers in Nairobi found that around one-third were living with HIV [13]. Female sex workers are reportedly better at protecting themselves from HIV transmission compared to other groups who are vulnerable to HIV such as men who have sex with men. This may be as a result of the fact that 76% of female sex workers were estimated to be able to access HIV services, higher than among other key population groups. Transactional sex which involves cash transfer, however, has had specific locations along the highway many of which are the towns along the highway. Early surveillance indicated a very strong relationship between the HIV prevalence and locations along the northern corridor highway between Mombasa and the Ugandan border towns according to Ferguson et. al [14]. The growth of HIV/AIDS led to its diffusion from the original focus along the main road arteries and into main towns and later to rural parts according to Ferguson and Morris[15]. Subsequently, the mobility of the sex workers along the highway and back to their rural areas has led to the spread of the HIV/AIDS epidemic off the main highway. In 2004 a study by the AIDS Control Unit (ACU) of the ministry of transport Kenya together with other stakeholders from the transport sector on the response to HIV and AIDS in the transport sector found that the prevalence of HIV infection among

employees in the transport sector and their irregular sexual partners had increased over the last 15 years depending on their geographical location up to 25% or more [14]. The HIV epidemic and sexually transmitted infections were due to unprotected heterosexual contact UNAIDS (2004). The epidemiology of HIV and AIDS in Kenya recorded extremely high rates of HIV and other sexually-transmitted infections (STIS) among sex workers (34-75%) and truckers (26%, 27%) [16, 7, 17]. More recent work has reaffirmed the same continuing high prevalence among these vulnerable groups [12, 18]. Waziri et. al [19] did a nonlinear deterministic mathematical model for HIV/AIDS dynamics with treatment and vertical transmission which was analysed by stability theory of differential equations. The results of the model indicated that the disease-free equilibrium was locally stable at a threshold parameter less than one and was unstable when the parameter was greater than one. The DFE was not globally unstable due to the existence of the forward bifurcation at a threshold parameter equal to one. It is important to also mention that fractional calculus has been adopted recently to identify the salient trends that might have been missed in the classic calulus (see [20, 21, 22, 23, 24]).

Due to dwindling donor funding for HIV/AIDS in Kenya, then this study developed an HIV/AIDS model to study the transmission dynamics between truckers and FSWs in Kenya. This study shall provide answers to the following research questions;

1. How does the rate of circumcision affect HIV transmission among truckers and FSWs in Kenya?
2. How does the rate of female-to-male HIV transmission affect HIV transmission among truckers and FSWs in Kenya?
3. How does the rate of female-to-uncircumcised-male HIV transmission affect HIV transmission among truckers and FSWs in Kenya?
4. How does the rate of female-to-circumcised-male HIV transmission affect HIV transmission among truckers and FSWs in Kenya?

2. Methodology

2.1. Mathematical Formulation

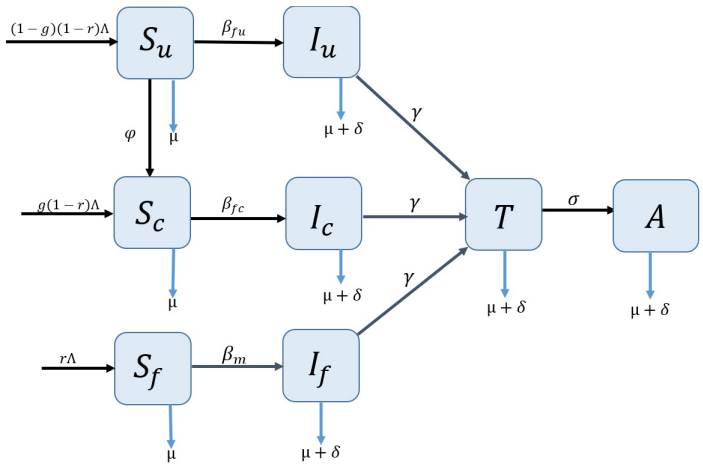

Figure 1: Flow diagram for the mathematical model

A mathematical model is formulated to study HIV/AIDS transmission dynamics between truckers and FSWs in Kenya. Figure 1 shows the diagrammatic representation of the mathematical model. The population is divided into eight compartments, namely; (1) three susceptible classes S_u, S_c, S_f of uncircumcised susceptible male group, circumcised susceptible male group and susceptible female group respectively. (2) three infected classes I_u, I_c, I_f of uncircumcised Infected males, circumcised Infected males and Infected females. (3) treated group T and (4) the AIDS group A. The model is based on the following assumptions:

1. All the infected individuals are subjected to treatment.
2. The main clients of the female sex workers were truckers.
3. Only the adult population is considered.
4. Uncircumcised male drivers can be subjected to VMC
5. No births
6. Death occurs at a natural rate of μ in all the classes.

The governing ordinary differential equations are;

$$\frac{dS_u}{dt} = (1-g)(1-r)\Lambda - \beta_{fu} S_u I_u - (\varphi + \mu) S_u, \tag{2.1}$$

$$\frac{dS_c}{dt} = g(1-r)\Lambda + \varphi S_u - \beta_{fc} S_c I_c - \mu S_c, \tag{2.2}$$

$$\frac{dS_f}{dt} = r\Lambda - \beta_m S_f I_f - \mu S_f, \tag{2.3}$$

$$\frac{dI_u}{dt} = \beta_{fu} S_u I_u - (\mu + \gamma + \delta) I_u, \tag{2.4}$$

$$\frac{dI_c}{dt} = \beta_{fc} S_c I_c - (\mu + \gamma + \delta) I_c, \tag{2.5}$$

$$\frac{dI_f}{dt} = \beta_m S_f I_f - (\mu + \gamma + \delta) I_f, \tag{2.6}$$

$$\frac{dT}{dt} = (I_u + I_c + I_f)\gamma - (\mu + \delta + \sigma) T, \tag{2.7}$$

$$\frac{dA}{dt} = \sigma T - (\mu + \delta) A, \tag{2.8}$$

g is the circumcised proportion of males, r is the proportion of females recruited into female sex workers, Λ is the recruitment rate into the susceptible population, γ is the rate of accessing treatment of infected males or females, σ is the rate of progression of treated individuals to AIDS, μ is the Natural mortality rate, δ is the disease-induced mortality rate, φ is the rate of circumcision, β_{fu} is the transmission rate of HIV from females to uncircumcised males, β_{fc} is the transmission rate of HIV from females to circumcised males, β_m is the transmission rate of HIV from males to females.

3. MODEL ANALYSIS

The total population size N satisfies the equation

$$N = S_u + S_c + S_f + I_u + I_c + I_f + T + A$$

and therefore

$$\frac{dN}{dt} = \frac{dS_u}{dt} + \frac{dS_c}{dt} + \frac{dS_f}{dt} + \frac{dI_u}{dt} + \frac{dI_c}{dt} + \frac{dI_f}{dt} + \frac{dT}{dt} + \frac{dA}{dt}$$
$$\Rightarrow \frac{dN}{dt} = \Lambda - \mu N - \delta(I + T + A) \leqslant \Lambda - \mu N$$

and hence $N(t) \to \frac{\Lambda}{\mu}$ as $t \to \infty$ therefore the biologically feasible region for our system is;

$$\Omega = \left\{ (S_u,\ S_c,\ S_f,\ I_u,\ I_c,\ I_f, T, A) \ : S_u,\ S_c,\ S_f,\ I_u,\ I_c,\ I_f, T, A \in \mathbb{R}^+ \text{ and } N \leqslant \frac{\Lambda}{\mu} \right\}$$

is bounded and positively invariant.

3.1. Disease Free Equilibrium (DFE) Point

The disease-free equilibrium is obtained by setting $I_u = I_c = I_f = T = A = 0$ in equation (2.1 - 2.8) and we get

$$S_u = \frac{(1-g)(1-r)\Lambda}{\varphi + \mu},\ S_c = \frac{\Lambda(1-r)(g\mu + \varphi)}{\mu(\varphi + \mu)},\ S_f = \frac{r\Lambda}{\mu}$$

Therefore, the D.F.E is given by

$$E^0 = \left(S_u^0, S_c^0, S_f^0, I_u^0, I_c^0, I_f^0, T^0, A^0\right)$$
$$= \left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\Lambda}{\mu}, 0, 0, 0, 0, 0, \right)$$

3.2. Basic Reproduction Number (R_0)

The reproduction number R_0 is the number of secondary infections that an infected individual can cause when introduced into a susceptible population. The next-generation matrix is used to obtain R_0. Define F_i and V_i as matrices that represent the rate at which new infections occurs and the rate of transfer of individuals out of compartments. The R_0 was obtained from the spectral radius of the $F_0 V_0^{-1}$ matrix. From system (2.1 - 2.8) then;

$$\mathcal{F} = \begin{pmatrix} \beta_{fu} S_u I_u \\ \beta_{fc} S_c I_c \\ \beta_m S_f I_f \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} (\mu + \gamma + \delta) I_u \\ (\mu + \gamma + \delta) I_c \\ (\mu + \gamma + \delta) I_f \\ -\gamma I_u - \gamma I_c - \gamma I_f + (\mu + \delta + \sigma) T \\ -\sigma T + (\mu + \delta) A \end{pmatrix}$$

Let F and V be the Jacobian of $\mathcal{F}$ at DFE and the Jacobian of $\mathcal{V}$ at DFE so that,

$$F = \begin{pmatrix} \beta_{fu} S_u^0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc} S_c^0 & 0 & 0 & 0 \\ 0 & 0 & \beta_m S_f^0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \mu+\gamma+\delta & 0 & 0 & 0 & 0 \\ 0 & \mu+\gamma+\delta & 0 & 0 & 0 \\ 0 & 0 & \mu+\gamma+\delta & 0 & 0 \\ -\gamma & -\gamma & -\gamma & \mu+\delta+\sigma & 0 \\ 0 & 0 & 0 & -\sigma & \mu+\delta \end{pmatrix}$$

Hence for system (2.1 - 2.8) the next generation matrix [25, 26, 27] is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{fu}S_u^0}{\mu+\gamma+\delta} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc}S_c^0}{\mu+\gamma+\delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m S_f^0}{\mu+\gamma+\delta} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The characteristic polynomial of $FV^{-1} - \lambda I$ at DFE is given by

$$\lambda^2 \left(-\lambda + \frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)}\right)\left(-\lambda + \frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)}\right)\left(-\lambda + \frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\varphi+\mu)(\mu+\gamma+\delta)}\right) = 0$$

The eigenvalues are

$$\lambda_1 = \lambda_2 = 0, \lambda_3 = \frac{\beta_m r\check{}}{\mu(\mu+\gamma+\delta)}, \lambda_4 = \frac{\beta_{fc}\check{}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)}, \lambda_5 = \frac{\beta_{fu}(1-g)(1-r)\check{}}{(\varphi+\mu)(\mu+\gamma+\delta)}$$

λ_3, λ_4 and λ_5 gives the basic reproduction numbers for the system (2.1 - 2.8) as follows;

$$R_{of} = \frac{\beta_m r\check{}}{\mu(\mu+\gamma+\delta)}, R_{oc} = \frac{\beta_{fc}\check{}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)}, \text{ and } R_{ou} = \frac{\beta_{fu}(1-g)(1-r)\check{}}{(\varphi+\mu)(\mu+\gamma+\delta)}.$$

3.3. Endemic Equilibrium Point (EEP)

This refers to disease spreading point within the population. Let

$$E^* = (S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*)$$

be the endemic equilibrium point where

$$S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^* > 0.$$

Consider

$$\begin{aligned}
&(1-g)(1-r)\check{} - \beta_{fu}S_u^*I_u^* - (\varphi+\mu)S_u^* = 0, \\
&g(1-r)\check{} + \varphi S_u^* - \beta_{fc}S_c^*I_c^* - \mu S_c^* = 0 \\
&r\check{} - \beta_m S_f^* I_f^* - \mu S_f^* = 0 \\
&\beta_{fu}S_u^*I_u^* - (\mu+\gamma+\delta)I_u^* = 0 \\
&\beta_{fc}S_c^*I_c^* - (\mu+\gamma+\delta)I_c^* = 0 \\
&\beta_m S_f^* I_f^* - (\mu+\gamma+\delta)I_f^* = 0 \\
&(I_u^* + I_c^* + I_f^*)\gamma - (\mu+\delta+\sigma)T^* = 0 \\
&\sigma T^* - (\mu+\delta)A^* = 0
\end{aligned}$$

From which we obtain the endemic equilibrium point;

$$S_u^* = \frac{\mu+\delta+\gamma}{\beta_{fu}},\ S_c^* = \frac{\mu+\delta+\gamma}{\beta_{fc}}, S_f^* = \frac{\mu+\delta+\gamma}{\beta_m}, I_u^* = \frac{\beta_{fu}(1-g)(1-r)\check{}-(\varphi+\mu)(\mu+\delta+\gamma)}{\beta_{fu}(\mu+\delta+\gamma)},$$

$$I_c^* = \frac{\beta_{fu}\beta_{fc}g(1-r)\check{}+\varphi\beta_{fc}(\mu+\delta+\gamma)-\mu\beta_{fu}(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}(\mu+\delta+\gamma)},\ \ I_f^* = \frac{\beta_m r\check{}-\mu(\mu+\delta+\gamma)}{\beta_m(\mu+\delta+\gamma)},$$

$$T^* = \frac{\beta_{fu}\beta_{fc}\beta_m\gamma(1-g)(1-r)\check{}-\beta_{fc}\beta_m\gamma(\mu+\varphi)(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)} + \frac{\beta_{fu}\beta_{fc}\beta_m r\check{}\gamma-\beta_{fu}\beta_{fc}\mu\gamma(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)}$$
$$+ \frac{\beta_{fu}\beta_{fc}\beta_m\gamma g(1-r)\check{}-\varphi\gamma\beta_{fc}\beta_m(\mu+\delta+\gamma)-\mu\gamma\beta_{fu}\beta_m(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)}$$

$$A^* = \frac{\beta_{fu}\beta_{fc}\beta_m\gamma\sigma(1-g)(1-r)\check{}-\beta_{fc}\beta_m\gamma\sigma(\mu+\varphi)(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$
$$+ \frac{\beta_{fu}\beta_{fc}\beta_m r\check{}\gamma\sigma-\beta_{fu}\beta_{fc}\mu\gamma\sigma(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$
$$+ \frac{\beta_{fu}\beta_{fc}\beta_m\gamma g\sigma(1-r)\check{}-\varphi\gamma\sigma\beta_{fc}\beta_m(\mu+\delta+\gamma)-\mu\gamma\sigma\beta_{fu}\beta_m(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$

3.4. Local Stability of the disease-free equilibrium and endemic equilibrium point

Theorem 3.1. *Define* $R_0 = \{R_{of}, R_{oc}, R_{ou}\}$*, then the disease-free equilibrium* E^0 *of the system (2.1 - 2.8) is locally asymptotically stable if* $R_0 < 1$ *while the endemic equilibrium point* E^* *is locally asymptotically stable if* $R_0 > 1$.

Proof. The Jacobian matrix of the system is given by

$$J = \begin{pmatrix}
-\beta_{fu}I_u-(\varphi+\mu) & 0 & 0 & -\beta_{fu}S_u & 0 & 0 & 0 & 0 \\
\varphi & -\beta_{fc}I_c-\mu & 0 & 0 & -\beta_{fc}S_c & 0 & 0 & 0 \\
0 & 0 & -\beta_m I_f-\mu & 0 & 0 & -\beta_m S_f & 0 & 0 \\
\beta_{fu}I_u & 0 & 0 & \beta_{fu}S_u-k & 0 & 0 & 0 & 0 \\
0 & \beta_{fc}I_c & 0 & 0 & \beta_{fc}S_c-k & 0 & 0 & 0 \\
0 & 0 & \beta_m I_f & 0 & 0 & \beta_m S_f-k & 0 & 0 \\
0 & 0 & 0 & \gamma & \gamma & \gamma & -k_1 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \sigma & -k_2
\end{pmatrix}$$

where $k = \mu+\gamma+\delta$, $k_1 = \mu+\delta+\sigma$, $k_2 = \mu+\delta$. The first two eigenvalues are $\lambda_1 = -(\mu+\delta)$, and $\lambda_2 = -(\mu+\delta+\sigma)$, and the remaining six eigenvalues $\lambda_3, \lambda_4, \cdots, \lambda_8$ are obtained from the equations;

$$\lambda^2 - (\beta_m(S_f - I_f) - k - \mu)\lambda + \beta_m(kI_f - \mu S_f) + \mu k = 0,$$
$$\lambda^2 - (\beta_{fc}(S_c - I_c) - k - \mu)\lambda + \beta_{fc}(kI_c - \mu S_c) + \mu k = 0,$$
$$\lambda^2 - (\beta_{fu}(S_u - I_u) - (\varphi+\mu) - k)\lambda + \beta_{fu}(kI_u - (\varphi+\mu)S_u) + k(\varphi+\mu) = 0.$$

It is clear that λ_1 and λ_2 are negative. The requirements for the other eigenvalues to be negative are given below; □

For negative λ_3, λ_4, we require

$$\frac{\beta_m (S_f - I_f)}{k+\mu} < 1, \text{ and } \frac{\beta_m (\mu S_f - k I_f)}{\mu k} < 1.$$

For negative λ_5, λ_6, we require

$$\frac{\beta_{fc} (S_c - I_c)}{k+\mu} < 1, \text{ and } \frac{\beta_{fc} (\mu S_c - k I_c)}{\mu k} < 1.$$

For negative λ_7, λ_8, we require

$$\frac{\beta_{fu} (S_u - I_u)}{k+\mu+\varphi} < 1, \text{ and } \frac{\beta_{fu} ((\mu+\varphi) S_u - k I_u)}{k (\mu+\varphi)} < 1.$$

CASE 1

Substituting DFE

$$E^0 = \left(\frac{(1-g)(1-r)\breve{}}{\varphi+\mu}, \frac{\breve{}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\breve{}}{\mu}, 0, 0, 0, 0, 0, \right)$$

The requirements for stability therefore are;

1. λ_3, λ_4, are negative if

$$\frac{\beta_m r\breve{}}{\mu(2\mu+\gamma+\delta)} < 1, \text{ and } \frac{\beta_m r\breve{}}{\mu(\mu+\gamma+\delta)} < 1$$

and thus

$$\frac{\beta_m r\breve{}}{\mu(2\mu+\gamma+\delta)} < \frac{\beta_m r\breve{}}{\mu(\mu+\gamma+\delta)} < 1 \Rightarrow R_{of} < 1.$$

2. λ_5, λ_6 are negative if

$$\frac{\breve{}\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(2\mu+\gamma+\delta)} < 1, \frac{\breve{}\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\mu+\gamma+\delta)(\varphi+\mu)} < 1$$

and thus

$$\frac{\breve{}\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(2\mu+\gamma+\delta)} < \frac{\breve{}\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\mu+\gamma+\delta)(\varphi+\mu)} < 1 \Rightarrow R_{oc} < 1$$

3. λ_7, λ_8 are negative if

$$\frac{\beta_{fu}(1-g)(1-r)\breve{}}{(2\mu+\gamma+\delta+\varphi)(\varphi+\mu)} < 1, \frac{\beta_{fu}(1-g)(1-r)\breve{}}{(\mu+\gamma+\delta)(\varphi+\mu)} < 1$$

and thus

$$\frac{\beta_{fu}(1-g)(1-r)\breve{}}{(2\mu+\gamma+\delta+\varphi)(\varphi+\mu)} < \frac{\beta_{fu}(1-g)(1-r)\breve{}}{(\mu+\gamma+\delta)(\varphi+\mu)} \Rightarrow R_{ou} < 1.$$

Hence, E^0 is locally asymptotically stable if $R_0 < 1$. ■

CASE 2

Substituting the EEP

$$E^* = (S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*),$$

the requirements for stability become;

1. λ_3, λ_4, are negative if

$$\frac{\beta_m (S_f^* - I_f^*)}{2\mu + \gamma + \delta} < 1, \text{ and } \frac{\beta_m (\mu S_f^* - (\mu + \gamma + \delta) I_f^*)}{\mu (\mu + \gamma + \delta)} < 1,$$

which, on substitution of S_f^* and I_f^*, become

$$\frac{\beta_m r\breve{}}{(\mu + \delta + \gamma)(2\mu + \gamma + \delta)} > 0, \text{ and } \frac{\beta_m r\breve{}}{\mu (\mu + \gamma + \delta)} = R_{of} > 1.$$

The first condition is already satisfied since $\beta_m, r, \breve{}, \mu, \delta, \gamma > 0$ and hence, λ_3 and λ_4 are negative if $R_{of} > 1$.

2. λ_5, λ_6 are negative if

$$\frac{\beta_{fc} (S_c^* - I_c^*)}{2\mu + \gamma + \delta} < 1, \text{ and } \frac{\beta_{fc} (\mu S_c^* - (\mu + \gamma + \delta) I_c^*)}{\mu (\mu + \gamma + \delta)} < 1$$

which, on substitution of S_c^* and I_c^*, become

$$\frac{(r-1) g\breve{} \beta_{fu} - \varphi (\mu + \delta + \gamma)}{(\mu + \delta + \gamma)(2\mu + \delta + \gamma) \beta_{fu}} < 1, \text{ and } \frac{\beta_{fc} ((1-r) g\breve{} \beta_{fu} + \varphi (\mu + \gamma + \delta))}{\mu \beta_{fu} (\mu + \gamma + \delta)} > 1.$$

The first condition is true since $r - 1 < 0$ implies that

$$(r-1) g\breve{} \beta_{fu} - \varphi (\mu + \delta + \gamma) < 0.$$

The second condition becomes

$$\frac{\beta_{fc} ((1-r) g\breve{} \beta_{fu} + \varphi (\mu + \gamma + \delta))}{\mu \beta_{fu} (\mu + \gamma + \delta)(\mu + \varphi)} > \frac{1}{(\mu + \varphi)}$$

$$\frac{\beta_{fc} (1-r) (g\mu + \varphi)\breve{}}{\mu (\mu + \gamma + \delta)(\mu + \varphi)} \frac{g}{(g\mu + \varphi)} + \frac{\beta_{fc} \varphi}{\mu \beta_{fu} (\mu + \varphi)} > \frac{1}{(\mu + \varphi)}$$

$$R_{oc} > \frac{(g\mu + \varphi)}{g (\mu + \varphi)} \left(1 - \frac{\beta_{fc} \varphi}{\mu \beta_{fu}}\right) > 1.$$

3. λ_7, λ_8 are negative if

$$\frac{\beta_{fu} (S_u^* - I_u^*)}{2\mu + \gamma + \delta + \varphi} < 1, \text{ and } \frac{\beta_{fu} ((\mu + \varphi) S_u^* - (\mu + \gamma + \delta) I_u^*)}{(\mu + \gamma + \delta)(\mu + \varphi)} < 1.$$

which, on substitution of S_u^* and I_u^*, become

$$\frac{\beta_{fu} (1-g) (1-r)\breve{}}{(2\mu + \gamma + \delta + \varphi)(\mu + \delta + \gamma)} > 0, \text{ and } \frac{\beta_{fu} (1-g) (1-r)\breve{}}{(\mu + \gamma + \delta)(\varphi + \mu)} > 1.$$

The first condition is already satisfied since $0 < g < 1$, $0 < r < 1$ and hence, λ_7 and λ_8 are negative if $R_{ou} > 1$.

Hence, E^* is locally asymptotically stable if $R_0 > 1$. ■

4. Analysis of Results and Discussion

The dynamics of HIV transmission among truckers and FSWs in Kenya modelled by equation (2.1 - 2.8) is solved numerically using MATLAB (see [28] for other methods of solution) and the results are extracted. Default values of the parameters shown in Table (4) form the basis of our analysis. The behaviour of different state variables over time is studied. This section is divided into two subsections; Analysis of Results and Discussion of Results. The Analysis of Results subsection highlights the results obtained from the numerical analysis. The Discussion of Results subsection gives a thorough and physical interpretation of the results obtained.

Table 1: default values of the parameters

Parameter	Value	Source
g	0.85	[29]
r	0.069	[30]
Λ	0.3	Estimated
γ	0.34	[31]
σ	0.08	Estimated
μ	0.0539	[31]
δ	0.016	[32]
φ	0.84	[33]
β_{fu}	0.0128	[33]
β_{fc}	0.0051	[33]
β_m	0.049	[34]

4.1. Analysis of Results

Figures (2 - 5) show the effect of rate β_{fc} of HIV transmission from females to males on the HIV transmission dynamics among truckers and FSWs. Figure (2) shows that increase in β_{fc} leads to an increase in the AIDS population in the system. As β_{fc} increases, Figure (3) shows an increase in I_c up to a peak value since more circumcised males are infected over a short period between approximately one and three years. In Figure (4), increase in β_{fc} produces a decrease in S_c population due to an increase in the number of infections. Figure (5) shows that within the first 8 years, an increase in β_{fc} leads to an increase in the population seeking treatment over a short period. Afterwards, the population seeking treatment reduced with time.

Figure (6) shows that increase in the rate β_{fu} of transmission from female to an uncircumcised male leads to an increase in the uncircumcised I_u truckers' population due to increased infections.

The effects of the rate β_m of transmission of HIV from males to females on the spread of HIV among truckers and FSWs are displayed in Figure (7 - 8). In figure (7), increase in β_m produces a decrease in S_f population. This is due to an increase in the number of infections caused by increased β_m. In figure (8), I_f population increases with β_m up to peak value in the second year, then decreases onwards.

The effects of the circumcision rate φ on the spread of HIV among truckers and FSWs are exhibited in Figures (9 - 13). Figure (9) shows that an increase in the circumcision

rate increases the treated population. Figure (10) shows that increase in φ produces a decrease in the S_u population as more individuals proceed to S_c. Figure (11) shows that increasing φ produces an increase in S_c because in Kenya the circumcision rate is slightly high approximately 0.84 implying most of the population is circumcised. In Figure (12), increase in φ significantly reduces I_u due to a decrease in S_u population. In figure (13), as φ increased there was a slight increase in I_c. This is due to a slightly high circumcision rate in Kenya hence increase in φ may not produce much impact on the circumcised susceptible population.

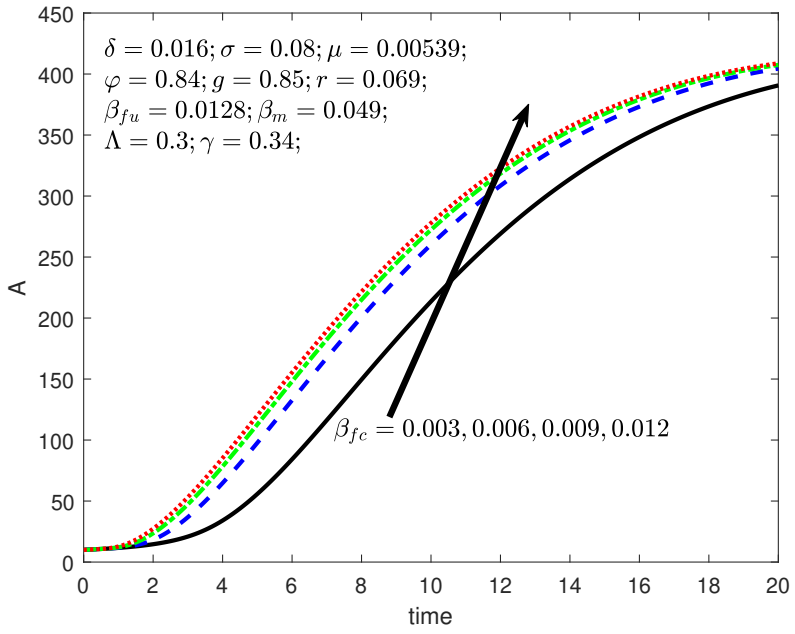

Figure 2: Variation of AIDS population with β_{fc}

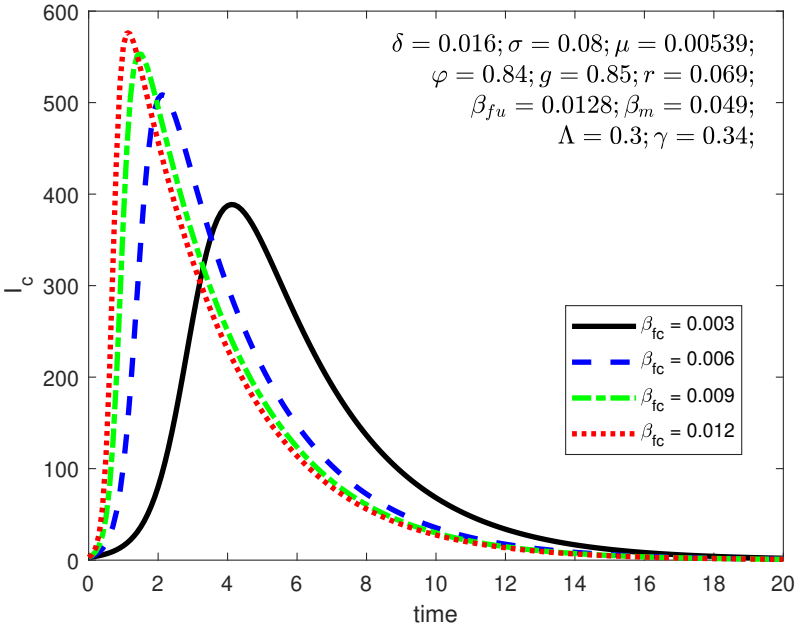

Figure 3: Variation of the infected circumcised male population with β_{fc}

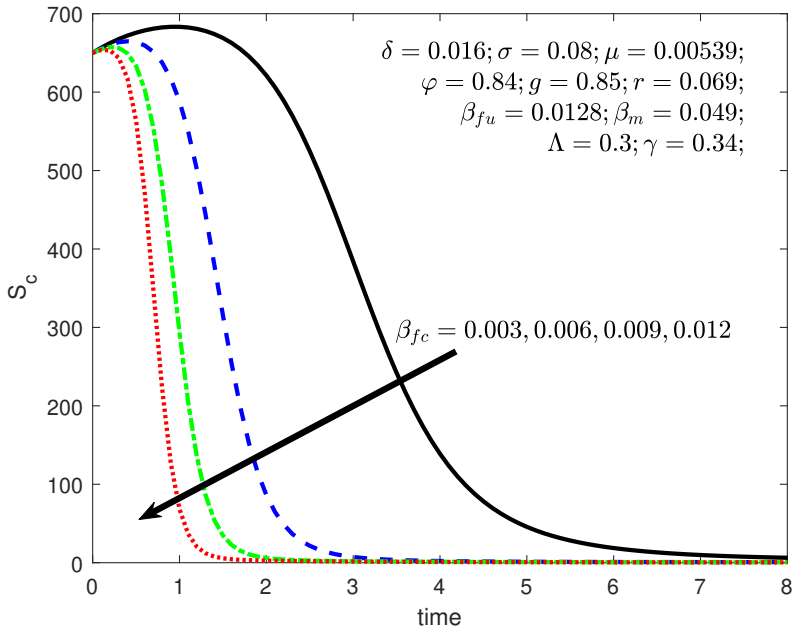

Figure 4: Variation of the susceptible circumcised male population with β_{fc}

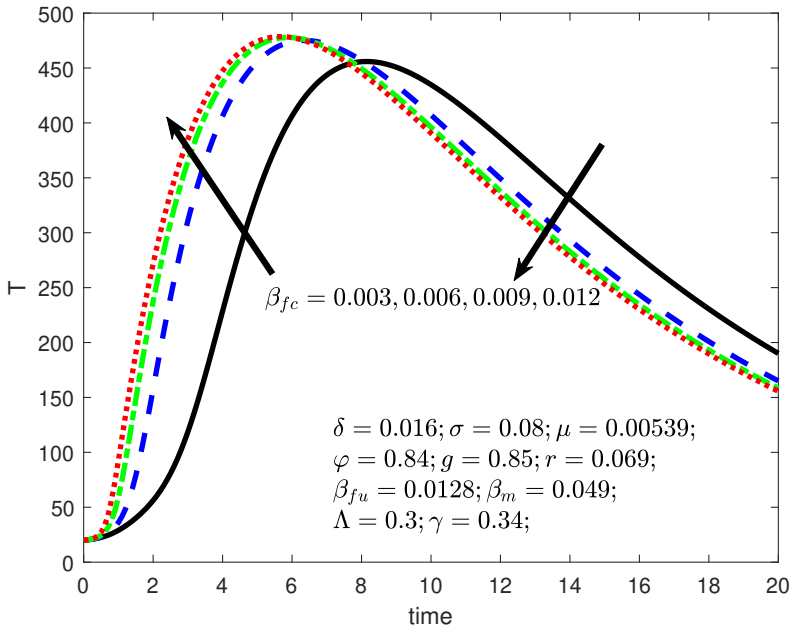

Figure 5: Variation of the treated population with β_{fc}

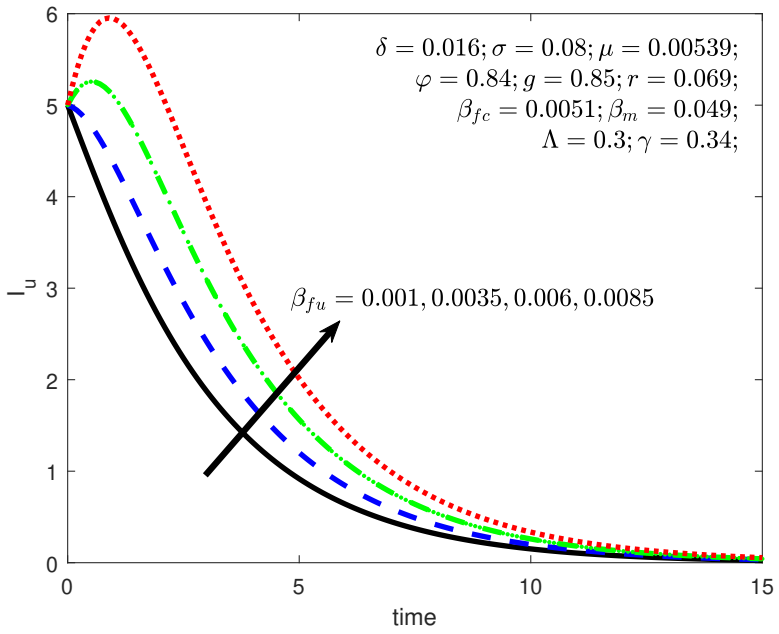

Figure 6: Variation of the infected uncircumcised male population with β_{fu}

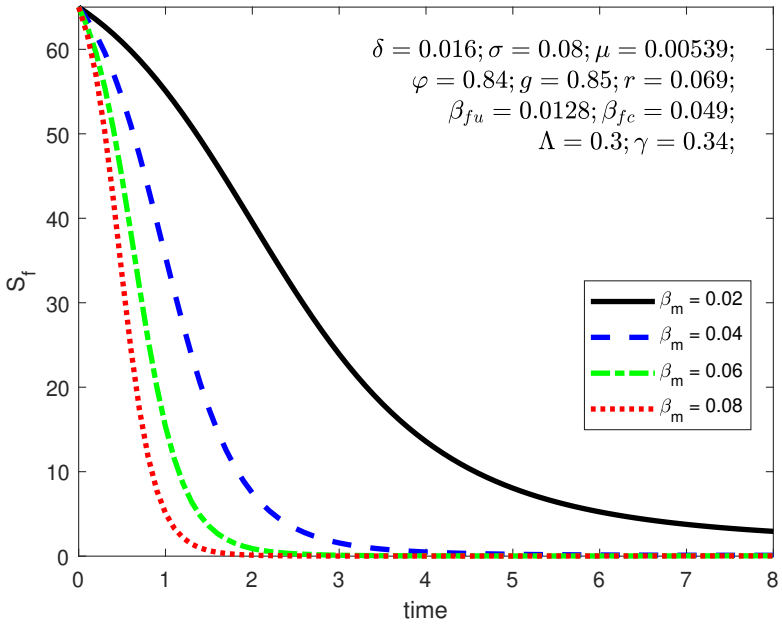

Figure 7: Variation of the susceptible female population with β_m

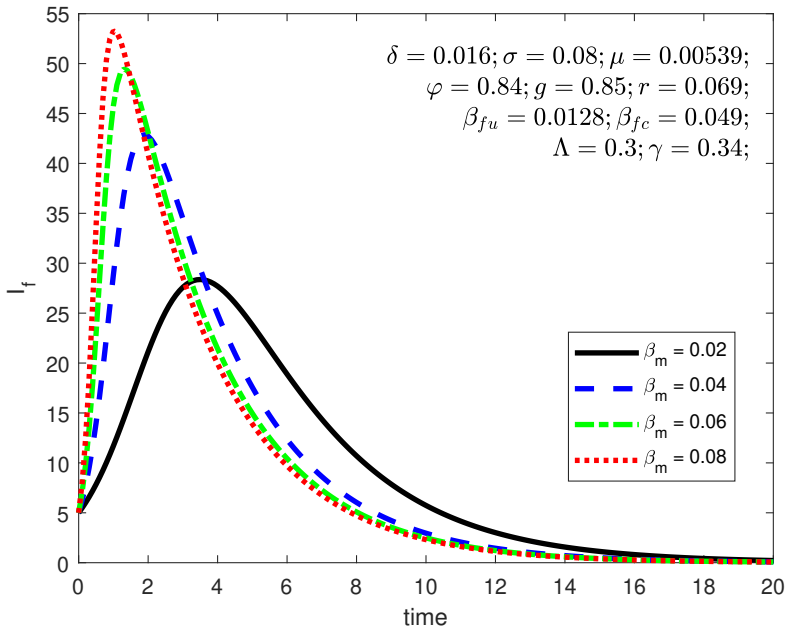

Figure 8: Variation of infected females with β_m

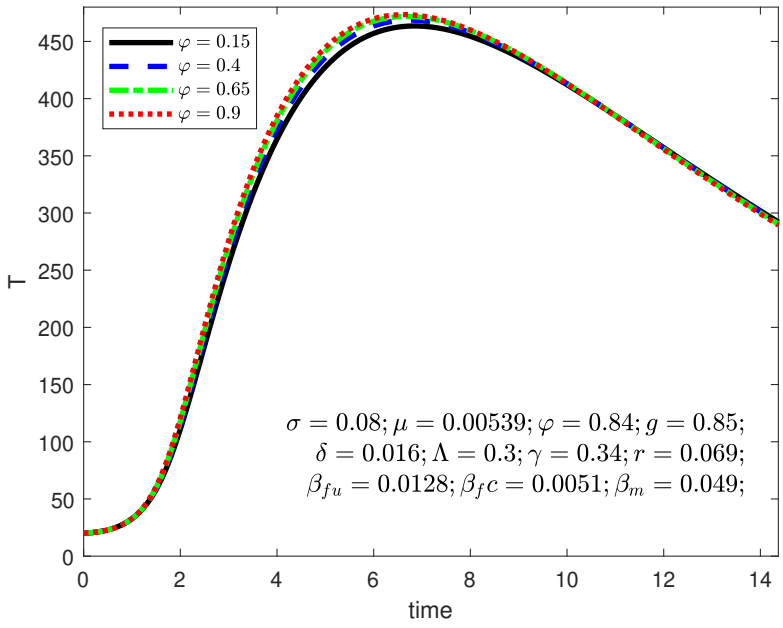

Figure 9: Variation of the treated population with φ

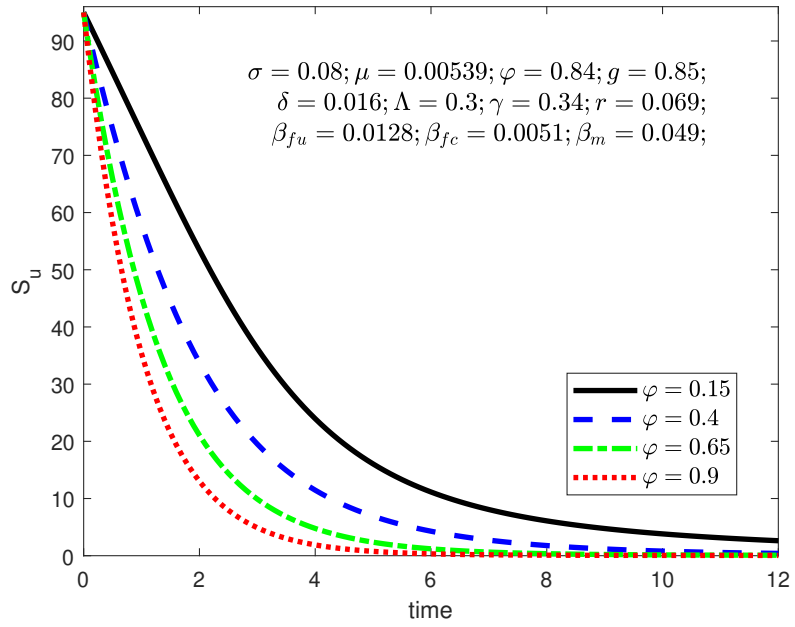

Figure 10: Variation of Uncircumcised male susceptible population with φ

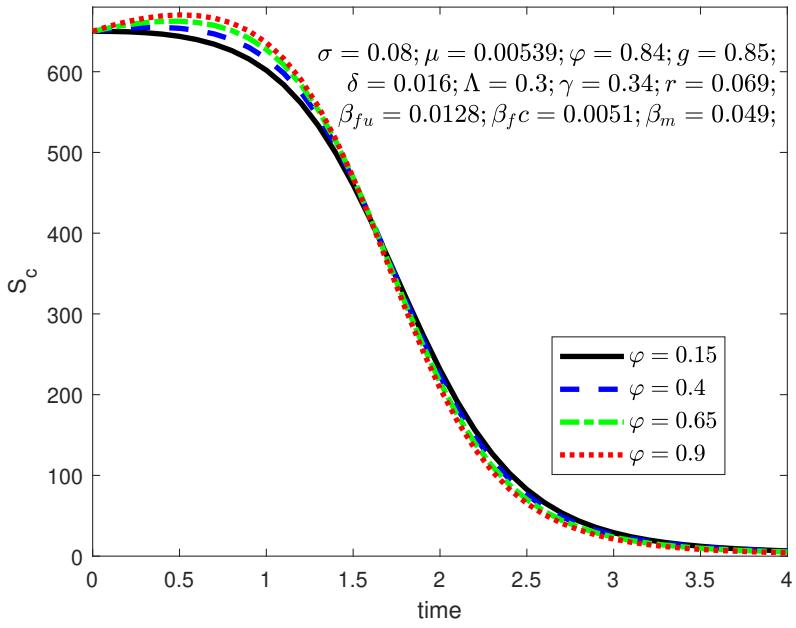

Figure 11: Variation of the circumcised male susceptible population with φ

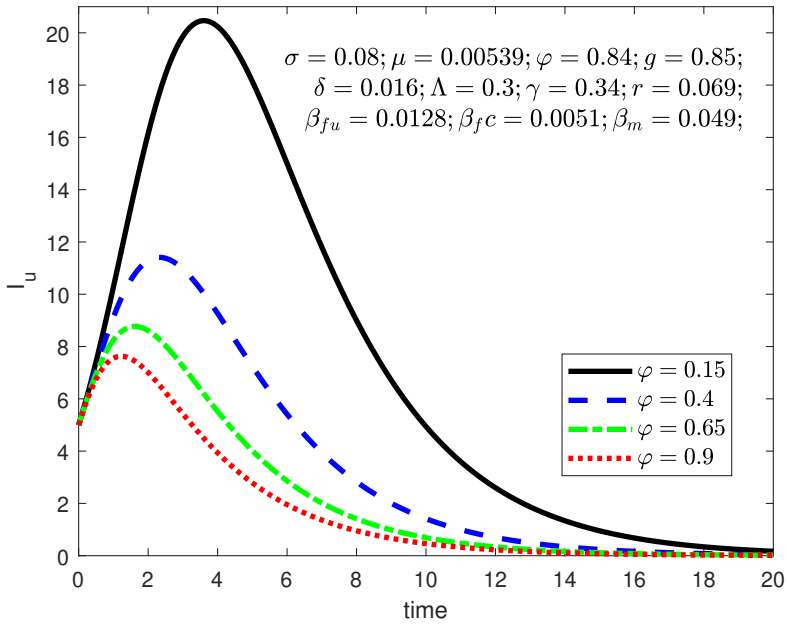

Figure 12: Variation of Uncircumcised infected male population with φ

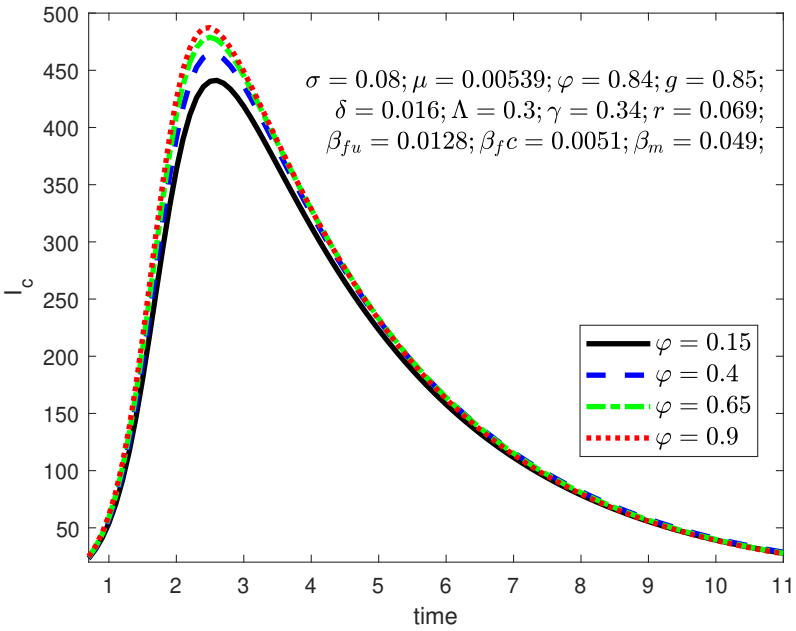

Figure 13: Variation of the circumcised infected male population with φ

4.2. Discussion of Results

4.2.1. Effects of HIV rate of transmission from females to circumcised and uncircumcised males

The effects are shown in figures (2 - 6). With any slight increase in the HIV rate of transmission from females to circumcised males, the AIDS population increases and the circumcised susceptible truckers' population decreases. This is due to the fact that the interactions between the truckers and the FSWs remain fixed while the rate of transmission to males increases. The infected circumcised population among the truckers rises rapidly between the first and the fourth year, but the infected circumcised population declines slowly after about four years due to the reduction in the total number of the susceptible population. The initial rise is enhanced the more the rate of transmission increases.

Figure (6) shows that increasing HIV rate of transmission from females to uncircumcised males causes an increase in the infected uncircumcised truckers' population. This is because the infected uncircumcised population responds in direct proportion to the rate of transmission from females to the uncircumcised infected population.

4.2.2. Effects of HIV rate of transmission from females to male

The effects are shown in figures (7 - 8). The infected FSWs population responds with high sensitivity to any little change in the rate of transmission from females to males. This is probably due to the fact that truckers are always on transit and therefore they have the tendency, once infected, to carry infection from one location to another. The infected female population increases rapidly in the first three years as the rate of transmission from female to male increases. The response is also highly directly proportional to the increase

in the rate of transmission β_m. Meanwhile, the susceptible female population reduces drastically as a response to the rise in the infected female population.

4.2.3. Effects of rate of circumcision

Figures (9 - 13) show the responses of different classes to the increase in the rate of circumcision. Increasing the rate of circumcision consequently increases migration from the susceptible uncircumcised population to the susceptible circumcised population. The consequence is an increase in the susceptible circumcised population while the susceptible uncircumcised population decreases. In addition, the infected uncircumcised population decreases with the increasing rate of circumcision since the susceptible uncircumcised population has dropped. Meanwhile, the infected circumcised population increases, but slowly. This shows that the rate of circumcision reduces the rate at which the truckers get infected.

5. Conclusion

In this paper, we formulated a model for HIV transmission dynamics between truckers and female sex workers. The model was solved and we obtained the reproduction numbers. It was noted that the disease-free equilibrium is stable when $R_{0u} < 1, R_{0c} < 1$ and $R_{0f} < 1$ while the endemic equilibrium point is stable when $R_{0u} > 1, R_{0c} > 1$ and $R_{0f} > 1$. The local stability of DFE and EEP was determined which was important in predicting whether the pandemic ends with time or not.

The model is solved using MATLAB and the dynamics of HIV between the truckers and FSWs are investigated. The analysis reveals that;

1. An increasing rate of female to (circumcised or uncircumcised) male transmission gives the disease the strength to persist in the population.
2. Increasing the rate of circumcision can reduce the spread of HIV among truckers and FSWs.

Hence, truckers should be sensitised to go for circumcision and reduce the interactions with the FSWs.

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