Modelling the Spread of HIV/AIDS in India Focusing Commercial Sex Worker

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Abstract—According to the major reports of the WHO, it has been observed that the female sex workers play an important role in transmission of the HIV/AIDS in India. In this work, we formulate and investigate a nine-dimensional nonlinear model to study the dynamics of HIV/AIDS epidemic considering the female sex workers as one of the most vulnerable groups in transmission of the HIV/AIDS in India. The model which assumes mass action incidence, is analyzed qualitatively to determine the stability of their equilibria. In addition, analysis of these models includes the presentation of the threshold, the basic reproduction number $R_0$, and numerical simulation that determines whether the diseases dies out or the disease remains endemic.

Index Terms—Basic reproduction number, female sex worker, HIV/AIDS, stability

I. INTRODUCTION

One of the largest and most populated countries in the world is India with over one billion inhabitants. India had no reported cases of HIV or AIDS at the beginning of 1986, despite over 20,000 reported HIV cases worldwide. The first case of HIV in India was diagnosed among sex workers in Chennai, Tamil Nadu later in the year. It was noted that contact with foreign visitors had played a role in initial infections among sex workers. Therefore, HIV screening centers were set up across the country and all visitors were asked to be screened for HIV. As infection rates continued to rise at the beginning of the 1990s, the government set up NACO (National AIDS Control Organization) in 1992 to oversee the formulation of policies, prevention work and control programs relating to HIV and AIDS. The government adopted the National AIDS Prevention and Control Policy in 2001.

According to the report of WHO/UNAIDS 2006 on the global HIV/AIDS epidemic, the number of people living with HIV in India has been increasing exponentially since the early nineties and the most recent AIDS estimate in India is about 5.7 million infected as of 2005 [1]. Since India has a large population, the HIV/AIDS epidemics in India will have a major impact on the overall spread of HIV/AIDS to other countries as well.

In India the majority (85%) of new infections are due to heterosexual transmission, particularly among female sex workers (FSWs), their clients and the sexual contacts of their clients [2–5]. However, in north-east India and in major cities injecting drug use is also a source of new infections [6–10], Several differential equation models for HIV/AIDS in India have been formulated and studied; see e.g. [11, 12] and references therein. Most models differ from one another depending on how the population groups are divided. The female commercial sex worker is playing an important role in spreading HIV/AIDS in Indian society. Considering this with importance, we have formulated the model that will focus on the heterosexual transmission of HIV among males, females and FSWs. Although the FSW are considered as the main source of HIV transmission in the model, those males who have sex with FSW as well as their wives, are also working here as an important factor.

In this work, we show that HIV/AIDS epidemics invade the Indian community based on the data as in references [1, 13, 14], (see Section III of this paper) where commercial sex work plays the important role, using mathematical model of HIV transmission. We use data collected in India to examine the predictions of epidemic size for an urban area in India.

Mathematical models serve a vital role in understanding sexually transmitted disease (STD) infection epidemiology and control. To illustrate/construct STD models, the whole population is divided into different population groups such as males, FSWs and other females based on the model desired structure. Then, for each population group, the concept of SI models is used to formulate differential equations describing the sexual relationship among the different population groups. As well, STD models describe the transition of the individuals in the population through a sequence of disease related stages for each group separately. One way to visualize these models is using state flow diagrams, where boxes represent compartments and arrows indicate the transitions (see Fig. 1, for example).

In the general article on Mathematical modelling of AIDS epidemic in India [12], author divides the whole population into three groups such as number of susceptible to HIV, number of HIV infective, number of AIDS cases etc. and has discussed briefly about mathematical equations along with numerical simulation. He has not presented any analytical analysis for his model. In the research paper [11], authors have formulated the models dividing the population into two groups such as males and females and their proposed model is demonstrated through a robust statistical approach with numerical simulation. We divide the population differently and consider multiple stages of infection for each group of population (for example, see [15]). In fact, in [15], they study the impact of vaccination on HIV/AIDS, but do not divide the
population according to gender or activity level, as is done here.

The HIV transmission model is developed. We discuss equilibria of the model, the Jacobian matrix and local stability. The basic reproduction number is also calculated. The analysis of the centre manifold is presented as well.

Numerical simulations and some important diagrams related to the model are illustrated based on the various real parameter values collected from one of the major cities, Delhi in India indicated in Table I.

II. MODEL OF HIV TRANSMISSION

A. Model Formulation

At first, we divide the population into three classes: Male, Female and FSW to derive the mathematical equations of the model. Populations of those not infected by HIV are denoted as those infected but who have not yet developed AIDS as , and those who subsequently develop AIDS as , for males, females and FSWs respectively. Here, it is assumed that HIV-infected individuals will clinically progress to AIDS (the advanced stage of HIV infection) with an average incubation period , for males, , for females, and , for FSWs. All transmissions are assumed to be heterosexual. Females transmit the virus to their male counterparts with a probability (at contact rate for female and for FSW), while males transmit to females with a probability (at contact rate for female and for FSW). Males, females and FSWs enter the susceptible class (susceptible here means those who can get the infection but are not infected yet) at constant rates , and respectively. Infected individuals are allowed to withdraw from risk behavior (at rates and , say) and die naturally (at rates and , say). These death rates are unrelated to AIDS, hence the same rates are applicable for non-infected individuals. Deaths which are due to AIDS are assumed to be the same (at rate , say) for male, female and FSW. The structure of HIV transmission in the model is summarized in Fig. 1. The differential equations for the model are given in Eq. (1).

\[
\begin{align*}
\frac{dM_1}{dt} &= \lambda_1 (\beta_1 M_2 + \beta_2 M_3) - \mu M_1 \\
\frac{dM_2}{dt} &= (\beta_1 M_1 + \beta_2 M_3) - (\alpha_1 + \alpha_2 + \mu) M_2 \\
\frac{dM_3}{dt} &= \alpha_1 M_2 \quad \text{(1)}
\end{align*}
\]

The system of ordinary differential equations (1) can be concised by eliminating equations for , since they are redundant for calculation as do not appear in the equations for the other variables. Now the system of Eqs. (1–2).

\[
\begin{align*}
\frac{dM_1}{dt} &= \lambda_1 (\beta_1 M_2 + \beta_2 M_3) - \mu M_1 \\
\frac{dM_2}{dt} &= (\beta_1 M_1 + \beta_2 M_3) - (\alpha_1 + \alpha_2 + \mu) M_2 \\
\frac{dF_1}{dt} &= \lambda_2 - \beta_2 F_1 M_3 - \mu F_1 \\
\frac{dF_2}{dt} &= \beta_2 F_1 M_3 - (\alpha_1 + \alpha_2 + \mu) F_2 \\
\frac{dF_3}{dt} &= \beta_2 F_1 M_3 - (\alpha_1 + \alpha_2 + \mu) F_3 \quad \text{(2)}
\end{align*}
\]

Conclusions about the system (1) can be easily recovered from the system (2) and we employ the system (2) from now on.

B. Equilibria of the Model

The disease-free equilibrium for the system (2) is given as

\[
[x_0] = \left( M_1^*, M_2^*, F_1^*, F_2^*, F_3^* \right)_{DEF} = \left( \frac{\lambda_1}{\mu_1}, \frac{\lambda_2}{\mu_2}, \frac{\lambda_3}{\mu_3} \right)
\]

where 0 indicates that there is no HIV affected people (i.e., no disease) in the population.

Now consider the endemic equilibrium with . By setting the left hand side of system (2) equal to 0, express in terms of by backward substitution. Then, substituting all these in second equation of (2), it is found that is determined by the solutions of the quadratic equation.
The position of the EEP (Endemic Equilibrium Point) depends on the solution of \( P(M_2) = 0 \), more specifically, \( M_2 = -\frac{B \pm \sqrt{B^2 - 4AC}}{2A} \). The EEP in system (2) is given by

\[
P(M_2) = AM_2^2 + BM_2 + C = 0
\]

where

\[
\begin{align*}
A &= k_1(d_1 \phi_1 + d_2 \phi_2) + k_2 \mu_1 \\
B &= k_1(d_1 \phi_1 + d_2 \phi_2) + k_2 \mu_1 \\
C &= k_1 \phi_1 \mu_1 - k_2 \phi_2 \mu_2
\end{align*}
\]

The Jacobian at disease free equilibrium is given by

\[
J_0 = Df(\mathbf{x}_0)
\]

where

\[
J_0 = \begin{bmatrix}
-k_1 & \frac{\beta_1 \phi_1}{\mu_1} & \frac{\beta_2 \phi_1}{\mu_1} & 0 & 0 \\
\frac{\beta_1 \phi_1}{\mu_1} & -k_2 & 0 & 0 & 0 \\
\frac{\beta_2 \phi_1}{\mu_1} & 0 & -k_3 & 0 & 0 \\
0 & \frac{\beta_2 \phi_1}{\mu_1} & \frac{\beta_2 \phi_1}{\mu_1} & -k_4 & 0 \\
0 & 0 & 0 & \frac{\beta_2 \phi_1}{\mu_1} & -k_5
\end{bmatrix}
\]

where the notation \( Df(\mathbf{x}_0) \) indicates the partial derivative of \( f \) with respect to \( \mathbf{x} \) evaluated at \( \mathbf{x}_0 \). The DFE, \( \mathbf{e}_0 \), is locally asymptotically stable if all the eigenvalues of the matrix \( J_0 \) have negative real parts and unstable if any eigenvalue of \( J_0 \) has a positive real part.

Since all the entries of column four, five and six of matrix \( J_0 \) are zero except the negative entries of the diagonal, three eigenvalues of \( J_0 \) are definitely negative. In order to check the other three eigenvalues of \( J_0 \), whether they are positive or negative, we use Lemma 3 discussed in [16]. Let \( J_1 \) be the upper left \( 3 \times 3 \) sub-matrix of \( J_0 \), namely, \( f \)

\[
\begin{bmatrix}
-k_1 & \frac{\beta_1 \phi_1}{\mu_1} & \frac{\beta_2 \phi_1}{\mu_1} \\
\frac{\beta_1 \phi_1}{\mu_1} & -k_2 & 0 \\
\frac{\beta_2 \phi_1}{\mu_1} & 0 & -k_3
\end{bmatrix}
\]

Following Lemma 3 of [17], for \( k_0 < 0 \), it can be shown that \( \lambda(\lambda_1), \lambda(\lambda_2) \) and \( \lambda(\lambda_3) \), where \( \lambda(\lambda_i) \) is the second additive compound of the matrix \( J_1 \) (see, e.g., [17] or [18]), are negative. This gives the result that the other three eigenvalues of \( J \) have negative real part as well if \( k_0 < 0 \) where \( k_0 \) is given below in (5). Thus, the DFE is locally asymptotically stable if \( k_0 < 0 \).

**C. Jacobian Matrix at DFE and Local Stability**

In order to find the Jacobian at disease free equilibrium we consider the system (2) as
D. The Basic Reproduction Number

Here, we have found the basic reproduction number using the method discussed in [19]. To find \( R_0 \) the following notations are used:

\[
\frac{dx}{dt} = \begin{bmatrix}
\frac{dx_1}{dt} \\
\frac{dx_2}{dt} \\
\frac{dx_3}{dt}
\end{bmatrix} = \begin{bmatrix}
\nu_1 \\
\nu_2 \\
\nu_3
\end{bmatrix}
\]

where

\[
\begin{pmatrix}
\nu_1 \\
\nu_2 \\
\nu_3
\end{pmatrix} = \begin{pmatrix}
\beta_1 c_1 x_2 + \beta_2 c_1 x_3 \\
\beta_2 c_2 x_1 \\
\beta_2 c_3 x_1 + \beta_3 c_3 x_2
\end{pmatrix}
\]

and

\[
\begin{pmatrix}
\nu_1 \\
\nu_2 \\
\nu_3
\end{pmatrix} = \begin{pmatrix}
k x_1 \\
k x_2 \\
k x_3
\end{pmatrix}
\]

Then, letting \( \sigma = (x_1, x_2, x_3)^T \), we let \( r \) and \( v \) be the Jacobian. Now we have

\[
F = \frac{\partial \sigma}{\partial y} = \begin{pmatrix}
\frac{\partial \nu_1}{\partial x_1} & \frac{\partial \nu_1}{\partial x_2} & \frac{\partial \nu_1}{\partial x_3} \\
\frac{\partial \nu_2}{\partial x_1} & \frac{\partial \nu_2}{\partial x_2} & \frac{\partial \nu_2}{\partial x_3} \\
\frac{\partial \nu_3}{\partial x_1} & \frac{\partial \nu_3}{\partial x_2} & \frac{\partial \nu_3}{\partial x_3}
\end{pmatrix}
\]

and

\[
v = \frac{\partial \nu}{\partial y} = \begin{pmatrix}
\frac{\partial \nu_1}{\partial x_1} & \frac{\partial \nu_1}{\partial x_2} & \frac{\partial \nu_1}{\partial x_3} \\
\frac{\partial \nu_2}{\partial x_1} & \frac{\partial \nu_2}{\partial x_2} & \frac{\partial \nu_2}{\partial x_3} \\
\frac{\partial \nu_3}{\partial x_1} & \frac{\partial \nu_3}{\partial x_2} & \frac{\partial \nu_3}{\partial x_3}
\end{pmatrix}
\]

Thus, following reference [19], \( Fv^{-1} \) is the next generation matrix for the model and

\[
R_0 = \rho(Fv^{-1}) = \frac{k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu) + k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu)}{\mu_1 \mu_2 \mu_3}
\]

where \( \rho(Fv^{-1}) \) denotes the spectral radius of the matrix \( Fv^{-1} \). Suppose \( R_0 > 1 \). Then

\[
R_0 > 1
\]

\[
\Rightarrow \frac{k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu)}{\mu_1 \mu_2 \mu_3} > k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu)
\]

\[
\Rightarrow \beta \mu c \lambda (c c k \mu k \mu + k c c k \mu) > k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu)
\]

\[
\Rightarrow k k \mu c \beta \lambda (c c k \mu k \mu + k c c k \mu) < 0
\]

Then the expressions for \( A, B \) and \( C \) given in (3) satisfy

\[
A = k (d d_1 + d d_2) + k k \mu d d_3
\]

\[
= k (d d_1 + d d_2) + k k \mu d d_3 > 0
\]

\[
C = k (d d_1 + d d_2) - k (d d_1 + d d_2)
\]

\[
= k (d d_1 + d d_2) - k (d d_1 + d d_2) < 0
\]

by (6), and

\[
B = k (d d_1 + d d_2) + k k \mu (d d_1 + d d_2) - k k \mu (d d_1 + d d_2)
\]

\[
= k (d d_1 + d d_2) + k k \mu (d d_1 + d d_2)
\]

\[
= k (d d_1 + d d_2) + k k \mu (d d_1 + d d_2)
\]

Then the expressions \( \sqrt{B^2 - 4AC} \) for \( A, B \) and \( C \) given in (5) satisfy

\[
M_2 = -B + \sqrt{B^2 - 4AC}
\]

\[
> 0
\]

Therefore, the EE exists (i.e. is positive) and is unique since the other root \( \frac{-B - \sqrt{B^2 - 4AC}}{2A} \) is negative whenever \( R_0 > 1 \).

E. Analysis of the Centre Manifold Near \( x = s, R_0 = 1 \)

According to [19], we consider the nature of the equilibrium solutions of the disease transmission model near the bifurcation point \( x = s, R_0 = 1 \). Since \( R_0 \) is often inconvenient to use directly as bifurcation parameter, we introduce a bifurcation parameter \( \theta \). Let \( \theta \) be a bifurcation parameter such that \( R_0 < 1 \) for \( \theta < 0 \) and \( R_0 > 1 \) for \( \theta > 0 \) and such that \( \epsilon \theta \) is a DFE for all values of \( \theta \). Let us consider the system

\[
\dot{x} = f(x, \theta)
\]

where \( f \) is as described in (4). The choice of \( \theta \) is given below, when the quantity \( b \) is calculated. The position of the DFE depends particularly on the choice of \( \theta \) and the local stability changes at the point \( (s_0, 0) \). The results of centre manifold theory [22] are used here to show that there are nontrivial (endemic) equilibria near the bifurcation point \( (s_0, 0) \).
We use the notation \( f_x = \frac{\partial f}{\partial x} \) for the partial derivative of \( f \) with respect to \( x \) evaluated at \( x = x_0 \) and \( \theta = 0 \). Let \( v \) and \( u \) be the corresponding left and right eigenvectors chosen such that \( v_j = 0 \) and \( J_{uv} = 0 \) with \( v = [v_1, v_2, v_3, v_4, v_5]^T \) and \( u = [u_1, u_2, u_3, u_4, u_5]^T \). Following [19], we define

\[
\begin{align*}
&v_1 = \frac{1}{Q} \\
&Q = \frac{\beta v_1 \beta v_2 \beta v_3 \lambda_1 \lambda_2 + \beta v_1 \beta v_2 \beta v_3 \lambda_2 \lambda_3}{k_1^2 \mu_2^2 + k_2^2 \mu_2^2}
\end{align*}
\]

Substituting the values of \( v \) and \( u \) in (7) and (8), we get

\[
\begin{align*}
a &= -\frac{\beta v_1}{\mu_1^2} + \frac{\beta v_2}{\mu_2^2} + \frac{\beta v_3}{\mu_3^2} \\
b &= -\frac{\beta v_1}{\mu_1^2} + \frac{\beta v_2}{\mu_2^2} + \frac{\beta v_3}{\mu_3^2}
\end{align*}
\]

Thus, it is obvious that \( a < 0 \) and \( b = 0 \) and hence by Theorem 2 of [19] there are locally asymptotically stable endemic equilibria near \( r_0 \) for \( 0 < \theta < \sigma \) where \( \sigma > 1 \). Therefore, the bifurcation as \( r_0 \) passes through 1 is a forward bifurcation.

Hence, the DFE is locally asymptotically stable if \( r_0 \) is less than one (i.e., \( \sigma < 0 \)), if \( r_0 \) is greater than one then the DFE is unstable and there is a locally asymptotically stable EE near the DFE.

### III. NUMERICAL SIMULATIONS AND DISCUSSIONS

#### A. Estimation of Parameters Used in the Model

To illustrate the various theoretical results already discussed, the model is simulated using parameter values/ranges shown in Table 1. Choice of numerical values for the model's parameter values is based upon published data on the transmission dynamics of HIV in one of the major cities, Delhi of India. All population sizes are measured here in thousands. Since AIDS is a sexually transmitted disease, we restrict my analysis to the population that is age 15 or older.

The life expectancy of males after 15 years of age is 55.6 years (so that \( \lambda_1 = 0.028 \) per year) and the life expectancy of females after 15 years of age is 58.8 years (so that \( \lambda_2 = 0.021 \) per year) [13]. The population size in reality is assumed equivalent to the population size at DFE (i.e., equal to \( N_i \) for \( i = 1, 2 \) and 3). Since the population size for males, females and FSWs in thousands are 5201, 4115 and 35 [13],[14]) then we have \( N_i = 5201 \) for \( i = 1, 2, 3 \).

Thus, the rate of HIV transmission from women to men is 0.0011-0.002 per unprotected sexual contact [20], so that \( \beta_1 = 0.0011, 0.002 \). The rate of HIV transmission from men to women is 0.0005-0.0016 per unprotected sexual contact [21].

Furthermore, for this population, the expected lifespan after diagnosis with AIDS is 20 months [15], so that \( \mu = 0.083 \) per year. The rate of HIV transmission from women to men is 0.0011-0.002 per unprotected sexual contact [20], so that \( \beta_1 = 0.0011, 0.002 \). The rate of HIV transmission from men to women is 0.0005-0.0016 per unprotected sexual contact [21].
so that $\beta_1(0.00005, 0.0016)$. In Delhi the average number of client contacts of FSWs is 1440 per year [14], so that $c_1 = 1.440$ per year. Because every FSW-client contact involves a client, the number of FSWs multiplied by their average number of client contacts per year must equal the average number of clients multiplied by their number of FSWs visit each year. Thus, we get $\bar{c}_s = \frac{c_1}{\bar{c}_1}$, $\bar{c}_2 = 0.00969$. The other parameters which are not estimated from data are taken as follows: $c_1 = 0.1$, $c_2 = 4115$, $c_3 = 0.0079$, $\phi_1 = 0$, $\phi_2 = 0$ and $\phi_3 = 0$.

The population of Delhi is approximately 9351 (age 15+) [13] where susceptible males and females are 5201 and 4115 according to reference [13] and susceptible FSWs are 35 according to reference [14] in 2001. HIV infected males, females and FSWs are 16,830, 8,896 and 2,334 respectively in Delhi [11, 13, 14]. It is assumed that the total number of population who developed AIDS in Delhi at 2001 is zero (so that the initial values of $M_j, F_j$ and $P_j$ are zero).

### B. Some Important Diagrams for the Model

In this section, we present plots for the model for various parameter sets. Fig. 2 shows solutions limiting to the DFE of the model as female-male and FSW-male coital contact rate per thousand per year are assumed here too low (i.e., $c_1 = 1.440$ and $c_2 = 0.00969$) whereas female-male coital contact rate per thousand per year is taken by assumption (i.e. $c_1 = 0.1$ and $c_2 = 0.0079$). Here, $R_0 = 4.5016$ and the disease persists, limiting to the EEP. Note that the final total population size is a lot smaller than the initial population size. So, HIV/AIDS has the potential to cause a population crash if all the parameter values remain constant. Fig. 5 depicts the combined effect of female-male sexual contact rate per year ($c_1$) and FSW-male sexual contact rate per year ($c_2$) on the basic reproduction number $R_0$. This contour plot shows a marked increase in $R_0$ with increasing both the contact rates for females and FSWs. Low rates of female-male and FSW-male unprotected sexual contact would be helpful to avoid the epidemic of AIDS (achieve $R_0 < 1$).

### TABLE I: DESCRIPTION AND ESTIMATION OF PARAMETERS FOR THE HIV TRANSMISSION MODEL

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
<th>Estimated value/range</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\lambda_1$</td>
<td>Recruitment rate of susceptible males into the community (in thousands)</td>
<td>92.58/year</td>
</tr>
<tr>
<td>$\lambda_2$</td>
<td>Recruitment rate of susceptible females into the community (in thousands)</td>
<td>69.95/year</td>
</tr>
<tr>
<td>$\lambda_3$</td>
<td>Recruitment rate of susceptible FSWs into the community (in thousands)</td>
<td>0.595/year</td>
</tr>
<tr>
<td>$\mu_1$</td>
<td>Average life expectancy for males</td>
<td>55.6 years</td>
</tr>
<tr>
<td>$\mu_2$</td>
<td>Average life expectancy for females</td>
<td>58.8 years</td>
</tr>
<tr>
<td>$\mu_3$</td>
<td>Average life expectancy for FSWs</td>
<td>58.8 years</td>
</tr>
<tr>
<td>$\mu_4$</td>
<td>Average life expectancy after diagnosis with AIDS</td>
<td>1.6 years</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>Female to male transmission probability per unprotected sexual contact</td>
<td>0.00155</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>Male to female transmission probability per unprotected sexual contact</td>
<td>0.000105</td>
</tr>
<tr>
<td>$\tau_1$</td>
<td>Average incubation period for males</td>
<td>10 years</td>
</tr>
<tr>
<td>$\tau_2$</td>
<td>Average incubation period for females</td>
<td>10 years</td>
</tr>
<tr>
<td>$\tau_3$</td>
<td>Average incubation period for FSWs</td>
<td>10 years</td>
</tr>
<tr>
<td>$\sigma_1$</td>
<td>The rate at which infected males withdraw from risk behaviour</td>
<td>0</td>
</tr>
<tr>
<td>$\sigma_2$</td>
<td>The rate at which infected females withdraw from risk behaviour</td>
<td>0</td>
</tr>
<tr>
<td>$\sigma_3$</td>
<td>The rate at which infected FSWs withdraw from risk behaviour</td>
<td>0</td>
</tr>
<tr>
<td>$c_1$</td>
<td>Female-male unprotected sexual contact rate</td>
<td>[0.0001, 1.1] /year</td>
</tr>
<tr>
<td>$c_2$</td>
<td>FSW-male unprotected sexual contact rate</td>
<td>1.440/year</td>
</tr>
<tr>
<td>$c_3$</td>
<td>Male-female unprotected sexual contact rate</td>
<td>[0.000079, 0.0079] /year</td>
</tr>
<tr>
<td>$c_4$</td>
<td>Male-FSW unprotected sexual contact rate</td>
<td>0.00969/year</td>
</tr>
</tbody>
</table>

Fig. 3 delineates the same solution graph as Fig. 2 where log scale is used along Y-axis. Fig. 4 represents solutions limiting to the EE of the model as FSW-male coital contact rate per thousand per year is taken as in reference [14] (i.e., $c_1 = 1.440$ and $c_2 = 0.00969$) whereas female-male coital contact rate per thousand per year is taken by assumption (i.e. $c_1 = 0.1$ and $c_2 = 0.0079$). Here, $R_0 = 4.5016$ and the disease persists, limiting to the EEP. Note that the final total population size is a lot smaller than the initial population size. So, HIV/AIDS has the potential to cause a population crash if all the parameter values remain constant. Fig. 5 depicts the combined effect of female-male sexual contact rate per year ($c_1$) and FSW-male sexual contact rate per year ($c_2$) on the basic reproduction number $R_0$. This contour plot shows a marked increase in $R_0$ with increasing both the contact rates for females and FSWs. Low rates of female-male and FSW-male unprotected sexual contact would be helpful to avoid the epidemic of AIDS (achieve $R_0 < 1$).

In particular, if the female-male sexual contact rate exceeds 68.4 per thousand per year (i.e., $c_1 > 0.0064$) or FSW-male sexual contact rate exceeds 2600 per thousand per year (i.e., $c_2 > 2.6$), then $R_0 > 1$ and HIV/AIDS will invade the population.

Furthermore, Fig. 6, the bifurcation diagram for the model is represented using $c_1 \in (0.00004, 0.00178)$ and all other parameter values are as in Table 1, which shows that the disease-free and endemic equilibria exchange stability when the basic reproduction number $R_0$ is 1. We note that $c_1$ is varied, a forward bifurcation occurs rather than a backward bifurcation. Note that $\mu = -0.0034$ (i.e., negative). Thus, for $R_0[c_1] < 1$, the DFE is stable whereas for $R_0[c_1] > 1$, the DFE is unstable and the EEP is stable.
CONCLUSIONS

In the model, we assess the potential impact of the presence of high-risk core group of people (i.e., FSWs) in increasing the spread and the persistency of AIDS epidemic in a heterosexual community like India. We intensively analyze the model to investigate the existence and stability of associated equilibria. Numerical simulations are carried out using reasonable sets of parameter values to assess the impact of unprotected coital contact rates between males and FSWs. We show, for this model, that the unprotected sexual interaction with FSWs is a major factor to occurring the HIV/AIDS epidemic in the community.

This model of HIV transmission has asymptotically stable disease-free equilibrium whenever the basic reproduction number is less than unity. When this number is greater than one, there is a unique endemic equilibrium. The model shows that there is a possibility of a population collapse if all the parameter values remain constant (i.e., no initiatives are taken for changing the behavioral parameters). These models can contribute to the planning of preventive procedures in case of FSW-driven sexual behavior in India. Behavioral parameters can also help public health planning. For example, lowering the values of parameters such as FSW-male unprotected sexual contact rate \(c_f\) for the model would be helpful for controlling the AIDS epidemic. Overall, this work shows that the HIV/AIDS epidemic would not occur in a heterosexual community, such as the Indian community if proper steps (for example, condom promotion for FSWs, advertisement for AIDS awareness etc.) are taken by government and non-government organizations and are implemented effectively.

This study can be extended incorporating infection distributed in a family setup for example when a married male carries sexual relationship with FSW and his wife.

CONFLICT OF INTEREST

The authors declare no conflict of interest.
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REFERENCES

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