Spread of HIV: A Mathematical Model

Anulekha Tapadar¹ & Koushik Ghosh²

¹Department of Mathematics
Dr. B.C. Roy Engineering College
Jemua Road, Fuljhore
Durgapur-713 206
INDIA
Email: anulekha.ju@gmail.com

²Department of Mathematics
University Institute of Technology
University of Burdwan
Golapbag (North)
Burdwan – 713 104
INDIA
Email: koushikg123@yahoo.co.uk

Abstract: Each year a large number of people all over the world die from HIV/AIDS. Although there are many complicating factors behind the spread of HIV, we still believe that relevant mathematical models can provide a good insight of the dynamics of the spread of it. If we can provide a satisfactory profile of this dynamics it will certainly help government officials to make timely remedial actions. In the present work we have established a mathematical model of epidemiology for the spread of HIV. We have made a search for equilibrium points for the system and discussed about their stabilities. Efforts have been made to find the solution of the proposed system. On the basis of extensive analysis relevant comments are made on mutual co-existence of the group infected by HIV and the group not infected by that.

Introduction

The Human Immunodeficiency Virus or HIV belongs to the family of Retroviruses, whose genetic material is RNA. In 1984, researchers discovered the primary causative viral agent, the human immunodeficiency virus type 1 (HIV-1). In 1986, a second type of HIV, called HIV-2 was discovered in West Africa, where it may have been present decades earlier. Both HIV-1 and HIV-2 have the same modes of transmission and are associated with similar opportunistic infections. In persons infected with HIV-2, immunodeficiency seems to develop more slowly. Compared with persons infected with HIV-1 those with HIV-2 are less infectious early in the course of infection. [1]

HIV is transmitted by direct inoculation during intimate and unsafe sexual contact, especially associated with the mucosal trauma of receptive rectal intercourse; transfusion of contaminated blood or blood products; sharing of contaminated needles or transplacental or postpartum transmission from an infected mother to the fetus (by cervical or blood contact at delivery and in breast milk). HIV is not transmitted by casual household or social contract [2].
Human CD4⁺T lymphocytes, macrophages, microglial, dendritic and Langerhans cells are believed to be targets for HIV-1 infection. The main target is CD4⁺T helper cell; a type of T cell. T cells are an important part of the immune system because they help to facilitate the body’s response to many common but potentially fatal infections. Without enough T-cells the body’s immune system is unable to defend itself against many infections. By ways that are not yet completely understood, HIV’s life cycle directly or indirectly causes a reduction in the number of T-cells in the body, eventually resulting in an increased risk of infections. Over time, there are not enough T-cells to defend the body. At this stage, a person is said to have Acquired Immunodeficiency Syndrome, or AIDS, and becomes susceptible to infections that a healthy immune system could deal with. The time in between the first infection and initiation of antibody synthesis is usually 6-12 weeks. The median time to receive an AIDS diagnosis among those infected with HIV is 7-10 years [1]. We here present a table which depicts the worldwide devastating profile of HIV/AIDS epidemic: [3]

<table>
<thead>
<tr>
<th>Group of People (in 2006)</th>
<th>Estimate (in million)</th>
</tr>
</thead>
<tbody>
<tr>
<td>People living with HIV/AIDS</td>
<td>39.50</td>
</tr>
<tr>
<td>Adults living with HIV/AIDS</td>
<td>37.20</td>
</tr>
<tr>
<td>Children living with HIV/AIDS</td>
<td>2.30</td>
</tr>
<tr>
<td>Women living with HIV/AIDS</td>
<td>17.70</td>
</tr>
<tr>
<td>People newly infected with HIV</td>
<td>4.33</td>
</tr>
<tr>
<td>Adults newly infected with HIV</td>
<td>3.80</td>
</tr>
<tr>
<td>Children newly infected with HIV</td>
<td>0.53</td>
</tr>
<tr>
<td>AIDS deaths</td>
<td>2.98</td>
</tr>
<tr>
<td>Adult AIDS deaths</td>
<td>2.60</td>
</tr>
<tr>
<td>Child AIDS deaths</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Bobashev et al. [4] developed a mathematical model of the spread of HIV by sharing of contaminated needles. In the present work we have worked a larger perspective by constructing a mathematical model of the spread of HIV taking into account unsafe sex, transfusion of contaminated blood or blood products or sharing of contaminated needles and birth and death in the group not infected by HIV as well as in the group infected by HIV.

**Formulation of the Model**

To construct the model we assume the following:

- \( I \) = Number of persons carrying HIV at time \( t \)
- \( S \) = Number of persons not carrying HIV at time \( t \)
- \( P_1 \) = Mass of the population, initially not infected by HIV, going for unsafe sex
- \( P_2 \) = Mass of the HIV infected population going for unsafe sex
- \( P_3 \) = Mass, initially not infected but getting infected by HIV due to transfusion of contaminated blood or blood products or by sharing of contaminated needles
- \( b \) = Birth rate per individual in the group not infected by HIV
\( d = \) Death rate per individual in the group not infected by HIV  
\( b' = \) Birth rate per individual in the group of HIV infected  
\( d'' = \) Death rate per individual in the group of HIV infected.

We consider,

\[
\begin{align*}
\frac{dS}{dt} &= -\alpha P_1 P_2 - \beta P_3 + bS - dS \\
\frac{dI}{dt} &= \alpha P_1 P_2 + \beta P_3 + b'I - d'I
\end{align*}
\]

where \( \alpha, \beta \) are the parameters characterizing the spread of HIV.

Again we have,

\[
\begin{align*}
P_1 &= Q_1 S \\
P_2 &= Q_2 I \\
P_3 &= Q_3 S
\end{align*}
\]

where \( Q_1, Q_2 \) and \( Q_3 \) are corresponding proportionality factors such that \( Q_i \in (0, 1) \) for \( i=1,2,3 \).

Initially let there be ‘\( n \)’ individuals not infected by HIV and ‘\( a \)’ individuals infected by HIV, i.e., \( S(0) = n, I(0) = a \).

So we get,

\[
\begin{align*}
\frac{dS}{dt} &= -\alpha Q_1 S Q_2 I - \beta Q_3 S + (b - d')S \\
\frac{dI}{dt} &= \alpha Q_1 S Q_2 I + \beta Q_3 S + (b' - d')I
\end{align*}
\]

with \( S(0) = n, I(0) = a \)

or,

\[
\begin{align*}
\frac{dS}{dt} &= -pSI - qS + rS \\
\frac{dI}{dt} &= pSI + qS + r'I
\end{align*}
\]

with \( S(0) = n, I(0) = a \)
where \( p = \alpha Q_1Q_2, \) \( q = \beta Q_3, \) \( r = b - d \) and \( r' = b' - d'. \)

**Search for Equilibrium Points**

For equilibrium point we must have,

\[
\begin{align*}
\frac{dS}{dt} &= 0 \quad \text{and} \quad \frac{dI}{dt} = 0 \\
\text{i.e.,} \quad -pSI - qS + rS &= 0 \\
psI + qS + r'I &= 0
\end{align*}
\]

Solving this system we get the points of equilibrium as \((0, 0)\) and \( \left(\frac{r'(q-r)}{pr}, \frac{r-q}{p}\right) \).

The second equilibrium point exists only if \( r > q \) and \( r' < 0 \) to maintain the non-negative identity of both \( S \) and \( I \).

**Analysis of Stability of Equilibrium Points**

An equilibrium point is considered to be stable if the system always returns to it after small disturbances. If the system moves away from the equilibrium after small disturbances, then the equilibrium is unstable. The number of eigen values is equal to the number of state variables. In our case there will be 2 eigen values. If both the eigen values are real then the equilibrium point is said to be a knot and if they are conjugate complex numbers then it is said to be a focus. If both the eigen values are positive then the equilibrium point is an unstable knot; if both the eigen values are negative then it is a stable knot and if one is positive and the other is negative then it is a saddle point. For complex eigen values if the real part is positive then the equilibrium point is an unstable focus and if the real part is negative then it is a stable focus.

For the present system we have the characteristic equation as

\[
\begin{vmatrix}
-pI - q + r - \lambda & -pS \\
pI + q & pS + r' - \lambda
\end{vmatrix} = 0
\]

For the equilibrium point \((0, 0)\) we have

\[
\begin{vmatrix}
-q + r - \lambda & 0 \\
q & r' - \lambda
\end{vmatrix} = 0
\]

which gives, \( \lambda = r', \ r-q \). If \( r > q, \ r' > 0 \) then both the eigen values are positive. In this case, the equilibrium point \((0, 0)\) is an unstable knot. If \( r > q, \ r' < 0 \) then it is a saddle point. If \( r < q, \ r' > 0 \) then it is again a saddle point. If \( r < q, \ r' < 0 \) then it is a stable knot.
In the domain \( r > q \), \( r' < 0 \) for the equilibrium point \( \left[ \frac{r'(q-r)}{pr}, \frac{r-q}{p} \right] \) we have the characteristic equation as

\[
\begin{vmatrix}
-\lambda & -r'(q-r) \\
r & r'q - \lambda \\
\end{vmatrix} = 0
\]

which gives

\[
\lambda = \frac{r'q \pm \sqrt{\frac{r'^2 q^2}{r^2} + 4r'(r-q)}}{2}
\]

If the discriminant \( \frac{r'^2 q^2}{r^2} + 4r'(r-q) \geq 0 \) then both the eigen values are negative and in that case it represents a stable knot. Again if the discriminant \( \frac{r'^2 q^2}{r^2} + 4r'(r-q) < 0 \) then the equilibrium point represents a stable focus.

**Search for Solution of the System**

We consider

\[
S = n + e_1 t + e_2 t^2 + e_3 t^3 + \ldots
\]

and

\[
I = a + f_1 t + f_2 t^2 + f_3 t^3 + \ldots
\]

remembering \( S(0) = n \) and \( I(0) = a \).

Then, \( \frac{dS}{dt} = e_1 + 2e_2 t + 3e_3 t^2 + \ldots \) \( \) and, \( \frac{dI}{dt} = f_1 + 2f_2 t + 3f_3 t^2 + \ldots \) \( \)

From the system of equations (4) we get,

\[
\frac{dS}{dt} + \frac{dI}{dt} = rS + r'I
\]

From (8) using (6) and (7) we get,
\[ e_1 + f_1 = rn + r'a \]
\[ 2(e_2 + f_2) = re_1 + r'f_1 \]
\[ 3(e_3 + f_3) = re_2 + r'f_2 \]

(9)

and so on.

Again using (6) and (7) in the first equation of (4) we get,

\[ e_1 = -pna - qn + rn \]
\[ e_2 = \frac{1}{2}[re_1 - qe_1 - p(nf_1 + ae_1)] \]
\[ e_3 = \frac{1}{3}[re_1 - qe_2 - p(ae_2 + nf_2 + e_1f_1)] \]

(10)

and so on.

From (9) and (10),

\[ f_1 = pna + qn + r'a \]
\[ e_2 = \frac{1}{2}[q^2n + r^2n - 2qrn - pr'an - 2aopr + a^2p^2n - ap^2n^2 - pqn^2] \]
\[ f_2 = \frac{1}{2}[aprn + qrn + 2pr'an + qr'n + r^2a - q^2n - 2pqan - a^2p^2n + ap^2n^2 + pqn^2] \]

(11)

Finally we can write the solution as

\[ S = n + (-pna - qn + rn) t + \frac{1}{2}[(q - r)^2n + 2pqan - pr'an - 2aopr + a^2p^2n
- ap^2n^2 - pqn^2] t^2 + \ldots \]

(12)

and

\[ I = a + (pna + qn + r'a) t + \frac{1}{2}[aprn + qrn + 2pr'an + qr'n + r^2a - q^2n
- 2pqan - a^2p^2n + ap^2n^2 + pqn^2] t^2 + \ldots \]

Mutual Co-existence of the HIV not-infected and HIV infected individuals

From (4) we get,

\[ \frac{dI}{dS} = \frac{pSI + qS + r'I}{-pSI - qS + rS} \]

(13)

Here we restrict ourselves in the domain \( r > q, r' < 0 \). In most of the cases it is expected that the birth rate per individual is less than the death rate per individual in the group of HIV infected people. So it looks good to assume \( r' < 0 \). Also the net rate of increase (rate of birth-rate of death) per individual in the group not infected by HIV is expected to be greater than the rate per individual of getting infected by HIV due to transfusion of contaminated blood or blood products.
or by sharing of contaminated needles in the same group. So, $r > q$ looks consistent in most of the cases.

**Case A:** $\frac{dI}{dS} > 0$

**Subcase (i):** $\frac{dI}{dt} > 0$ and $\frac{dS}{dt} > 0$ i.e., $pSI + qS + r'I > 0$ and $-pSI - qS + rS > 0$. In this case we have, $I < \frac{r-q}{p}$. We consider $I = \frac{r-q}{p} - h$ for $h > 0$. From the inequality $pSI + qS + r'I > 0$ we have,

$$S > \frac{1}{(r-ph)} \left[ r'h - \frac{r'(r-q)}{p} \right]$$

provided $h < \frac{r}{p}$. But as $I > 0$, $f < \frac{r-q}{p} < \frac{r}{p}$. So, the above range for $S$ is a consistent range for $S$ in this case.

We take, $f(h) = \frac{1}{r-ph} \left[ r'h - \frac{r'(r-q)}{p} \right]$

Therefore, $f'(h) = \frac{r'q}{(r-ph)^2} < 0$ since $r' < 0$

So, $f(h)$ is a decreasing function of $h$. Hence $f(h) < f(0)$ for $h > 0$, i.e.,

$$\frac{1}{r-ph} \left[ r'h - \frac{r'(r-q)}{p} \right] < \frac{r'(q-r)}{pr}$$

Hence in this case we fail to get any specific range for $S$.

**Subcase (ii):** $\frac{dI}{dt} < 0$ and $\frac{dS}{dt} < 0$, i.e., $pSI + qS + r'I < 0$ and $-pSI - qS + rS < 0$. In this case we have $I > \frac{r-q}{p}$. We consider $I = \frac{r-q}{p} + h$ for $h > 0$. From the inequality $pSI + qS + r'I < 0$ we have,

$$S < \frac{1}{(r+ph)} \left[ \frac{r'(q-r)}{p} - r'h \right]$$

We take $g(h) = \frac{1}{(r+ph)} \left[ \frac{r'(q-r)}{p} - r'h \right]$

Therefore, $g'(h) = -\frac{r'q}{(r+ph)^2} > 0$ since $r' < 0$. 
So, \( g(h) \) is an increasing function of \( h \). Hence \( g(h) > g(0) \) for \( h > 0 \), i.e.,

\[
\frac{1}{(r + ph)} \left[ \frac{r'(q - r) - r'h}{p} \right] > \frac{r'(q - r)}{pr}
\]

Hence in this case also we fail to get any specific range for \( S \).

**Case B:** \( \frac{dI}{dS} < 0 \)

**Subcase (i):** \( \frac{dI}{dt} < 0 \) and \( \frac{dS}{dt} > 0 \), i.e., \( pSI + qS + r'I < 0 \) and \( -pSI - qS + rS > 0 \). In this case, we have \( I < \frac{r - q}{p} \) and we take \( I = \frac{r - q}{p} - h \) where \( h > 0 \). From the inequality \( pSI + qS + r'I < 0 \) we get

\[
S < \frac{1}{(r + ph)} \left[ r'h - \frac{r'(r-q)}{p} \right]
\]

provided \( h < \frac{r}{p} \). But as \( I > 0 \), \( h < \frac{r - q}{p} < \frac{r}{p} \). So the above range for \( S \) is a consistent range for \( S \) in this case. By previous argument in Case A : subcase (i) we have

\[
\frac{1}{(r + ph)} \left[ r'h - \frac{r'(r-q)}{p} \right] < \frac{r'(q - r)}{pr}
\]

Hence, \( S < \frac{r'(q - r)}{pr} \)

**Subcase (ii):** \( \frac{dI}{dt} > 0 \) and \( \frac{dS}{dt} < 0 \), i.e., \( pSI + qS + r'I > 0 \) and \( -pSI - qS + rS < 0 \). In this case

\[
I > \frac{r - q}{p}
\]

and we take \( I = \frac{r - q}{p} + h \) where \( h > 0 \). From the inequality \( pSI + qS + r'I > 0 \). We get,

\[
S > \frac{1}{(r + ph)} \left[ \frac{r'(q - r)}{p} - r'h \right].
\]

Now by previous argument in case A : subcase (ii) we have,

\[
\frac{1}{(r + ph)} \left[ r'(q - r) - r'h \right] > \frac{r'(q - r)}{pr}
\]
Hence, \( S > \frac{r'(q-r)}{pr} \).

Keeping in view all the above facts we can have the following table:

<table>
<thead>
<tr>
<th>Sign of ( dI/dS )</th>
<th>Serial No.</th>
<th>Sign of ( dS/dt )</th>
<th>Sign of ( dI/dt )</th>
<th>Range of ( S )</th>
<th>Range of ( I )</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>1</td>
<td>+</td>
<td>+</td>
<td>No specific range for ( S )</td>
<td>( I &lt; \frac{r-q}{p} )</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-</td>
<td>-</td>
<td>No specific range for ( S )</td>
<td>( I &gt; \frac{r-q}{p} )</td>
</tr>
<tr>
<td>-</td>
<td>3</td>
<td>+</td>
<td>-</td>
<td>( S &lt; \frac{r'(q-r)}{pr} )</td>
<td>( I &lt; \frac{r-q}{p} )</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>-</td>
<td>+</td>
<td>( S &gt; \frac{r'(q-r)}{pr} )</td>
<td>( I &gt; \frac{r-q}{p} )</td>
</tr>
</tbody>
</table>

Situation 3 is analogous to the model where the group not infected by HIV increases the number, and the group infected by HIV decreases its number. On the other hand, situation 4 is very alarming where the group not infected by HIV reduces its number as well as the group infected by HIV expands with time. This situation can be termed as critically epidemic situation.

**Conclusion**

The present work analyses the localized phenomenon of the spread of HIV. All the parameters involved in this extensive mathematical analysis are local in nature. Although, these parameters are not really confined in the micro-domain of any urban or rural area inside any specific country but we cannot think of their range bigger than a state or a small country. Unlike different infectious diseases HIV/AIDS is not limited within any specific geographical region. In reality, it is a global problem and it is very much needed to concentrate on the modelling of geographical distribution of HIV/AIDS. Oluwoye [5] used the spatial interaction and gravity models to understand and predict the rate of HIV infection, location of activities, spread of HIV infection and movement of HIV infected people. The flows of people between rural areas, villages, cities and countries are forms of spatial interaction that are central to disease transmission. A good fusion of the spread of HIV in local domains and the spatial interaction between different local units can be a very interesting future study in the process of understanding the global dynamics of the spread of HIV/AIDS.

Recently one survey was performed by the Department of Anthropology, Harvard University and it was reported that in the Sub-Saharan countries in Africa continent the more is the Muslim population the less is the rate of HIV infection. In the Muslim-dominated countries in Middle and West Asia as well as in the South and South-East Asia the graph of HIV infection is significantly decreasing. These reports in turn suggest that the process of circumcision in male provides protection against HIV acquisition. Talukdar et al. [6] made a survey among the homeless Muslim (circumcised) and Hindu (uncircumcised) men aged 18-49 years in Kolkata, India, total 485 in number (105 Muslims and 380 Hindus), to compare the risk of HIV and sexually transmitted diseases. They found the odds ratio for HIV among Muslims (circumcised) compared to Hindus (uncircumcised) is 0.43 (95% confidence interval 0.29-0.67). This survey also suggests that the rate of HIV infection is less than 1% in the group of circumcised men who
usually have more than one partner and visit to commercial sex workers and that rate is more than 3% in the group of uncircumcised men going for unsafe sex. These results suggest that a biological effect of circumcision protects against HIV infection. This beneficial effect of circumcision should be communicated to high-risk groups, as well as to the general population and future mathematical models about the dynamics of the spread of HIV should ensure the presence of the protective effect of circumcision in them.

References


