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HIV/AIDS Transmission Dynamics: Modelling the Roles of Long Distance Truck Drivers

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Abstract

A mathematical model to investigate the roles of long distance truck drivers on HIV transmission dynamics was formulated and analyzed to establish the existence of disease free and endemic equilibrium points. The results show that, the disease free equilibrium point is asymptotically stable when the basic reproduction number is less than unity and unstable otherwise. Furthermore, comprehensive analyses on the two steady states (infection free and endemic) have shown that they are both globally and asymptotically stable. Sensitivity analysis is performed on the reproduction number in order to establish the relative importance of parameters, and it shows that the truck drivers have the potential of increasing the rates of transmission of HIV infections, which concurs with the numerical simulation results.

Keywords: Truck drivers; stability; equilibrium; reproduction number; sensitivity.

Introduction

Human mobility is often associated with dissemination of emerging and re-emerging infectious diseases. For example, SARS epidemic in 2003 and H1N1 were greatly disseminated by air travels at the worldwide scale in 2009; see Findlater and Bogoch (2018) for details. Also, the spread of human immunodeficiency virus (HIV) is associated with human mobility and high incidences of HIV infections were observed in areas with reportedly high migration flows and which are situated along major transport corridors. Long distance truck drivers (who from now are referred to as truck drivers) are of particular concern to HIV prevention and control programmes because they travel frequently, often to areas with high levels of HIV incidences. For instance, surveys carried out in Kenya and Uganda along highways from 1989 to 2005 showed high rates of HIV infections in the truck driving populations (Carswell et al. 1989). The patterns of sexual behaviours increase the likelihood that truck drivers experiencing primary HIV infections transmit the virus to other people and spouses on coming back home (Hudson 1996).

Some circumstances that seem to increase the risks of HIV infections among truck drivers are the long separation from spouses and family, multiple partners, delays at border crossings and lack of access to health services (Babinard and Gause 2009). The need for entertainment and female companionship makes them very likely to use the services of commercial sex workers in stop-over towns on major transportation routes. These truck stop towns have developed an entire infrastructure of networks and services meeting the business and recreation needs of truck drivers, including gas stations, inspection points, lodges, bars and brothels and a high concentration of commercial sex workers. Also, according to IOM (2003), risk factors of migration are felt at four sites, namely where they are coming from, en-route, where they are going and upon return

to their sources. While in en-route, the drivers meet many people in desperate need for their companionship while paying with sexual favours. Moreover, being away from their wives they may need to satisfy their sexual needs and also overnight accommodation which is cheaper. Ultimately they will return to their families with HIV infections which they would have contracted along the way. Furthermore, unmarried truck drivers who are generally young and do not have any socially accepted steady sexual partners are more likely to engage in riskier behaviours, including commercial sex workers if exposed to the environmental factors associated with trucking industry, such as high mobility with anonymity, easy availability of female sex workers and other women (Pandey et al. 2012). Also, married men are believed to have more favourable attitudes toward obtaining, discussing and using condoms with non-marital partners possibly because they attempt to protect themselves and their wives by using condoms with non-regular sexual partners, rather than using it with their wives (Ford and Chamrathrithirong 2007). Condom use with wife or with intimate partners could send a strong signal of infidelity and thus both partners have a disincentive to insist on using condoms (Dude et al. 2009).

Much work has been done on HIV infections, for instance, Mushayabasa and Bhunu (2011) and Oduwole and Shehu (2013), modelled HIV dynamics by assuming sexual contact as a means of HIV transmission among immigrants, prisoners and prostitutes. However, little has been done to investigate the roles of truck drivers in the spread of HIV/AIDS. For example, Kribs-Zaleta et al. (2005) revealed that HIV/AIDS has great impacts on the transportation sector through the loss of truck drivers. This paper intends to develop a mathematical model to investigate how truck drivers are more at risks of contracting HIV infections than their counterpart non-truck drivers. In such a model, the population of potential truck drivers is divided into truck drivers (as an experimental group) and the non-truck drivers (as control group), and later the groups are compared to derive their contributions in the dynamics of HIV infections.

Materials and Methods

In this paper a flow chart (see Figure 1) has been used to describe the movements of individuals among epidemiological compartments depending on their disease status. The flow chart was then used to formulate a mathematical model for the roles of truck drivers on the transmission of HIV in the community of truck drivers.

Model formulation

The total population at any time t denoted by $N(t)$ is divided into the following mutually exclusive epidemiological classes: susceptible truck drivers ($S_\tau(t)$), susceptible non-truck drivers ($S_n(t)$), truck drivers infected with HIV ($I_\tau(t)$), non-truck drivers infected with HIV ($I_n(t)$), and full blown AIDS cases ($A(t)$). We assume that the number of truck drivers in the community is increased (recruitment) by the new truck driving aspirants at a rate Λ. All recruited individuals are assumed to be susceptible to the disease. A proportion η of recruited individuals are assumed to join the class of truck drivers S_τ, and the remaining proportion $(1-\eta)$ goes to the class of non-truck drivers class S_n. It is assumed that susceptible truck drivers may quit driving job and join the non-truck drivers at a rate α_1 and the infected truck drivers may quit the job and join the class of infected non-truck drivers at a rate α_2. Here $\alpha_2 > \alpha_1$, since infected drivers are more likely to quit the job than healthy drivers. Susceptible truck drivers and non-truck drivers get infected with the disease from their female partners at the rates $\lambda_\tau = \frac{\beta(I_\tau + \varepsilon A)}{N}$ and $\lambda_n = \frac{\beta(I_n + \varepsilon A)}{N}$, respectively. The parameter β is the effective

contact rate (that is, contact that may result into HIV infection) and $\varepsilon > 1$ accounts for the relative infectiousness of truck drivers with AIDS symptoms in comparison to the corresponding infected truck drivers with no AIDS symptoms. It is assumed that infected non-truck drivers (those who were once truck drivers and have quit the job due to infections) transmit the disease at a reduced rate modified by a factor κ, where $0 < \kappa < 1$. In other words, HIV infected non-truck drivers transmit the infections at a slower rate in comparison to infected truck drivers because the former are less mobile than the latter. Thus, susceptible non-truck drivers acquire infections at a reduced rate $(1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]$, where $0 < \rho < 1$ is the factor for reducing the risk behaviour of HIV transmission per contact of contracting the disease. Infected truck drivers progress to AIDS at a rate of γ_τ, while infected non-truck drivers progress to AIDS at a reduced rate $\gamma_n < \gamma_\tau$ (that is, infected non-truck drivers progress to AIDS at slower rate in comparison to infected truck drivers). Individuals in all the classes suffer from natural deaths at a rate μ. Additionally, individuals with AIDS die of the disease at a rate δ. Putting together the flow diagram in Figure 1 and the above descriptions and assumptions on the dynamics of the disease the model takes the following form of differential equations:

$$\begin{aligned}
\frac{dS_\tau}{dt} &= \eta\Lambda - \left[\mu + \alpha_1 + \lambda_\tau + (1-\kappa)\lambda_n\right]S_\tau \\
\frac{dS_n}{dt} &= (1-\eta)\Lambda + \alpha_1 S_\tau - \left[\mu + (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]\right]S_n \\
\frac{dI_\tau}{dt} &= \left[\lambda_\tau + (1-\kappa)\lambda_n\right]S_\tau - (\mu + \alpha_2 + \gamma_\tau)I_\tau \\
\frac{dI_n}{dt} &= (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]S_n + \alpha_2 I_\tau - (\mu + \gamma_n)I_n \\
\frac{dA}{dt} &= \gamma_\tau I_\tau + \gamma_n I_n - (\mu + \delta)A,
\end{aligned} \tag{1}$$

subject to the following initial conditions: $S_\tau(0) > 0,\ S_n(0) > 0, I_\tau(0) \geq 0, I_n(0) \geq 0$ and $A(0) \geq 0$. The forces of infections associated with HIV transmission by truck drivers (at the rate λ_τ) and non-truck drivers (at the rate λ_n) are $\lambda_\tau = \dfrac{\beta(I_\tau + \varepsilon A)}{N}$ and $\lambda_n = \dfrac{\beta(I_n + \varepsilon A)}{N}$, respectively.

Descriptions of variables and parameters of model (1) are given in Table 1.

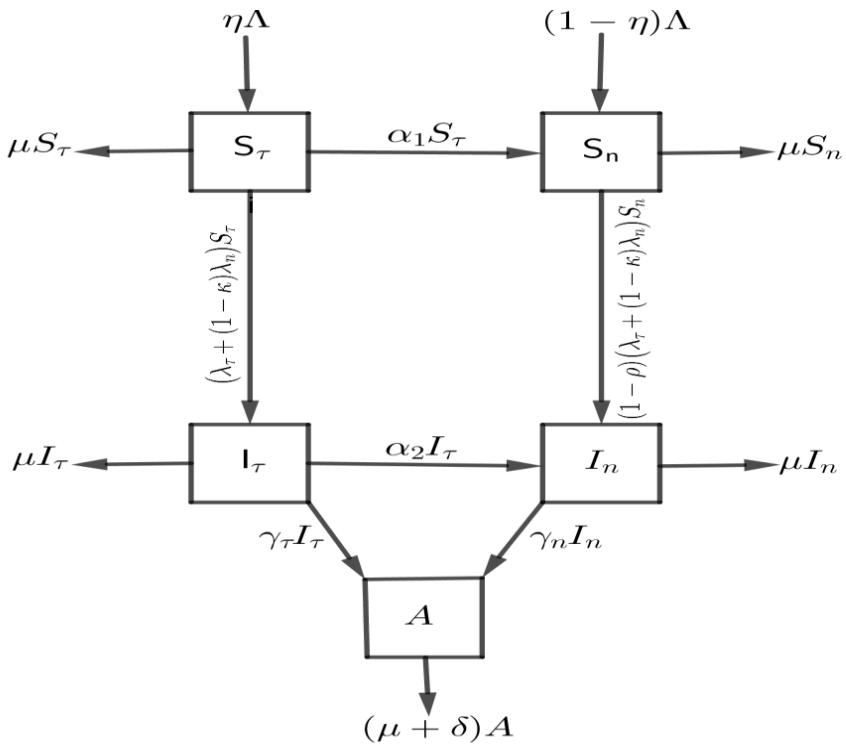

Figure 1: Flowchart for HIV dynamics in truck and non-truck drivers.

Table 1: Descriptions of variables and parameters of model (1)

Symbols	Descriptions
S_τ	Susceptible truck drivers
S_n	Susceptible non-truck drivers
I_τ	Infected truck drivers with no AIDS symptoms
I_n	Infected non-truck drivers without AIDS symptoms
A	Infected individuals with AIDS symptoms
$\lambda_i,\ i=\tau,\ n$	The force of infections for truck and non-truck drivers, respectively
Λ	Recruitment rate for susceptible individuals
η	Fraction of newly recruited truck drivers
β	Effective contact rate that can lead to transmission of infection
κ	Factor for reducing transmission in non-truck drivers
$\gamma_\tau,\ \gamma_n$	Progression rates to AIDS from infected truck and non-truck drivers, respectively
ρ	Factor for reducing the risk behaviour of HIV transmission from non-truck drivers per contact.

Model Analysis

First positivity of the solution of the variables of the model needs to be proved:

Positivity of the solution

Lemma 1: *Let* $t \geq 0$. *If the initial conditions satisfy* $S_\tau(0) > 0$, $S_n(0) > 0$, $I_\tau(0) > 0$, $I_n(0) > 0$, $A(0) > 0$ *then for all* $t \geq 0$, $S_\tau(t)$, $S_n(t)$, $I_\tau(t)$, $I_n(t)$, $A(t)$ *will remain positive in* R_+^5.

Proof: It should be proved that for all $t \geq 0$, $S_\tau(t)$, $S_n(t)$, $I_\tau(t)$, $I_n(t)$, $A(t)$ will remain positive in R_+^5. It is known that all parameters used in the model system (1) are positive. Hence, we can place lower bounds on each of the equations given in the model (1). Thus,

$$\frac{dS_\tau}{dt} = \eta\Lambda - \left[\mu + \alpha_1 + \lambda_\tau + (1-\kappa)\lambda_n\right]S_\tau \geq -\left[\mu + \alpha_1 + (1-\kappa)\lambda_n\right]S_\tau$$

$$\frac{dS_n}{dt} = (1-\eta)\Lambda + \alpha_1 S_\tau - \left[\mu + (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]\right]S_n \geq -\left[\mu + (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]\right]S_n$$

$$\frac{dI_\tau}{dt} = \left[\lambda_\tau + (1-\kappa)\lambda_n\right]S_\tau - (\mu + \alpha_2 + \gamma_\tau)I_\tau \geq -(\mu + \alpha_2 + \gamma_\tau)I_\tau$$

$$\frac{dI_n}{dt} = (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]S_n + \alpha_2 I_\tau \geq -(\mu + \gamma_n)I_n \geq (\mu + \gamma_n)I_n$$

$$\frac{dA}{dt} = \gamma_\tau I_\tau + \gamma_n I_n - (\mu + \delta)A \geq (\mu + \sigma)A$$

Through basic differential equations methods, we can resolve the inequalities and produce:

$$S_\tau \geq S_\tau(0)\exp\left\{-\int(\mu + \alpha_1 + \lambda_\tau + (1-\kappa)\lambda)dt\right\} \geq 0$$

$$S_n \geq S_n(0)\exp\left\{-\int(\mu + (1-\rho)[\lambda_\tau + (1-\kappa)]\lambda_n)dt\right\} \geq 0$$

$$I_\tau \geq I_\tau(0)\exp\left\{-\int(\mu + \alpha_2 + \gamma_\tau)dt\right\} \geq 0$$

$$I_n \geq I_n(0)\exp\left\{-\int(\mu + \gamma_n)dt\right\} \geq 0$$

$$A \geq A(0)\exp\left\{-\int(\mu + \sigma)dt\right\} \geq 0$$

Thus, for all $t \geq 0$, $S_\tau(t)$, $S_n(t)$, $I_\tau(t)$, $I_n(t)$, $A(t)$ will be positive and remain in R_+^5.

Invariant Region

Model (1) describes the dynamics of HIV/AIDS infections/disease in the populations of truck and non-truck drivers through different disease status. All associated parameters and variables of the model are assumed to be non-negative for all $t \geq 0$. Hence, we prove the following lemma:

Lemma 2: *The closed set* $\Phi = \left\{(S_\tau, S_n, I_\tau, I_n, A) \in R_+^5 : N \leq \max\left\{N_0, \frac{\Lambda}{\mu}\right\}\right\}$ *is positively invariant and attracting with respect to the model (1).*

Proof: Adding all the equations in the model (1) gives $\frac{dN}{dt} = \Lambda - \mu N - \delta A,$ where $N = S_\tau + S_n + I_\tau + I_n + A.$ In the absence of the disease we have, $\frac{dN}{dt} \le \Lambda - \mu N,$ and it follows that $\frac{dN}{dt} \le 0$ if $N(t) > \frac{\Lambda}{\mu}.$ The standard comparison theorem (Lakshmikantham et al. 2015) can be used to show that $N(t) \le N(0)e^{-\mu t} + \frac{\Lambda}{\mu}\left(1 - e^{-\mu t}\right).$ In particular, $N(t) \le \frac{\Lambda}{\mu}$ if $N(0) \le \frac{\Lambda}{\mu}$. Thus Φ is positively invariant. Furthermore, if $N(0) > \frac{\Lambda}{\mu},$ then $N(t) \le N_0.$ Hence, $N(t) \le \max\left\{N_0, \frac{\Lambda}{\mu}\right\}$ for all $t \ge 0.$ Therefore, the model is mathematically well posed and epidemiologically reasonable since all the variables remain non-negative for all $t \ge 0$. Hence, it is sufficient to consider the dynamics of the model (1) in Φ.

Disease free equilibrium

When the disease is not present in the community (that is $I_\tau = I_n = A = 0$), the solution of the model (1) gives the disease free equilibrium points. This is the scenario whereby in this study the HIV infections become zero and everyone in the population under consideration is once again susceptible to the disease. The equilibrium points are found by equating the derivatives in model (1) to zero. Thus, the disease free equilibrium of model (1) is given by

$$E^0\left(S_\tau^0,\ S_n^0,\ I_\tau^0,\ I_n^0,\ A\right) = \left(\frac{\eta\Lambda}{\mu + \alpha_1},\ \frac{\Lambda}{\mu}\left[\frac{(1-\eta)(\mu+\alpha_1) + \alpha_1\mu}{\mu + \alpha_1}\right], 0,\ 0,\ 0\right).$$

Local stability of disease free equilibrium

Local stability of the disease free equilibrium is governed by the basic reproduction number (see for instance Brauer and Catillo-Chavez (2001), Castillo-Chavez et al. (2002), and Hethcote (2000)). Epidemiologically, the basic reproduction number of the disease tells us about the number of secondary cases one infected individual produces in an entirely susceptible population during his/her infectious period. We investigate the stability by using the next generation operator and the notations as used in the work of Van den Driessche and Watmough (2002). The matrices F and V for the gain (new infections) terms and loss (transfer) terms, respectively (noting that $N = \frac{\Lambda}{\mu}$ at disease free equilibrium E^0) are:

$$F = \begin{bmatrix} \beta\frac{S_\tau^0}{N^0} & \beta(1-\kappa)\frac{S_\tau^0}{N^0} & \beta(2-\kappa)\varepsilon\frac{S_\tau^0}{N^0} \\ (1-\rho)\beta\frac{S_\tau^0}{N^0} & (1-\rho)\beta(1-\kappa)\frac{S_\tau^0}{N^0} & (1-\rho)\beta(2-\kappa)\varepsilon\frac{S_\tau^0}{N^0} \\ 0 & 0 & 0 \end{bmatrix} \quad (2)$$

and

$$V = \begin{bmatrix} \mu + \alpha_2 + \gamma_\tau & 0 & 0 \\ -\alpha_2 & \mu + \gamma_n & 0 \\ -\gamma_\tau & -\gamma_n & \mu + \delta \end{bmatrix} \quad (3)$$

giving the basic reproduction number R_0 as the spectral radius $\rho\left(FV^{-1}\right)$. That is,

$$R_0 = \rho\left(FV^{-1}\right) = \frac{\mu\beta\eta B_2\left(A_1 + A_2\right) + (1-\rho)B_1A_3A_4}{B_1B_2} \quad (4)$$

where $A_1 = (\delta+\mu)\left[(\mu+\gamma_n) + (1-\kappa)\alpha_2\right]$, $A_2 = \varepsilon(2-\kappa)\left[\alpha_2\gamma_n + (\mu+\gamma_n)\gamma_\tau\right]$,
$A_3 = (1-\kappa)(\delta+\mu) + \varepsilon\gamma_n(2-\kappa)$, $A_4 = \beta\left[\eta\alpha_1 + (1-\eta)(\mu+\alpha_1)\right]$,
$B_1 = (\delta+\mu)(\mu+\alpha_1)(\mu+\gamma_n)(\mu+\alpha_2\lambda_\tau)$, $B_2 = (\delta+\mu)(\mu+\alpha_1)(\mu+\gamma_n)$.

It should be noted that the $A_{i's}, i = 1,..,4$ in the above expressions are different from the "A" that has been used to represent AIDS class. The basic reproduction number R_0 is the sum of the average number of infections generated by typical infectious individuals from the truck drivers ($R_{0\tau}$) and non-truck drivers ($R_{0\eta}$), where $R_{0\tau} = \frac{\rho\mu\beta\left(A_1 + A_2\right)}{B_1}$ and $R_{0\eta} = (1-\rho)\frac{A_3A_4}{B_2}$

Using Theorem 2 of Van den Driessche and Watmough (2002), the following result is established.

Theorem 1: The disease free equilibrium Φ of the model system (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 1 implies that infections of HIV can be minimal if $R_0 < 1$, provided the initial sizes of the sub-populations are within the domain of attraction of Φ. To ensure that stability of the disease free equilibrium is independent of the initial sizes of the sub-populations, we need to show that it is globally asymptotically stable.

Global stability of the disease free equilibrium

Equilibrium is globally stable if it is stable for almost all initial conditions, not just those that are close to it (Martcheva 2015). The following result follows on the global stability of the disease free equilibrium E^0.

Theorem 2: If $R_0 < 1$ the disease-free equilibrium, of the model system (1) is globally asymptotically stable and unstable if $R_0 > 1$.

Proof: By the comparison theorem, the rate of change of the variables representing the infected components of model system (1) can be re-written as

$$\begin{bmatrix} I'_\tau(t) \\ I'_\eta(t) \\ A'(t) \end{bmatrix} = (F-V)\begin{bmatrix} I_\tau \\ I_\eta \\ A \end{bmatrix} - \begin{bmatrix} (\lambda_\tau + (1-\kappa)\lambda_n)S_\tau \\ (1-\rho)(\lambda_\tau + (1-\kappa)\lambda_n)S_n \\ 0 \end{bmatrix}$$

where the matrices F and V are defined as in (2) and (3), respectively. However, we also note that $S_\tau < \frac{\eta\Lambda}{\mu+\alpha_1}$ and $S_n \le \frac{\Lambda}{\mu_1}\left[\frac{(\mu+\alpha_1)(1-\eta)+\alpha_1\eta}{\mu+\alpha_1}\right]$ for all $t \ge 0$ in Φ. Thus

$$\begin{bmatrix} I'_\tau(t) \\ I'_\eta(t) \\ A'(t) \end{bmatrix} \le (F-V)\begin{bmatrix} I_\tau \\ I_\eta \\ A \end{bmatrix} \quad (5)$$

Using the fact that the eigenvalues of the matrix $(F-V)$ all have negative real parts, it follows that the linearized differential inequality system (5), is stable whenever $R_0 < 1$ (see, for example Mushayabasa et al. (2011) for details). Consequently, from the fact that

$$\frac{dI_\tau}{dt} \geq -(\mu+\alpha_2+\gamma_\tau)I_\tau, \frac{dS_n}{dt} \geq -(\mu+(1-\rho)[\lambda_\tau+(1-\kappa)\lambda_n])S_n \text{ and}$$

$$\frac{dA}{dt} \geq -(\mu+\delta)A, \text{ it follows that } (I_\tau, I_n, A) \to (0, 0, 0) \text{ as } t \to \infty.$$

Thus, by comparison theorem according to Lakshmikantham et al (2015), $(I_\tau, I_n, A) \to (0, 0, 0)$ as $t \to \infty$ and solving system (1) at $I_\tau = I_n = A = 0$ gives

$$S_\tau \to \frac{\eta\Lambda}{\mu+\alpha_1} \text{ and}$$

$$S_n \to \frac{\Lambda}{\mu_1}\left[\frac{(\mu+\alpha_1)(1-\eta)+\alpha_1\eta}{\mu+\alpha_1}\right] \quad \text{for}$$

$R_0 < 1$. Hence, the disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

Endemic Equilibrium

Here, the interest is to explore the long term persistence and endemic dynamics of HIV infections. The endemic stage is facilitated in the population by the influx of new susceptible individuals. The endemic equilibrium points are given by $E^* = (S_\tau^*, S_n^*, I_\tau^*, I_n^*, A^*)$, where

$$S_\tau^* = \frac{\eta\Lambda}{\mu+\alpha_1+\lambda^*}, \quad S_n^* = \frac{\Lambda[D_4(\mu+\alpha_1+\lambda^*)+\alpha_1\eta]}{(\mu+\alpha_1+\lambda^*)(\mu+D_3\lambda^*)}, \quad I_\tau^* = \frac{\lambda^*\eta\Lambda}{D_5(\mu+\alpha_1+\lambda^*)},$$

$$I_n^* = \frac{\Lambda[\lambda^* D_3D_4D_5(\mu+\alpha_1+\lambda^*)+\lambda^*\alpha_1\eta D_3D_5+\alpha_2\eta\lambda^*(\mu+D_3\lambda^*)]}{(\mu+\alpha_1+\lambda^*)(\mu+D_3\lambda^*)D_3D_5}$$

$$A^* = \frac{\Lambda[\gamma_\tau\eta\lambda^* D_2(\mu+D_3\lambda^*)+D_3D_4D_5\gamma_n\lambda^*(\mu+\alpha_1+\lambda^*)+\lambda^*\alpha_1\eta+\gamma_n\alpha_2\eta\lambda^*(\mu+D_3\lambda^*)]}{(\mu+\alpha_1+\lambda^*)(\mu+D_3\lambda^*)D_1D_2D_5}$$

$$\text{with } \lambda^* = \frac{\beta[I_\tau^*+(1-\kappa)I_n^*+(2-\kappa)\varepsilon A^*]}{N^*} \qquad (6)$$

and $D_1 = \mu+\delta,\ D_2 = \mu+\gamma_n,\ D_3 = 1-\rho,\ D_4 = 1-\eta,\ D_5 = \mu+\alpha_2+\gamma_\tau,$
$D_6 = \mu+\alpha_1,\ D_7 = 1-\kappa$.

Substituting $S_\tau^*, S_n^*, I_\tau^*, I_n^*, A^*$ into Equation (6) and simplifying, yields the following polynomial $\lambda^* f(\lambda^*) = \lambda^*(A_0(\lambda^*)^2 + B_0\lambda^* + C_0)$ (7)

where,

$$A_0 = \eta D_1D_2D_3 + D_1D_2D_3D_4D_5 + \alpha_2\eta D_1D_2 + \gamma_\tau\eta D_2D_3 + \gamma_n D_3D_4D_5 + \gamma_n\alpha_2\eta D_3,$$

$$B_0 = \eta D_1D_2D_3D_5 + D_1D_2D_4D_5 + \mu\eta D_1D_2 + D_1D_3D_4D_5 + \alpha_1\eta D_1D_3D_5 + \alpha_2\eta\mu D_1 + \mu\gamma_\tau\eta D_2$$
$$+ \gamma_n\alpha_2\eta\mu + \gamma_n\alpha_1\eta D_3D_4 + \gamma_n D_3D_4D_5D_6 - \beta D_1D_3D_4D_5D_7 - (2-\kappa)\varepsilon\beta\gamma_n D_3D_4D_5$$
$$- \beta\eta[D_1D_2D_3 + \alpha_2 D_1D_3D_7 + (2-\kappa)\varepsilon D_2D_3 + (2-\kappa)\varepsilon\gamma_n\alpha_2 D_3],$$

$$C_0 = D_1D_2D_5D_6[1-R_0].$$

One of the solutions of Equation (7) is $\lambda_1^* = 0$ and this corresponds to the disease free equilibrium point E^0. The other solutions are obtained from $f(\lambda^*) = A(\lambda^*)^2 + B_0\lambda^* + C_0$. These correspond to the endemic equilibrium point since the disease is present in the population. There are three cases to consider for $\lambda^* = 0$ in Equation (7), depending on the signs of B_0 and C_0 since A_0 is always positive. That is:

1. If $B_0 < 0$ and $C_0 = 0$ or $B_0^2 - 4A_0C_0 = 0$, then Equation (7) has unique endemic equilibrium point (one positive root) and no possibility of backward bifurcation.
2. If $B_0 > 0$ and $C_0 > 0$ or $B_0^2 - 4A_0C_0 > 0$, then Equation (7) has two equilibrium points (two positive roots) and there is a possibility of backward bifurcation.
3. Otherwise there is none.

However, it is important to note that C_0 is always positive if $R_0 < 1$ and negative if $R_0 > 1$. Hence, the above explanation leads to the following theorem:

Theorem 3: The model system (1) has precisely

(i) one unique endemic equilibrium if $C_0 < 0$ and if and only if $R_0 > 1$,

(ii) one unique endemic equilibrium if $B_0 < 0$ and if and only if $C_0 = 0$ or $B_0^2 - 4A_0C_0 = 0$,

(iii) two endemic equilibria if $B_0 < 0, C_0 > 0$ and $B_0^2 - 4A_0C_0 > 0$,

(iv) Otherwise it has no (biologically meaningful) solution.

Bifurcation Analysis

The stability of the endemic equilibrium point can be determined by computing the eigenvalues of the Jacobian matrix at the endemic equilibrium. However, due to the mathematical complexity of this approach for model system (1), the Centre manifold theory will be used to analyze the stability near the disease-free equilibrium point, E^0 and $R_0 = 1$. Let β be a bifurcation parameter and $R_0 = 1$ be the bifurcation point. Then solving for β from Equation (4) yields

$$\beta = \frac{B_1B_2}{\mu\beta\eta\, B_2(A_1 + A_2) + (1-\rho)B_1A_3A_4} \quad 8)$$

Let $S_\tau = x_1$, $S_n = x_2$, $I_\tau = x_3$, $I_n = x_4$, $A = x_5$. Then the model system (1) is rewritten as follows

$$\frac{dx}{dt} = (f_1(x), f_2(x), f_3(x), f_4(x), f_5(x))^T \quad (9)$$

such that model system (1) becomes

$$
\begin{aligned}
\frac{dx_1}{dt} &= f_1(x_1) = \eta\Lambda - \left[\mu + \alpha_1 + \lambda_\tau + (1-\kappa)\lambda_n\right]x_1,\\
\frac{dx_2}{dt} &= f_2(x_1, x_2) = (1-\eta)\Lambda + \alpha_1 x_1 - \left[\mu + (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]\right]x_2,\\
\frac{dx_3}{dt} &= f_3(x_1, x_3) = \left[\lambda_\tau + (1-\kappa)\lambda_n\right]x_1 - (\mu + \alpha_2 + \gamma_\tau)\, x_3, \qquad (10)\\
\frac{dx_4}{dt} &= f_4(x_2, x_3, x_4) = (1-\rho)\left[\lambda_\tau + (1-\kappa)\lambda_n\right]x_2 + \alpha_2\, x_3 - (\mu + \gamma_n)\, x_4,\\
\frac{dx_5}{dt} &= f_5(x_3, x_4, x_5) = \gamma_\tau\, x_3 + \gamma_n\, x_4 - (\mu + \delta)\, x_5.
\end{aligned}
$$

Thus, the Jacobian matrix of the system (10) at the disease free equilibrium becomes

$$
J_{E^0} = \begin{bmatrix}
-(\alpha_1 + \mu) & 0 & -M & -L & -G \\
\alpha_1 & -\mu & -P & -Q & -H \\
0 & 0 & M - R & L & I \\
0 & 0 & P + \alpha_2 & Q - U & J \\
0 & 0 & \gamma_\tau & \gamma_n & -(\mu + \delta)
\end{bmatrix} \qquad (11)
$$

where $M = \dfrac{\beta x_1}{N}$, $L = \dfrac{(1-\kappa)\beta x_1}{N}$, $P = \dfrac{(1-\rho)\beta x_2}{N}$, $Q = \dfrac{(1-\rho)(1-\kappa)\beta x_2}{N}$,

$R = \mu + \alpha_2 + \gamma_\tau$, $U = \mu + \gamma_n$, $G = \dfrac{\beta\varepsilon(2-\kappa)x_1}{N}$, $H = \dfrac{\beta\varepsilon(1-\rho)(2-\kappa)x_2}{N}$,

$$
I = \frac{\beta\varepsilon(1-\kappa)x_1}{N}, \quad J = \frac{\beta\varepsilon(1-\rho)(2-\kappa)x_2}{N}.
$$

It is noted that zero is a simple eigenvalue of matrix (11). Let $w = (w_1, w_2, w_3, w_4, w_5)$ be the right eigenvector associated with the eigenvalue zero. Then we have

$$
\begin{bmatrix}
-(\alpha_1 + \mu) & 0 & -M & -L & -G \\
\alpha_1 & -\mu & -P & -Q & -H \\
0 & 0 & M - R & L & I \\
0 & 0 & P + \alpha_2 & Q - U & J \\
0 & 0 & \gamma_\tau & \gamma_n & -(\mu + \delta)
\end{bmatrix}
\begin{bmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \end{bmatrix} =
\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \qquad (12)
$$

Solving the system (12) for w_i, $i = 1, \ldots, 5$ gives the following:

$$
w_1 = \frac{-Mw_3 - Lw_4 - Gw_5}{\mu + \alpha_1}, \; w_2 = \frac{\alpha_1 w_1 - Pw_3 - Qw_4 - Hw_5}{\mu}, \; w_3 = \frac{-Bw_4 - I\, w_5}{R - M}, \qquad (13)
$$

$$
w_4 = \frac{-(P - \alpha_2)w_3 - J\, w_5}{U - Q}, \quad w_5 = \frac{\gamma_\tau w_3 + \gamma_n w_4}{\mu + \delta}.
$$

To calculate the left eigenvector $\underline{v} = (v_1, v_2, v_3, v_4, v_5)$ and satisfying $\underline{v} \cdot \underline{w} = 1$, we transpose (11) which leads to the following system of equations:

$$\begin{aligned}
&-(\alpha_1 + \mu)v_1 + \alpha_2 v_2 = 0 \\
&-\mu v_2 = 0 \\
&-M v_1 - P v_2 + (M - R)v_3 + (P + \alpha_2)v_4 + \gamma_\tau v_5 = 0 \\
&-L v_1 - Q v_2 + L v_3 + (Q - U)v_4 + \gamma_n v_5 = 0 \\
&-G v_1 - H v_2 + I v_3 + J v_4 - (\delta + \mu)v_5 = 0
\end{aligned} \tag{14}$$

From (14) the left eigenvector is

$$\begin{aligned}
&v_1 = \frac{\alpha_2 v_2}{\alpha_1 + \mu},\ v_2 = v_2 > 0,\ v_3 = \frac{M v_1 + P v_2 - (P + \alpha_2)v_4 - \gamma_\tau v_5}{M - R} \\
&v_4 = \frac{L v_1 + Q v_2 - L v_3 - \gamma_n v_5}{Q - U},\ \ v_5 = \frac{G v_1 + H v_2 - I v_3 - J v_4}{\mu + \delta}
\end{aligned} \tag{15}$$

Now, to prove the local stability of endemic equilibrium point near $R_0 = 1$, we apply the following Theorem as outlined in Castillo-Chavez and Song (2004):

Theorem 5: Consider the following system of ordinary differential equations with a parameter ϕ

$$\frac{dx}{dt} = f(x, \phi),\ f : \Re^n \times \Re \to \Re^n \text{ and } f \in C^2(\Re^n \times \Re) \tag{16}$$

where 0 is the equilibrium point of the system, that is, $f(0, \phi) \equiv 0\ \ \forall \phi$ and

(A1) $W_0 = D_x f(0,0) = \left[\frac{\partial f_i}{\partial x_j}(0,0) \right]$ is the linearization matrix of the system around the equilibrium 0 with ϕ evaluated at 0;

(A2) Zero is a simple eigenvalue of W_0 and all other eigenvalues of W_0 have negative real parts;

(A3) Matrix W_0 has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i\, dx_j}(0,\ 0),\ \ b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(0,0). \tag{17}$$

The local dynamics of system (17) around 0 are totally governed by the signs of *a* and *b*.

(i) $a > 0, b > 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi << 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;

(ii) $a < 0, b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is unstable; when $0 < \phi << 1$, 0 is asymptotically stable, and there exists a positive unstable equilibrium;

(iii) $a > 0, b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is unstable; and there exists

a locally asymptotically stable negative equilibrium; when $0 < \phi << 1$, 0 is stable, and a positive unstable equilibrium appears;

(iv) $a < 0, b > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $a > 0$ and $b > 0$, then a backward bifurcation occurs at $\phi = 0$.

Computation of a and b: For the system (18), the associated non-zero second order partial derivatives (at the disease free equilibrium point) for *a* and *b* are given by:

$$a = v_1 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_1}{\partial x_i \partial x_j}(0,0) + v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + v_3 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_3}{\partial x_i \partial x_j}(0,0)$$
$$+ v_4 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_4}{\partial x_i \partial x_j}(0,0) + v_5 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0),$$

$$b = v_1 \sum_{i=1}^{5} w_i \frac{\partial^2 f_1}{\partial x_i \partial \phi}(0,0) + v_2 \sum_{i=1}^{5} w_i \frac{\partial^2 f_2}{\partial x_i \partial \phi}(0,0) + v_3 \sum_{i,j=1}^{5} w_i \frac{\partial^2 f_3}{\partial x_i \partial \phi}(0,0)$$
$$+ v_4 \sum_{i=1}^{5} w_i \frac{\partial^2 f_4}{\partial x_i \partial \phi}(0,0) + v_5 \sum_{i=1}^{5} w_i \frac{\partial^2 f_5}{\partial x_i \partial \phi}(0,0),$$

But the non-zero second order partial derivatives at the disease free equilibrium point are given by

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_3} = \frac{\partial^2 f_1}{\partial x_3 \partial x_1} = -\frac{\beta}{N}, \quad \frac{\partial^2 f_1}{\partial x_1 \partial x_4} = \frac{\partial^2 f_1}{\partial x_4 \partial x_1} = -\frac{(1-\kappa)\beta}{N}.$$

Results and Discussion

Numerical simulations of the model (1) were carried out by using the set of parameter values given in Table 1. Some parameter values were obtained from different literatures and others were assumed. The model system has been simulated by using MATLAB-ODE solvers and the following initial conditions (which are just arbitrary numbers) have been considered: $S_\tau(0) = 6{,}000{,}000$, $S_n(0) = 9{,}000{,}000$, $I_\tau(0) = 300{,}000$, $I_n(0) = 800{,}000$ and $A(0) = 50{,}000$. The parameter values for model (1) are presented in Table 2.

Table 2: Parameter values for model (1)

Parameter	Value (per year)	Source
Λ	10	Hassan (2013)
η	0.4	Assumed
α_1	0.3	Assumed
α_2	0.5	Hassan (2013)
μ	0.02	Mushayabasa et al. (2011)
ρ	0.99	Assumed
γ_τ	2.6	Hethcote (1999)
γ_n	0.06	Hethcote (1999)
δ	0.333	Bhunu et al. (2009)
ε	1.2	Sharom and Gumel (2008)
κ	0.99	Hassan (2013)
β	0.4	Elibasha and Gumel (2006)

Figure 2 shows that $R_{0\eta} < R_{0\tau} < R_0$, implying that truck drivers have significant roles in the transmission dynamics of HIV infections, since the average number of new infections from a typical infectious truck driver $R_{0\tau}$ (middle curve in Figure 2) is greater than the number of infections produced by a non-truck driver R_{0n} (lower curve in Figure 2). That is, the contribution of non-truck drivers in the transmission of infections is less than their counterparts (truck drivers) because they have minimal interactions with potential sex workers available on the truck routes.

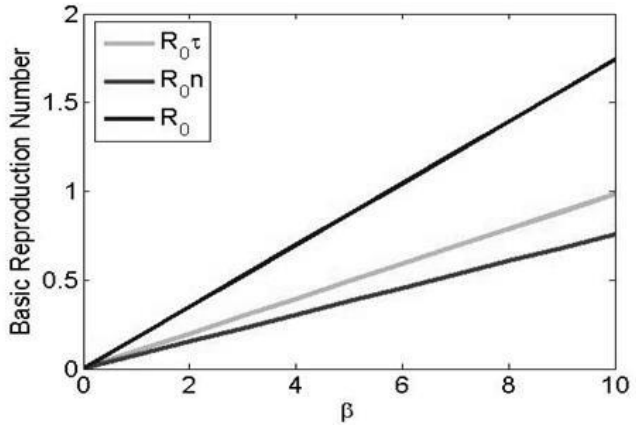

Figure 2: The comparison of reproduction numbers from truck drivers ($R_{0\tau}$) and non-truck drivers ($R_{0\eta}$) against the effective contact rate β.

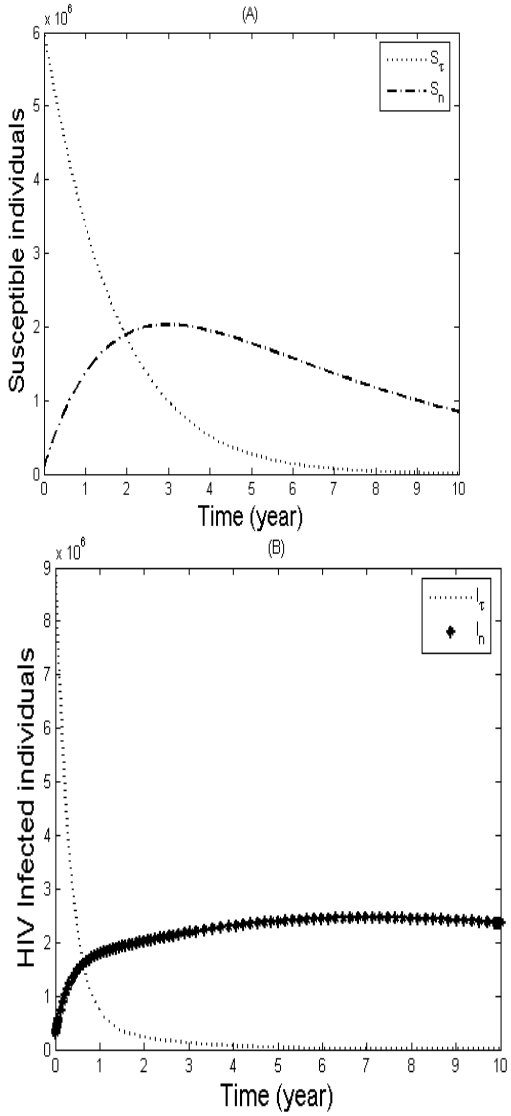

Figure 3: (A) Simulation results showing general trends in the dynamics of the disease between susceptible truck drivers (S_τ, dotted curve) and non-truck drivers (S_n, dash dotted curve). (B) Similarly, for infected truck drivers (I_τ, dotted curve) and non-truck drivers (I_n, star curve): $R_0 = 1.2462$.

Figure 3 (A) shows that susceptible population of truck drivers decreases (dotted curve), while susceptible population of non-truck drivers increases slowly and then decreases (dash dotted curve). This may be due to the nature of their work which exposes susceptible truck drivers to the risk behaviours of HIV infections. Also, some of the susceptible truck drivers may quit the job and join the susceptible non-truck drivers. On the other hand, the population of susceptible non-truck drivers increases up to its equilibrium point, then decreases. This could be due to the fact that non-truck drivers are not as exposed to high risk behaviours as susceptible truck drivers. Similarly, Figure 3 (B) shows that most of the HIV infected truck drivers (dotted curve) progress faster to full blown AIDS than the HIV infected non-truck drivers (star curve). This is due to fact that the HIV infected truck drivers have high probability of contracting the disease per contact and even re-infection compared to non-truck drivers.

Sensitivity analysis

Sensitivity analysis is performed in order to determine the relative importance of the model parameters on the disease transmission and prevalence. The analysis is performed by calculating the sensitivity indices of the basic reproduction number R_0 with respect to the parameters. The interest is to determine parameters that significantly affect the reproduction number since these are the parameters that should be taken into considered when control strategies are to be implemented (see Chitnis and Hyman (2008) for details). The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the parameter. Since the reproduction is a differentiable function of the parameters, the sensitivity indices may alternatively be defined using partial derivatives. For instance, computation of the sensitivity index of R_0 with respect to β using parameter values in Table 2 is given by $r_\beta^{R_0} = \frac{\beta}{R_0} \times \frac{\partial R_0}{\partial \beta} = 1 > 0.$ This shows

that R_0 is an increasing function of β and the parameter β has very great influence on the spread of HIV infections in the communities. The indices of the remaining parameters are tabulated in Table 3. Parameters whose sensitivity indices have negative signs decrease the value of the reproduction number as their values increase, whereas those with positive signs increase the value of R_0 as they increase. The system is most sensitive to κ (the factor for adjusting transmission probability per contact from non-truck drivers) followed by ρ (the factor for adjusting the risk behaviours of transmission per contact). We note that increasing (decreasing) κ by 10% decreases (increases) R_0 by 23.6%. Also, increasing (decreasing) the parameter β (rate of effective contact that can lead to infection) by 10% increases by 10%.

Table 3: Numerical values of sensitivity indices of R_0

Parameter	Sensitivity index
β	+1.00
ρ	-2.23
δ	-0.86
κ	-2.36
μ	-0.19
α_1	-0.87
α_2	-0.03
γ_τ	-0.03
γ_n	+0.06

Effects of varying some parameters

When looking at different values of the modification factor κ (i.e. the factor for adjusting transmission probability per contact from non-truck drivers), the result suggests that an increase of κ increases the number of susceptible truck drivers as shown in Figure 4 (A) (dash-dotted curve), because of high reduction of transmission probability per contact of HIV. This implies that there are few people who are exposed to the infections. In case of non-truck drivers population, the increase of κ leads to the increase of susceptible non-truck drivers which implies that there are very few people who are exposed to the infections. This is due to the fact that, transmission probability per contact of HIV has been highly reduced as shown in Figure 4 (B) dash-dotted curve. Similarly, Figure 4 (C) shows that as κ increases, it has slightly decreased the number of the HIV infected truck drivers (dotted curve). Figure 4 (D) shows that as κ increases, the numbers of the HIV infected non-truck drivers decrease (dash-dotted curve).

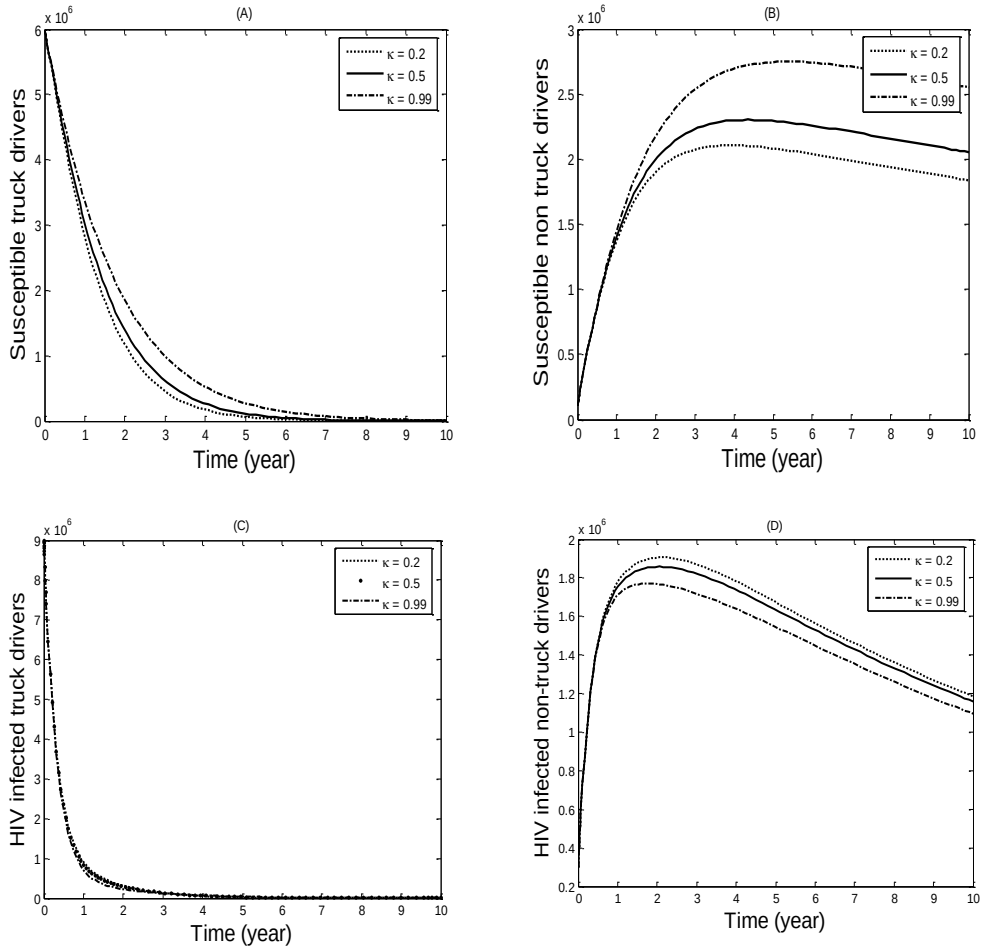

Figure 4: The effect of varying the factor for reducing the risks of HIV transmissions per contact (κ) for susceptible truck drivers (A), susceptible non-truck driver (B), infected truck drivers (C) and infected non-truck drivers (D), where κ = 0.2, 0.5, and 0.99.

Conclusion

A mathematical model for investigating the roles of long distance truck drivers on the transmission dynamics of HIV infection in a population was formulated and analysed. The analysis of the model has been done to investigate the existence and stability of the disease free and endemic equilibrium. The next generation matrix has been used to calculate the basic reproduction number R_0, as well as to investigate the local stability of the disease free equilibrium. The global stability of the disease free equilibrium was investigated by using the Centre Manifold Theory and conditions for the global stability were derived by using the comparison method. Then the disease free equilibrium was shown to be locally asymptotically stable and globally asymptotically stable when the basic reproduction number, R_0 is less than one. Also by using the Centre Manifold Theory, the HIV model of long distance truck drivers is shown to have a unique and locally

asymptotically stable endemic equilibrium when the basic reproduction number R_0 is greater than one. Therefore, it is concluded that the model does not exhibit backward bifurcation, since a stable disease free equilibrium cannot coexist with a stable endemic equilibrium when the basic reproduction number R_0 is less than unity. By analysing the associated basic reproduction number R_0, it showed that long distance truck drivers increase the rates of transmission of HIV infections.

The basic reproductive numbers of the model were computed and compared, which enable the assessment of the roles of long distance truck drivers in transmission dynamics of HIV. The obtained results showed that, the overall basic reproduction number R_0 was greater than that of the long distance truck drivers $R_{0\tau}$, which was also greater than that of the non-truck drivers R_{0n}. These results clearly indicate that long distance truck drivers are at higher risks of infections as compared to non-truck drivers because truck drivers have wider sexual networks than non-truck drivers. Simulations and sensitivity analysis were carried out to illustrate analytical results and determine the key factors influencing the behaviour of the disease. Results from the sensitivity analysis of R_0 suggested that more efforts should put on reducing effective contact rate β, and this can be attained through comprehensive healthcare services and HIV interventions and prevention programmes like condom distribution and voluntary HIV counselling and testing. Thus, from the simulation part, it can be concluded that, long distance truck drivers increase the risks in the transmission of HIV.

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