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Mathematical Modelling of the Effects Funding on HIV Dynamics Among Truckers and Female Sex Workers Along the Kenyan Northern Corridor Highway

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Abstract: The Southern and Eastern parts of Africa are the most hit by HIV/AIDS in the world and a huge financial commitment is required to control the spread of the disease. Of these countries, Kenya and South Africa have been able to increase prevention and treatment services due to their financial commitment to fighting the epidemic. However, studies have shown that most of the financial commitment comes from private donors and the private sectors are recently becoming reluctant to release funds. It is therefore important to ensure that the available funding is effectively utilised. Studies in 2018 show that infections occurred mostly among the key populations on the Kenyan Northern Corridor highway; such as sex workers and truckers. Moreso, transactional sex which involves cash transfer is the main mode of transmission of HIV/AIDS along the Northern corridor highway in Kenya. In this paper, we study the effect of funding on HIV transmission between truckers and female sex workers. A mathematical model with funding parameters is developed and analysed to determine the effects of funding on the HIV transmission dynamics between truckers and female sex workers. The reproduction number is obtained using the next-generation matrix and the conditions for the stability of the equilibrium points are established. The model is fed into the MATLAB ode45 solver and a numerical simulation is carried out. The results show that increasing circumcision funding reduces the rate of migration from the Susceptible class to the Infected class. Also, increasing treatment funding increases the Treatment class and reduces the overall number of AIDS-related.

Keywords: HIV/AIDS, Funding, Transmission Dynamics, Truckers, Female Sex Workers

1. Introduction

HIV/AIDS has continued to be a significant public health concern across the world. In 2020, an approximate of 37.7 million individuals, including 1.7 million children, were living with HIV. According to UNAIDS, the global HIV prevalence among adults was 0.7 percent, with around 16 percent of this population not knowing their HIV status. Since the first case of HIV was reported, 79.3 million individuals have been infected with 36.3 million deaths associated with AIDS. In the year 2020, about 680,000 deaths were associated with illnesses arising from AIDS. This represented an approximately 64% decrease since 2004 when 1.9 million were recorded, and 1.3 million deaths were recorded in 2010 due to AIDS-related illnesses [1]. The

majority of HIV-positive persons live in low- and middle-income nations. According to UNAIDS, East Africa and Southern Africa are the world's most HIV-affected areas, where there are approximately 20.6 million HIV-infected individuals and an estimated 670,000 infections in 2020 [1]. The African region accounts for more than two-thirds of the people living with HIV worldwide. In Kenya, the first HIV case was detected in 1984 and by the mid-90s, HIV has become a major cause of illnesses in the country. In 1996, 10.5% of Kenyans were living with HIV and the prevalence almost halved since then, standing at 5.9% by 2015. This resulted from the rapid scaling up of HIV treatment and care according to Jumba [2]. The HIV epidemic in Kenya is driven by sexual transmission and affects all sections of the population including children, young people, adults, women and men. In Kenya, Female sex workersC:\node\384 (FSWs) have the highest HIV prevalence. For example, in 1991, a study among truck drivers in Kenya found that 18% tested HIV-positive [3]; 61% of the sample reported having visited FSWs and only 32% had ever used condoms [4]. The separation of truckers from their wives or girlfriends is associated with truck driving and may lead the truckers to look for commercial sex services or have multiple non-commercial partnerships along their trucking route. About 29.3% of FSWs were living with HIV in 2013 [5], and 30% of FSWs were HIV-positive in 2013, compared with 5.4% of the population as a whole [6, 7]. In 2015, a study of FSWs in Nairobi found that approximately one-third were living with HIV according to Musyoki *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 [8, 9].

Ferguson and Morris [10] also found that a stopover was termed a hot spot depending on transactional sex taking place there. This was mainly determined by the truckers and the number of FSWs present at the location. They categorized the stopovers into three main categories which included: weighbridge and border crossing points were trucks delayed due to bureaucracy which included Mariakani and Mlolongo weighbridge stations, Malaba and Busia as border crossing points; “pure” truck shops which were exclusively serving the needs of truckers and other travellers with Salgaa, Machakos Junction, Mtito Andei, Maungu and Mai Mahiu serving as examples and finally were stopovers with diversified functions which included acting as border crossing points, administrative centres and market centres with Mlolongo and Busia falling under this category. They found that out of 7700 clients for the FSWs, 30% were truckers, 14.5% were drivers of other vehicles and 7.4% were police officers. Hence our research focuses on the truckers and FSWs as a key population to study the effect of funding on the HIV transmission dynamics of these vulnerable groups. The above statistics show that HIV is an issue of major concern, especially among the vulnerable groups which include truckers, female sex drivers, men who have sex with men and people who inject drugs.

Global HIV funding is still not adequate to respond to the rising infections in the general population and the vulnerable populations. Recently, resources for HIV responses in low- and middle-income countries have been decreasing since 2018. UNAIDS’ ambitious Fast-Track approach is committed to ending the global HIV epidemic public health threat by 2030. As a result, UNAIDS estimated an annual investment of 26.2 billion USD for HIV response in 2020, which would decrease steadily to 23.9 billion USD by 2030 according to UNAIDS [11]. Increases in resources for HIV responses in low- and middle-income countries stopped in 2017, with funding decreasing by 7% between 2017 and 2019 [12]. By the end of 2019, there was 18.6 billion USD available for the HIV response in low- and middle-income countries which was approximately 71% of the 2020 target. UNAIDS estimates that annual investments need to be increased to 29 billion USD by 2025 to get the AIDS response back on track in low- and middle-income countries where Kenya is part [1].

To deliver enhanced integrated health care for critical groups in Kenya, HIV financing remains a major barrier that must be tackled. Donor funding accounted for nearly 75% of Kenya's national HIV response in 2015 [13]. However, diminishing foreign donor money creates a difficulty for Kenya's HIV response because government expenditure nearly doubled between 2006 and 2012 (from USD 57.49 million to USD 153 million) [14]. Kenya has made significant progress in combating the HIV pandemic through pioneering HIV prevention, including the use of VMMC, self-testing, and PrEP. Also, the Kenya HIV Prevention Revolution Road Map is aiming to reduce 1.1 million new HIV infections and 761,000 AIDS-related deaths by 2030 [15]. This information indicates a very close relationship between the HIV pandemic and funding and hence, this study considers the effect of funding on HIV transmission dynamics between truckers and female sex workers. This study identifies the trend to expect at the different funding levels.

2. Methodology

2.1. Mathematical Formulation

A mathematical model is formulated to study the effect of funding on HIV/AIDS transmission dynamics between truckers and FSWs along the Northern Corridor highway 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 infected population is subjected to treatment. (2) Circumcision is done at a young stage but adult male truckers can be subjected to VMC. (3) The main clients of the female sex workers were truckers. (4) Only the adult population is considered. (5) No births.

As shown in Figure 1, Γ is the recruitment rate into the

susceptible population, the proportion of susceptible circumcised males is g, and a proportion r of females are recruited into FSWs. Infected uncircumcised males, circumcised males and infected females access treatment at rates $\gamma_u, \gamma_c, \gamma_f$ respectively. Treated individuals progress to AIDS at the rate σ and the population have a natural mortality rate μ and disease-induced mortality rate δ. Males get circumcised at a rate ϕ. Transmission rates of HIV from females to uncircumcised males, from females to circumcised males, and from males to females are $\beta_{fc}, \beta_m, \beta_{fu}$ respectively. Infected uncircumcised males, circumcised males and infected females are funded for treatment at rates $\kappa_1, \kappa_2, \kappa_3$ and circumcision is funded at a rate κ.

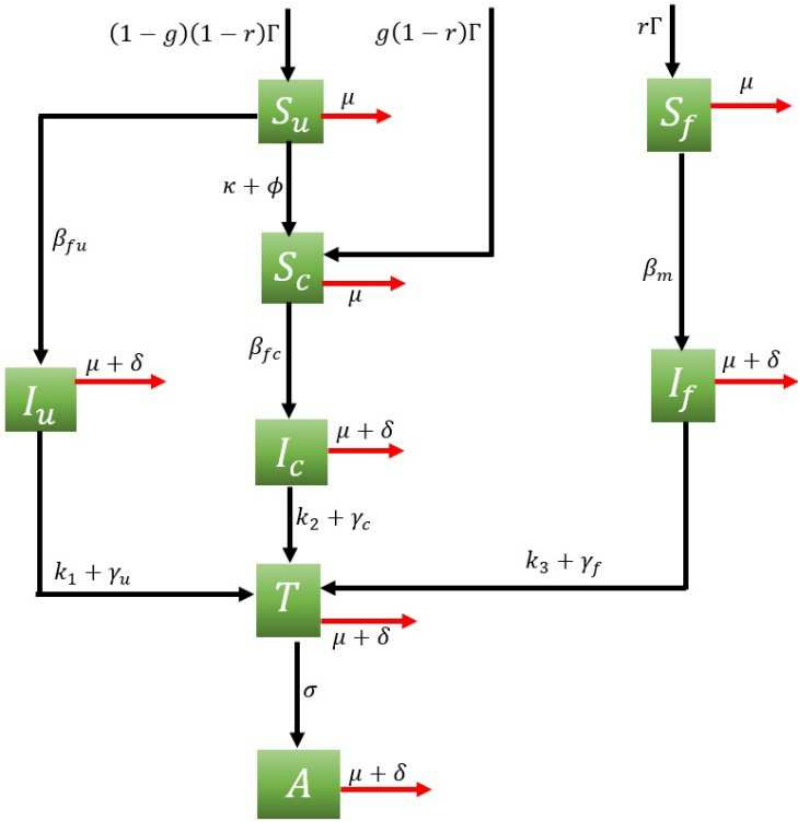

Figure 1. *Flow diagram for the mathematical model.*

The model is governed by the following ordinary differential equations;

$$\left.\begin{aligned}
\frac{dS_u}{dt} &= (1-g)(1-r)\Gamma - \beta_{fu}S_uI_u - (\kappa+\phi+\mu)S_u \\
\frac{dS_c}{dt} &= g(1-r)\Gamma + (\kappa+\phi)S_u - \beta_{fc}S_cI_c - \mu S_c \\
\frac{dS_f}{dt} &= r\Lambda - \beta_m S_f I_f - \mu S_f \\
\frac{dI_u}{dt} &= \beta_{fu}S_uI_u - [(\kappa_1+\gamma_u) - (\mu+\delta)]I_u \\
\frac{dI_c}{dt} &= \beta_{fc}S_cI_c - [(\kappa_2+\gamma_c) - (\mu+\delta)]I_c \\
\frac{dI_f}{dt} &= \beta_m S_f I_f - \left[(\kappa_3+\gamma_f) - (\mu+\delta)\right]I_f \\
\frac{dT}{dt} &= (\kappa_1+\gamma_u)I_u + (\kappa_2+\gamma_c)I_c + (\kappa_3+\gamma_f)I_f - ((\mu+\delta+\sigma)T \\
\frac{dA}{dt} &= \sigma T - (\mu+\delta)A
\end{aligned}\right\} \quad (1)$$

Where all parameters lie between 0 and 1 and $0 < \kappa + \phi + \beta_{fu} < 1, 0 < \beta_{fc} + \mu < 1, 0 < \beta_m + \mu < 1,$

$$0 < \kappa_1 + \gamma_u + \mu + \delta < 1, 0 < \kappa_2 + \gamma_c + \mu + \delta < 1, 0 < \kappa_3 + \gamma_f + \mu + \delta < 1, 0 < \sigma + \mu + \delta < 1,$$

2.2. The Equilibrium Points

The Disease-Free Equilibrium (DFE) Point is obtained by setting $I_u = I_c = I_f = T = A = 0$ in the system (1) to get

$$S_u = \frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, S_c = \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}, S_f = \frac{r\Gamma}{\mu}$$

Therefore, the DFE is given by

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

Endemic equilibrium points EEP are points at which the disease persists within the population. For this system, let $E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$, then the EEP is

$$S_{u2}^* = \frac{\kappa_1+\gamma_u+\kappa_5}{\beta_{fu}}, S_{c2}^* = \frac{\kappa_2+\gamma_c+\kappa_5}{\beta_{fc}}, S_{f1}^* = \frac{\kappa_3+\gamma_f+\kappa_5}{\beta_m},$$

$$I_{u2}^* = \frac{\beta_{fu}(1-g)(1-r)\Gamma-(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\kappa_5)}{\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)}, I_{f2}^* = \frac{\beta_m r\Gamma-\mu(\kappa_3+\gamma_f+\kappa_5)}{\beta_m(\kappa_3+\gamma_f+\kappa_5)},$$

$$I_{c2}^* = \frac{\beta_{fc}(1-r)\Gamma(g\mu+\kappa+\phi)-\mu(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)},$$

$$T_2^* = \frac{(\kappa_1+\gamma_u)\beta_{fu}(1-g)(1-r)\Gamma-(\kappa_1+\gamma_u)(\kappa_1+\gamma_u+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)} + \frac{(\kappa_2+\gamma_c)\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma-\mu(\kappa_2+\gamma_c)(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)} + \frac{(\kappa_3+\gamma_f)\beta_m r\Gamma-\mu(\kappa_3+\gamma_f)(\kappa_3+\gamma_f+\kappa_5)}{\kappa_4\beta_m(\kappa_3+\gamma_f+\kappa_5)},$$

$$\mathrm{A}_2^* = \frac{\sigma}{\kappa_4\kappa_5}\left[\frac{(\kappa_1+\gamma_u)\beta_{fu}(1-g)(1-r)\Gamma-(\kappa_1+\gamma_u)(\kappa_1+\gamma_u+\kappa_5)(\kappa+\phi+\mu)}{\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)} + \frac{(\kappa_2+\gamma_c)\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma-\mu(\kappa_2+\gamma_c)(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)} + \frac{(\kappa_3+\gamma_f)\beta_m r\Gamma-\mu(\kappa_3+\gamma_f)(\kappa_3+\gamma_f+\kappa_5)}{\beta_m(\kappa_3+\gamma_f+\kappa_5)}\right].$$

2.3. The Reproduction Number

The reproduction number R_0 is the number of secondary infections that an infectious 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 occur and the rate of transfer of individuals out of compartments. The R_0 was obtained from the spectral radius of the $F_0V_0^{-1}$ matrix. From system (1) then;

$$\mathcal{F} = \begin{pmatrix} \beta_{fu}S_uI_u \\ \beta_{fc}S_cI_c \\ \beta_mS_fI_f \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} (\kappa_1+\gamma_u+\mu+\delta)I_u \\ (\kappa_2+\gamma_c+\mu+\delta)I_c \\ (\kappa_3+\gamma_f+\mu+\delta)I_f \\ -(\kappa_1+\gamma_u)I_u-(\kappa_2+\gamma_c)I_c-(\kappa_3+\gamma_f)I_f+(\mu+\delta+\sigma)T \\ -\sigma T+(\mu+\delta)A \end{pmatrix}$$

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

$$F = \begin{pmatrix} \beta_{fu}S_u^0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc}S_c^0 & 0 & 0 & 0 \\ 0 & 0 & \beta_mS_f^0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} \kappa_1+\gamma_u+\mu+\delta & 0 & 0 & 0 & 0 \\ 0 & \kappa_2+\gamma_c+\mu+\delta & 0 & 0 & 0 \\ 0 & 0 & \kappa_3+\gamma_f+\mu+\delta & 0 & 0 \\ -\kappa_1-\gamma_u & -\kappa_2-\gamma_c & -\kappa_3-\gamma_f & \mu+\delta+\sigma & 0 \\ 0 & 0 & 0 & -\sigma & \mu+\delta \end{pmatrix}$$

Hence for system (1) the next generation matrix [16, 17, 24] is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{fu}S_u^0}{\kappa_1+\gamma_u+\mu+\delta} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc}S_c^0}{\kappa_2+\gamma_c+\mu+\delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_mS_f^0}{\kappa_3+\gamma_f+\mu+\delta} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

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

$$\lambda^2\left(-\lambda+\frac{\beta_m r\Gamma}{\mu(\kappa_3+\gamma_f+\mu+\delta)}\right)\left(-\lambda+\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)}\right)\left(-\lambda+\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)}\right)=0$$

The eigenvalues are

$$\lambda_1=\lambda_2=0,\lambda_3=\frac{\beta_m r\Gamma}{\mu(\kappa_3+\gamma_f+\mu+\delta)},\lambda_4=\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)},\lambda_5=\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)},$$

λ_3,λ_4 and λ_5 gives the basic reproduction numbers for the system (1) as;

$$R_{of}=\frac{\beta_m r\Gamma}{\mu(\kappa_3+\gamma_f+\mu+\delta)},R_{oc}=\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)},\text{and } R_{ou}=\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)}.$$

Theorem 1: Let $R_0=\{R_{of},R_{oc},R_{ou}\}$, then the disease-free equilibrium E^0 of the system (1) 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

$$J=\begin{pmatrix}
-\beta_{fu}I_u-(\kappa+\phi+\mu) & 0 & 0 & -\beta_{fu}S_u & 0 & 0 & 0 & 0\\
\kappa+\phi & -\beta_{fc}I_c-\mu & 0 & 0 & -\beta_{fc}S_c & 0 & 0 & 0\\
0 & 0 & -\beta_m I_f-\mu & 0 & 0 & -\beta_m S_f & 0 & 0\\
\beta_{fu}I_u & 0 & 0 & \beta_{fu}S_u-(k_1+\gamma_u+\kappa_5) & 0 & 0 & 0 & 0\\
0 & \beta_{fc}I_c & 0 & 0 & \beta_{fc}S_c-(k_2+\gamma_c+\kappa_5) & 0 & 0 & 0\\
0 & 0 & \beta_m I_f & 0 & 0 & \beta_m S_f-(k_3+\gamma_f+\kappa_5) & 0 & 0\\
0 & 0 & 0 & k_1+\gamma_u & k_2+\gamma_c & k_3+\gamma_f & -k_4 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & \sigma & -k_5
\end{pmatrix}$$

where $k_4=\mu+\delta+\sigma,k_5=\mu+\delta$. The first two eigenvalues are $\lambda_1=-(\mu+\delta)$, and $\lambda_2=-(\mu+\delta+\sigma)$. λ_3 and λ_4 are obtained from;

$$\lambda^2-\left(\left(\beta_{fu}S_u-(k_1+\gamma_u+\kappa_5)\right)-\left(\beta_{fu}I_u-(\kappa+\phi+\mu)\right)\right)\lambda+\beta_{fu}\left(I_u(k_1+\gamma_u+\kappa_5)-S_u(\kappa+\phi+\mu)\right)+(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)=0,$$

and therefore λ_3 and λ_4 are negative if

$$\frac{\beta_{fu}(S_u-I_u)}{\mu+k_1+\gamma_u+\kappa_5}<1,\text{and }\frac{\beta_{fu}\left(S_u(\kappa+\phi+\mu)-I_u(k_1+\gamma_u+\kappa_5)\right)}{(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)}<1.$$

Similarly, λ_5 and λ_6 are obtained from

$$\lambda^2-\left(\left(\beta_{fc}S_c-(k_2+\gamma_c+\kappa_5)\right)-\left(\beta_{fc}I_c+\mu\right)\right)\lambda+\beta_{fc}(I_c(k_2+\gamma_c+\kappa_5)-\mu S_c)+\mu(k_2+\gamma_c+\kappa_5)=0,$$

and therefore λ_5 and λ_6 are negative if

$$\frac{\beta_{fc}(S_c-I_c)}{\mu+k_2+\gamma_c+\kappa_5}<1,\text{and }\frac{\beta_{fc}\left(\mu S_c-I_c\left(k_c+\gamma_c+\kappa_5\right)\right)}{\mu(k_2+\gamma_c+\kappa_5)}<1.$$

In a similar manner λ_7 and λ_8 are obtained from

$$\lambda^2-\left(\beta_m(S_f-I_f)-(\mu+k_3+\gamma_f+\kappa_5)\right)\lambda+\beta_m\left(I_f\left(k_3+\gamma_f+\kappa_5\right)-\mu S_f\right)+\mu\left(k_3+\gamma_f+\kappa_5\right)=0$$

and λ_7 and λ_8 are negative if

$$\frac{\beta_m(S_f-I_f)}{\mu+k_3+\gamma_f+\kappa_5}<1,\text{and }\frac{\beta_m\left(\mu S_f-I_f\left(k_3+\gamma_f+\kappa_5\right)\right)}{\mu\left(k_3+\gamma_f+\kappa_5\right)}<1.$$

Case 1: At the disease-free-equilibrium *DFE*

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

The requirements for stability therefore are;

1) λ_3,λ_4, are negative if

$$\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(2\mu+k_1+\gamma_u+\delta)(\kappa+\phi+\mu)}<1,\text{and }\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)}<1$$

and thus

$$\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(2\mu+k_1+\gamma_u+\delta)(\kappa+\phi+\mu)} < \frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)} < 1 \Rightarrow R_{ou} < 1.$$

2) λ_5, λ_6 are negative if

$$\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(2\mu+k_2+\gamma_c+\delta)} < 1, \frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(k_2+\gamma_c+\mu+\delta)} < 1$$

and thus

$$\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(2\mu+k_2+\gamma_c+\delta)} < \frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(k_2+\gamma_c+\mu+\delta)} < 1 \Rightarrow R_{oc} < 1$$

3) λ_3, λ_4, are negative if

$$\frac{\beta_m r\Gamma}{\mu(2\mu+k_3+\gamma_f+\delta)} < 1, \text{and } \frac{\beta_m r\Gamma}{\mu(k_3+\gamma_f+\mu+\delta)} < 1$$

and thus

$$\frac{\beta_m r\Gamma}{\mu(2\mu+k_3+\gamma_f+\delta)} < \frac{\beta_m r\Gamma}{\mu(k_3+\gamma_f+\mu+\delta)} < 1 \Rightarrow R_{of} < 1.$$

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

Case 2: At the endemic equilibrium point *EEP*, E^*

$$E^* = \left(S_{u2}^*, S_{c2}^*, S_{f2}^*, I_{u2}^*, I_{c2}^*, I_{f2}^*, \mathrm{T}_2^*, \mathrm{A}_2^*\right)$$

The first two eigenvalues are $\lambda_1 = -(\mu+\delta)$, and $\lambda_2 = -(\mu+\delta+\sigma)$ and the other eigenvalues $\lambda_3 \ldots \lambda_8$ are negative under the following conditions:

1) λ_3 and λ_4 are negative if

$$\frac{\beta_{fu}(S_{u2}^* - I_{u2}^*)}{\mu+k_1+\gamma_u+\kappa_5} < 1, \text{and } \frac{\beta_{fu}\left(S_{u2}^*(\kappa+\phi+\mu) - I_{u2}^*(k_1+\gamma_u+\kappa_5)\right)}{(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)} < 1$$

which, on substitution of S_{u2}^* and I_{u2}^*, become

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

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

2) λ_5 and λ_6 are negative if

$$\frac{\beta_{fc}(S_{c2}^* - I_{c2}^*)}{\mu+k_2+\gamma_c+\kappa_5} < 1, \text{and } \frac{\beta_{fc}\left(\mu S_{c2}^* - I_{c2}^*(k_c+\gamma_c+\kappa_5)\right)}{\mu(k_2+\gamma_c+\kappa_5)} < 1.$$

which, on substitution of S_{c2}^* and I_{c2}^*, become

$$\frac{(r-1)g\beta_{fu}\Gamma - (\kappa+\phi)(\mu+\delta+\gamma)}{(\mu+\delta+\gamma)(2\mu+\delta+\gamma)\beta_{fu}} < 1, \text{and } \frac{\beta_{fc}\left((1-r)g\beta_{fu}\Gamma + (\kappa+\phi)(\mu+\gamma+\delta)\right)}{\mu\beta_{fu}(\mu+\gamma+\delta)} > 1.$$

The first condition is true since $r - 1 < 0$ implies that $(r-1)g\beta_{fu}\Gamma - \phi(\mu+\delta+\gamma) < 0$. The second condition becomes

$$\frac{\beta_{fc}\left((1-r)g\beta_{fu}\Gamma + (\kappa+\phi)(\mu+\gamma+\delta)\right)}{\mu\beta_{fu}(\mu+\gamma+\delta)(\mu+\kappa+\phi)} > \frac{1}{(\mu+\kappa+\phi)}$$

$$\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\mu+\gamma+\delta)(\mu+\kappa+\phi)}\frac{g}{(g\mu+\kappa+\phi)} + \frac{\beta_{fc}(\kappa+\phi)}{\mu\beta_{fu}(\mu+\kappa+\phi)} > \frac{1}{(\mu+\kappa+\phi)}$$

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

3) λ_7 and λ_8 are negative if

$$\frac{\beta_m\left(S_{f2}^* - I_{f2}^*\right)}{\mu+k_3+\gamma_f+\kappa_5} < 1, \text{and } \frac{\beta_m\left(\mu S_{f2}^* - I_{f2}^*(k_3+\gamma_f+\kappa_5)\right)}{\mu(k_3+\gamma_f+\kappa_5)} < 1.$$

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

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

The first condition is already satisfied since $\beta_m, r, \Gamma, \mu, \delta, \gamma > 0$ and hence, λ_3 and λ_4 are negative if $R_{of} > 1$. Hence, E^* is locally asymptotically stable if $R_0 > 1$. ■

3. Analysis and Discussion of Results

The system (1) is solved numerically using MATLAB ode45 solver and the effect of funding parameters κ, k_1, k_2 and k_3 are investigated on HIV/AIDS dynamics among truckers/FSWs in the Northern corridor highway in Kenya (see [18] for other methods of solution). The parameters used for simulation are shown in Table 1 and are chosen to suit the Truckers/FSWs along the Northern corridor highway in Kenya.

Table 1. *Parameter values suitable to model Trucker-FSWs HIV/AIDS dynamics.*

Parameter	g	r	Γ	γ	σ	μ	δ	ϕ	β_{fu}	β_{fc}	β_m
Value	0.8500	0.0690	0.3000	0.3400	0.0800	0.0539	0.0160	0.8400	0.0128	0.0051	0.0490
Source	[19]	[20]	Estimated	[21]	Estimated	[21]	[1]	[22]	[23]	[25]	[25]

3.1. Analysis of Results

Effects of funding for male circumcision κ are shown in Figures 2–4. The value $\kappa = 0$ represents a total removal of circumcision funding while the maximum rate of circumcision funding is 0.14. As circumcision funding increases from zero to maximum, the class of infected circumcised males increases slightly (Figure 2), the class of infected uncircumcised males decreases (Figure 3) and the class of susceptible uncircumcised males decreases (Figure 4).

Effects of funding for treatment for the infected uncircumcised males are shown in Figures (5–6). $k_1 = 0$ represents the absence of treatment funding for the infected uncircumcised males and $k_1 = 0.6$ represents the maximum funding possible for the infected uncircumcised males. As funding increases for the treatment of infected uncircumcised males, the class of infected uncircumcised males decreases (Figure 5) and the class of susceptible uncircumcised males increases (Figure 6).

Effects of funding of treatment for circumcised males are shown in Figures 7–10. $k_2 = 0$ represents the absence of treatment funding for circumcised males and $k_1 = 0.6$ represents the maximum funding possible for circumcised males. As funding increases for the treatment of infected circumcised males, the AIDS population increases (Figure 7), the Treated class increases (Figure 8), the class of infected circumcised males decreases (Figure 9) and the class of susceptible circumcised males increases (Figure 10).

Effects of treatment funding for FSWs are shown in Figures (11–14). $k_3 = 0$ represents the absence of treatment funding for FSWs and $k_3 = 0.6$ represents the maximum funding possible for FSWs. As funding increases for the treatment of FSWs, the AIDS population increases (Figure 11), the Treated class increases (Figure 12), the class of infected FSWs decreases (Figure 13) and the class of susceptible FSWs increases (Figure 14).

Effects of increasing overall funding are shown in Figures (15–22). In this case, overall funding refers to combined funding of male truckers' circumcision and funding of treatment of all classes of infected individuals. Increasing overall funding leads to a decrease in susceptible uncircumcised male truckers (shown in Figure 15), an increase in susceptible circumcised male truckers (shown in Figure 16), an increase in susceptible FSWs (shown in Figure 17), a decrease in infected uncircumcised male truckers (shown in Figure 18), an increase in infected circumcised male truckers (shown in Figure 19), an increase in infected FSWs (shown in Figure 20), a decrease in Treated (shown in Figure 21), and an increase in the AIDS population (shown in Figure 22).

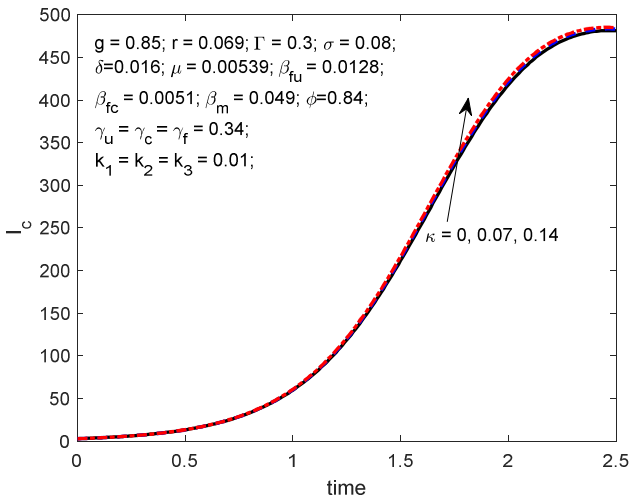

Figure 2. *Variation of Infected Circumcised Male with circumcision funding parameter.*

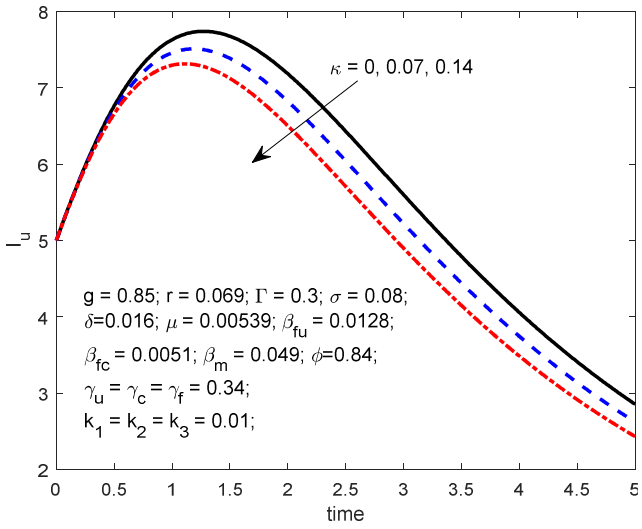

Figure 3. *Variation of Infected uncircumcised Male with circumcision funding parameter.*

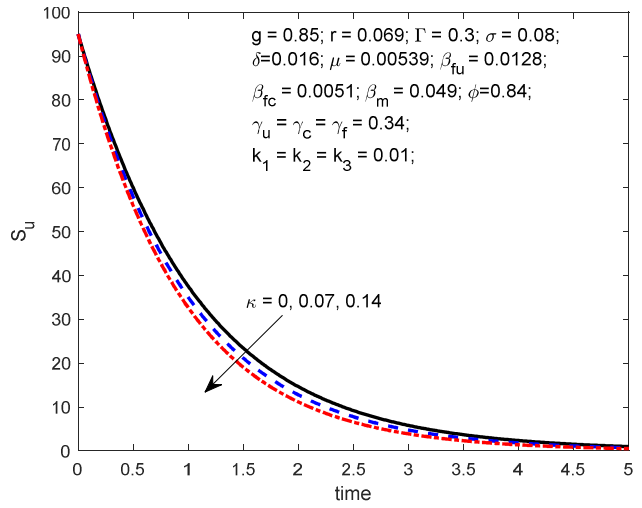

Figure 4. *Susceptible uncircumcised Male with circumcision funding parameter.*

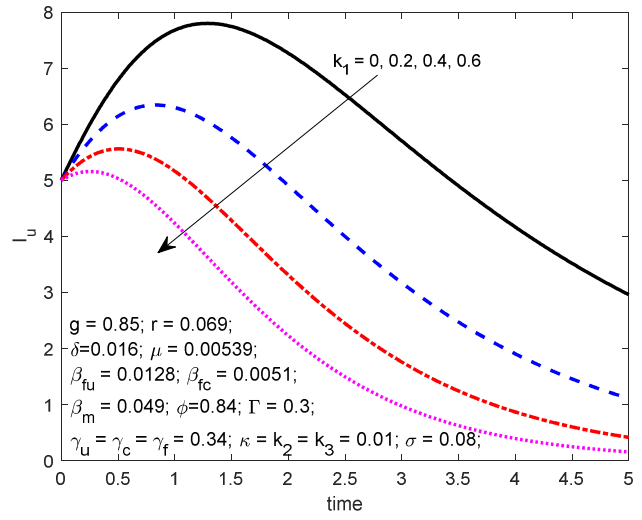

Figure 5. *Infected uncircumcised male with treatment funding for the uncircumcised male.*

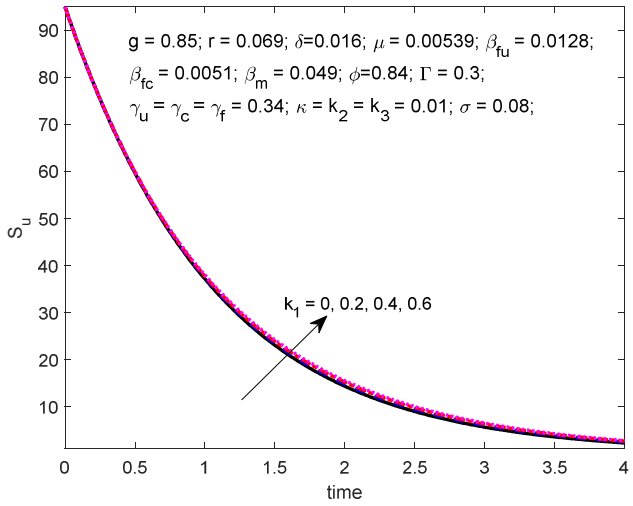

Figure 6. *Variation of Susceptible uncircumcised males with treatment funding for the uncircumcised male.*

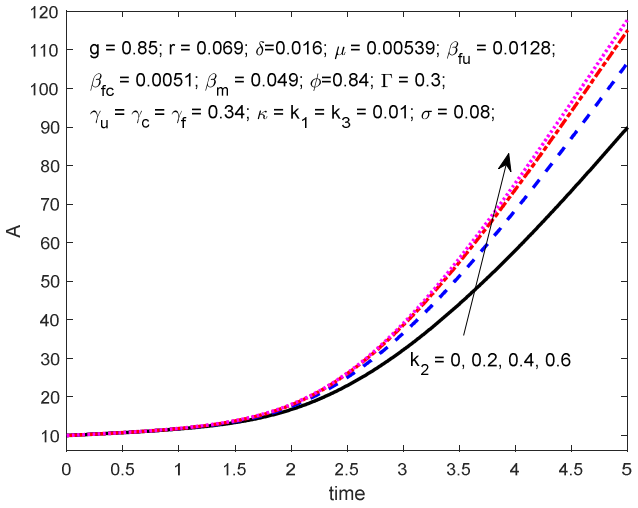

Figure 7. *Variation of the AIDS population with treatment funding for the circumcised male.*

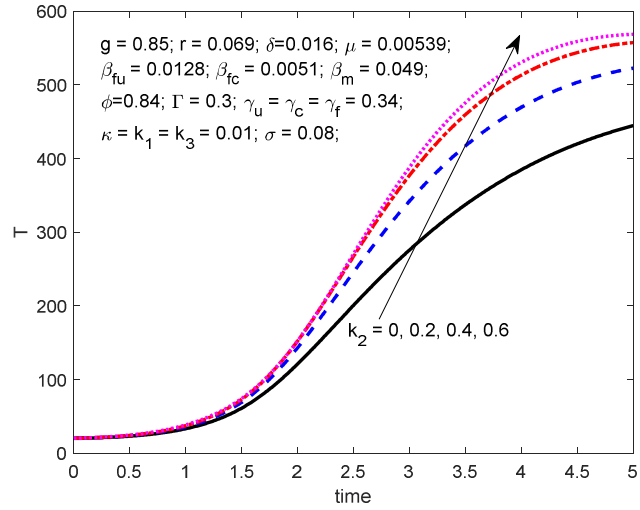

Figure 8. *Variation of Treatment class with treatment funding for the circumcised male.*

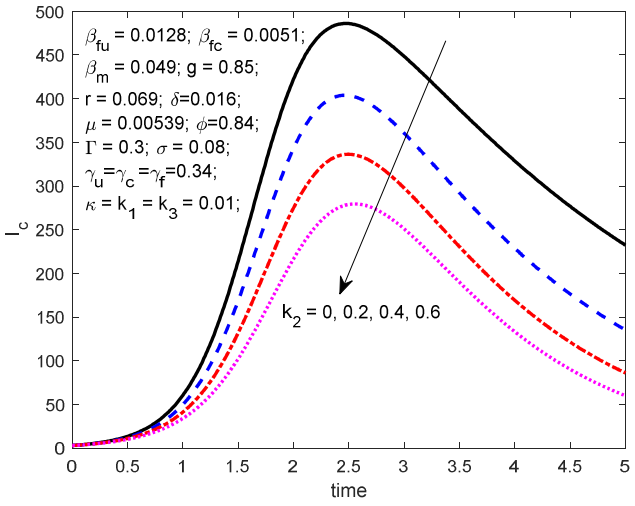

Figure 9. *Variation of Infected circumcised male with treatment funding for the circumcised male.*

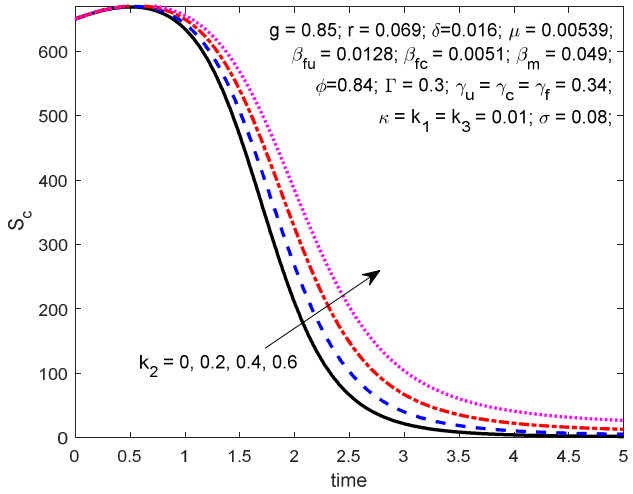

Figure 10. *Variation of Susceptible circumcised male with treatment funding for the circumcised male.*

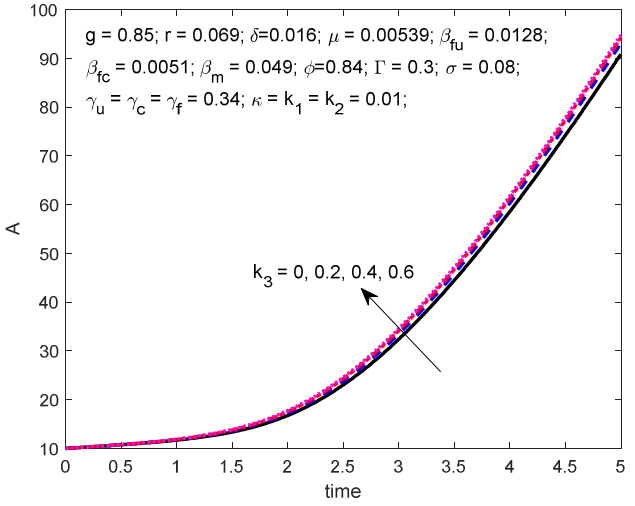

Figure 11. *Variation of the AIDS population with treatment funding for FSWs.*

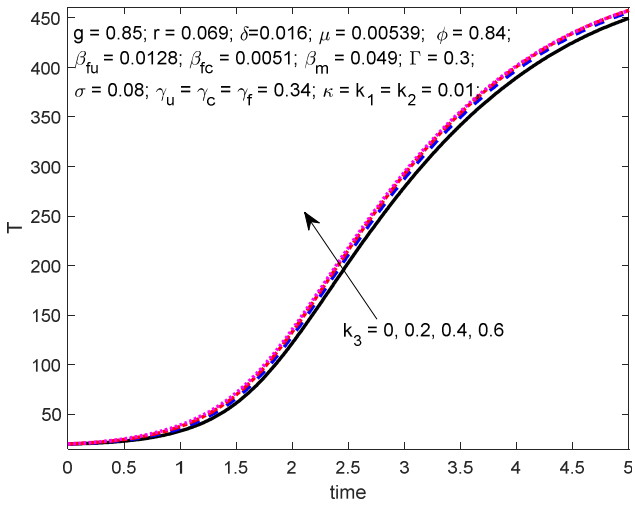

Figure 12. *Variation of Treatment class with treatment funding for FSWs.*

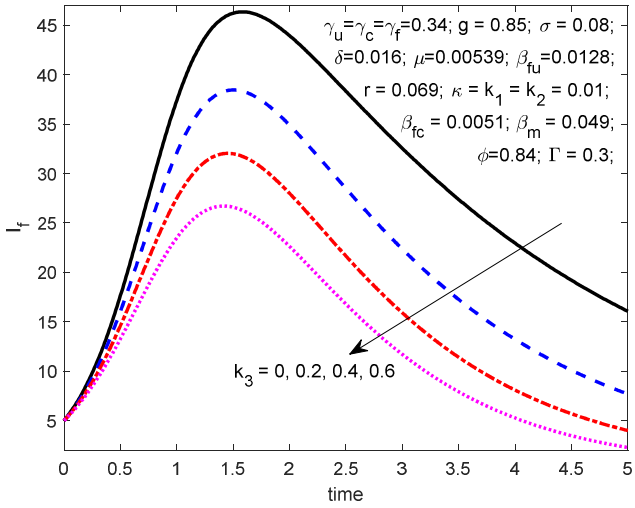

Figure 13. *Variation of Infected FSWs with treatment funding for FSWs.*

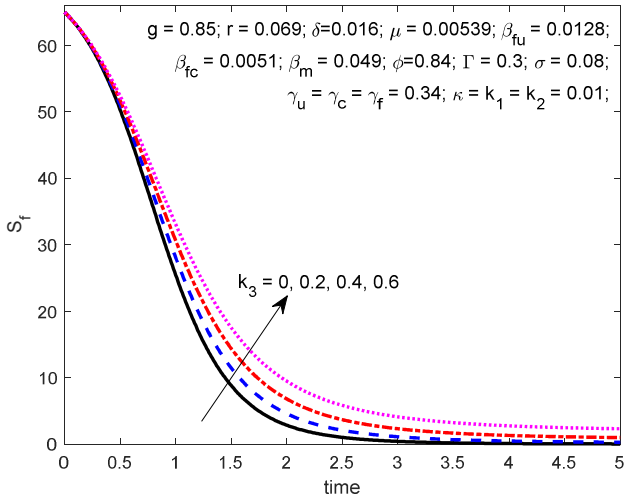

Figure 14. *Variation of Susceptible FSWs with treatment funding for FSWs.*

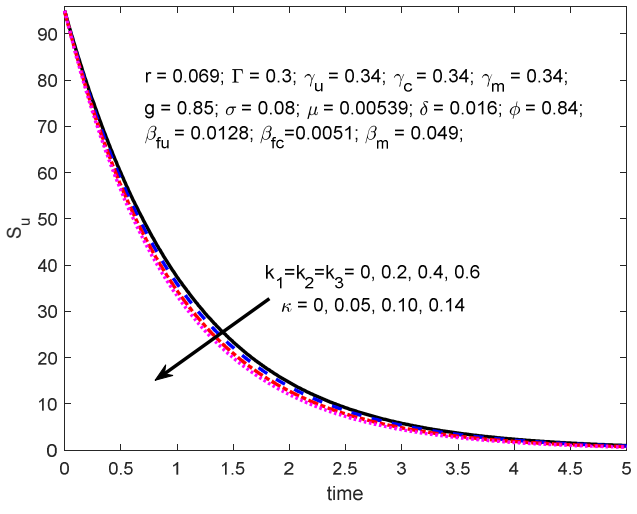

Figure 15. *Variation of Susceptible uncircumcised male with funding.*

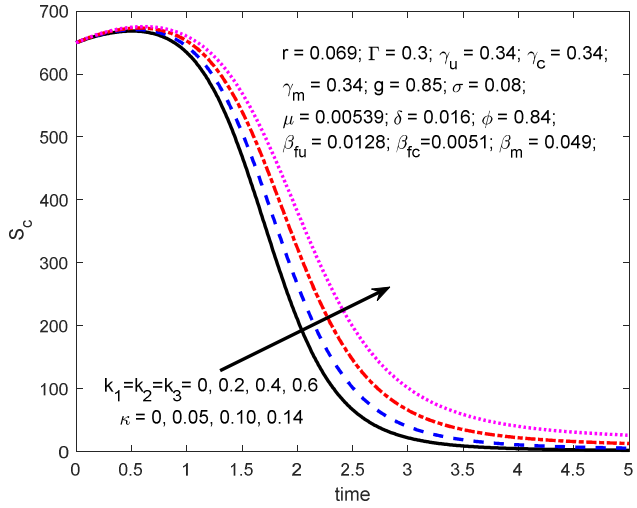

Figure 16. *Variation of Susceptible circumcised male with funding.*

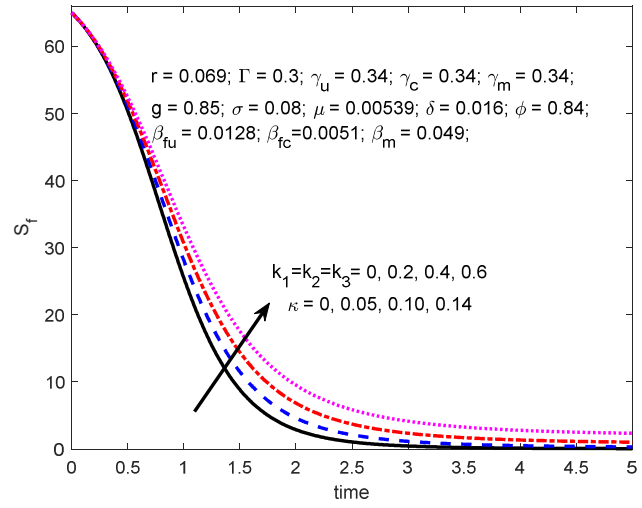

Figure 17. *Variation of infected FSWs with funding.*

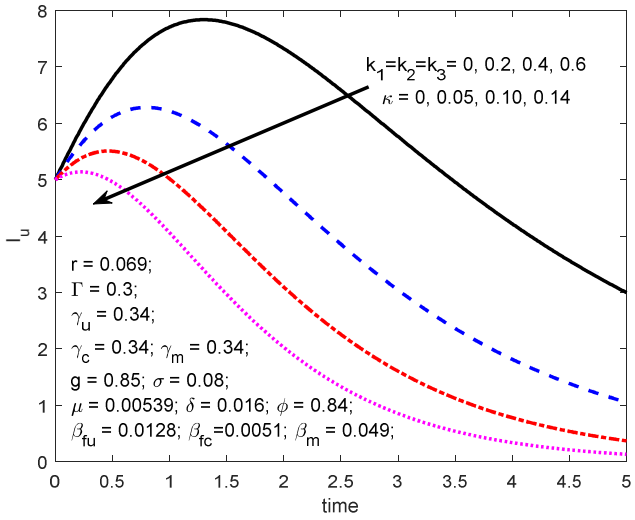

Figure 18. *Variation of infected uncircumcised male with funding.*

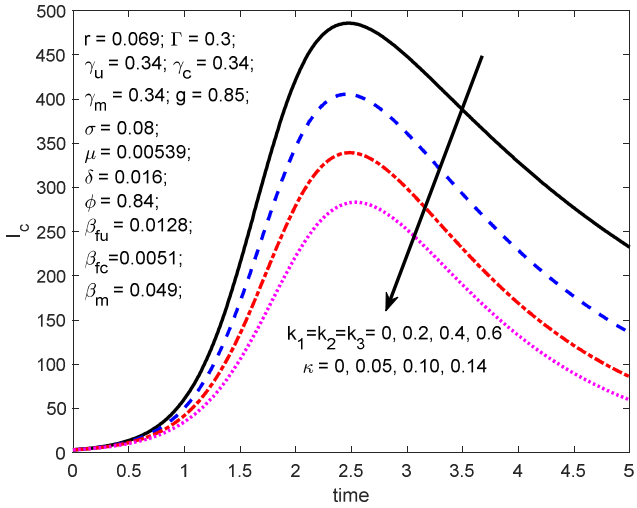

Figure 19. *Variation of infected circumcised males with funding.*

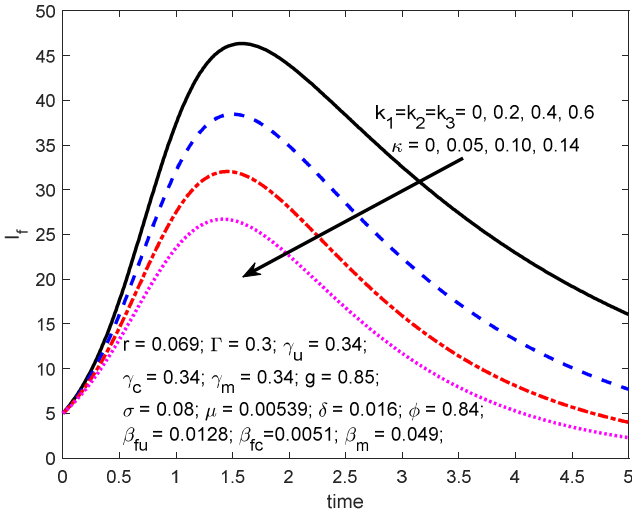

Figure 20. *Variation of infected FSWs with funding.*

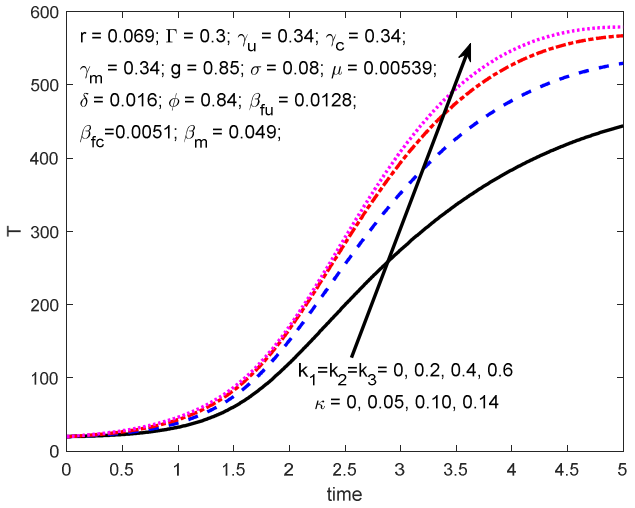

Figure 21. *Variation of Treated class with funding.*

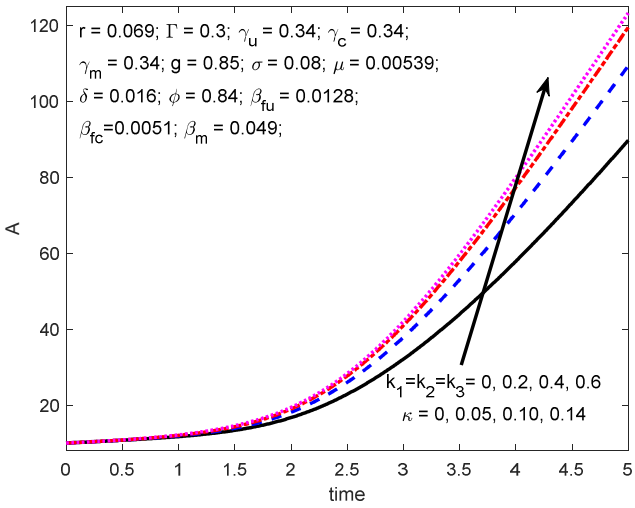

Figure 22. *Variation of the AIDS population with funding.*

3.2. Discussion of Results

The effects of circumcision funding are discussed here. Funding for male truckers' circumcision increases the chances of migration from the uncircumcised class to the circumcised class. This means an increase in the number of circumcised male truckers susceptible to contracting HIV and a reduction in the number of uncircumcised male truckers susceptible to contracting HIV. Meanwhile, a circumcised male trucker is less likely to contract HIV than an uncircumcised male trucker. The reduction in the uncircumcised class, therefore, means a reduction in the chance for any trucker to migrate to the infected class. Hence, the overall number of male truckers that are recruited to the infected class will decrease overall.

Investigating the effects of funding of treatment for the infected uncircumcised males shows that increasing funding leads to a decrease in the class of infected uncircumcised males while the class of susceptible uncircumcised males increases. Increasing funding of treatment for the infected uncircumcised males means more infected uncircumcised male truckers can assess treatment more readily and thereby many migrate to the Treatment class.

Increasing funding of treatment for the infected circumcised males means more infected circumcised male truckers can assess treatment more readily and thereby many migrate to the Treatment class. An increase in funding for treating infected circumcised male truckers brings about an increase in the number of Treated individuals.

Funding treatment for FSWs increases the number of infected FSWs that get treatment and thereby reduces the number of individuals entering the AIDS class.

In general, if circumcision is funded at a higher rate and all infected classes are funded for treatment, then the overall funding improves. Studying the effects of the improved overall funding on the entire truckers/FSWs population. Due to improved circumcision, more percentage of the susceptible population enters the susceptible circumcised male. Due to the reduced chance of infection for the susceptible circumcised male truckers, it means the total number of infected individuals will drop. The Treated class will also increase because of the increased funding and thereby reduced the number of new migrations into the AIDS population.

4. Conclusion

This study formulates a mathematical model for the dynamics of HIV/AIDS among Kenyan truckers/FSWs on the Northern corridor highway. Funding for circumcision and funding for treatment of the different infected classes are incorporated into the model. The resulting model is solved numerically using MATLAB ode45 solver. The results show that;

1) increasing funding for circumcision reduces the rate of migration from the Susceptible class to the Infected class.
2) Increasing funding for treatment of any class increases the Treatment class and reduces the overall number of AIDS-related.

In conclusion, funding circumcision (such as subsidizing the cost of circumcision, providing quality free post-circumcision services, and/or setting up circumcision clinics along the Kenya Northern highway corridors) can reduce the rate at which truckers contract HIV/AIDS in Kenya. Also, increasing funding for treatment of the infected classes (such as providing free or subsidized anti-retroviral drugs, providing free counselling to infected individuals, etc.) can help ameliorate the overall number of AIDS-related death.

Declarations

Competing Interests

Authors declare no conflicts of interests.

Authors' Contributions

Study conception: Kimulu, Mutuku.
Design: Kimulu, Mwalili, Maloza.

Analysis: Oke, Mwalili.

Interpretation of results: Kimulu, Oke.

Draft manuscript: Kimulu, Mutuku.

Preparation: Kimulu, Oke, Malonza.

All authors reviewed the results and approved the final version of the manuscript.

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