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# Modelling and Analysis of HIV/AIDS Menace using Differential Equations

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## ABSTRACT

Mathematical models play important role in understanding the population dynamics of HIV/AIDS. In this study, a mathematical model is formulated for a community which has the structure of two classes with different levels of sexual activity – one is high activity group that include commercial sex workers and their male customers; and the other is low activity group. These two groups are further divided into two sub-groups as HIV infected and unaware, and HIV infected and aware after screening. It is assumed that people in low activity group when become aware, do not spread infection any more by means of either not participating in sexual activity at all or by taking some preventive measures. The model is analysed using stability theory of differential equations, numerical simulation and sensitivity analysis.

## Keywords

Mathematical Model; HIV/AIDS; Numerical Simulation; Stability.

## Academic Discipline And Sub-Disciplines

Mathematical Modelling of Epidemics

## TYPE (METHOD/APPROACH)

Modeling and simulation of the dynamics of infectious diseases using differential equation.

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## INTRODUCTION

AIDS (Acquired Immune Deficiency Syndrome), caused by HIV (Human Immuno-deficiency Virus), has become one of the world's most serious health and development challenges. Out of 340 lakh global AIDS cases, 48 lakh cases are in Asia and India has the highest (49%) number of people affected by this deadliest disease in the continent as revealed by UNAIDS report.

Due to lack of knowledge about AIDS people have fear in their mind against the AIDS patients and due to fear of social boycott, people try to hide their disease status.

After initial infection, the virus becomes less active in the body, although it is still present. During this period many people do not have any symptoms of HIV infection. This period is called latency phase. When person's immune system is severely damaged by the virus and has difficulty in fighting diseases, then at this stage the person is said to have AIDS. Thus AIDS is the late stage of HIV infection. Before development of certain medications, people with HIV could progress to AIDS in just a few years. Currently people can live much longer – even decades – with HIV before they develop AIDS.

In this paper, a mathematical model is proposed for predicting the epidemiological status of disease in long term and to find the right parameters to be worked upon for controlling the spread of the disease.

Mathematical models have been used since decades for the better understanding of HIV/AIDS scenario and for applying control measures. From the very beginning in 1989, L. Johnson gave a note on basics of HIV/AIDS modelling [2]. J.M. Hymen used many models from simple to complex and addressed various risk factors [1]. A.S.R. Srinivasa Rao et al. presented a simple AIDS epidemic model and analysed it for India [3], in [4] he applied convolution to understand the prevalence. Y. Hsieh et al. highly structured the population into various classes on the basis of their activity type, gender etc. [6]. R. Naresh et al. divided the infected class into two parts on the basis of their activity levels [7]. N.J.D. Nagelkerke modelled the impact of various intervention methods and concluded that it is possible to have drug-resistant HIV after some years [8]. S.D. Hove-Musekwa et al. included screening while modelling [9]. For analysis we refer [10] - [14]. For data, we used [5], [7], and [15] - [19].

## History/Symptoms/Transmission of HIV/AIDS

Scientists believe HIV came from a particular king of chimpanzee in Western Africa. Humans probably came in contact with HIV when they hunted and ate infected animals.

Human Immunodeficiency Virus is lot like other viruses, including those that cause the common cold. But there is an important difference – over the time, our immune system can clear most viruses out of our body but not HIV. It belongs to an unusual group of viruses called retroviruses found in monkeys and apes, sheep and goats.

There are two main strains of HIV i.e. HIV-1 and HIV-2. HIV-1 has caused the majority (80%) of infections and AIDS cases and HIV-2 is contracted in Africa and in some parts of India.

Once the virus enters the body, it attacks CD4+ type of white blood cells (WBCs) in blood and gradually kills them. These cells help us to fight against various infections. Once they are destroyed, our body's resistance goes down and the person suffers from lots of infections.

Initially an HIV infected person looks completely normal and healthy. As early as 3 - months after exposure to HIV, people can experience an acute illness, often described as the "worst flu ever". This is called Acute Retroviral Syndrome (ARS) or primary HIV infection, and it is the body's natural response to HIV infection. During primary HIV infection, there are higher levels of virus circulating in the blood, which means the person can more easily transmit the virus to others. It is important to remember that not everyone gets ARS when they become infected with HIV.

The general symptoms may include fever, rashes, night-sweats, diarrhea, mouth ulcers, sudden weight loss and other opportunistic illnesses.

HIV do not survive outside the human body and therefore, can only be transmitted directly from person to person, either by sexual contact, exchange of blood or body fluids or from mother to child.

HIV incidence and dynamics varies from country to country and from region to region, depending on various risk factors. Since this disease is driven mainly by sexual transmission, the level and intensity of risk behaviour are the main determinants of the spread of the virus.

## Mathematical Model

Here, we divide the entire population N into six compartments. At time *t*, there are $S$ susceptible or HIV negatives, $H_n$ $(L_n)$ HIV positive high (low) sexual activity group who are not aware that they are infected, $H_a$ $(L_a)$ HIV positive high (low) sexual activity group who are aware that they are infected, $A$ is population with AIDS. The population dynamics among these compartments is shown in Fig.1:

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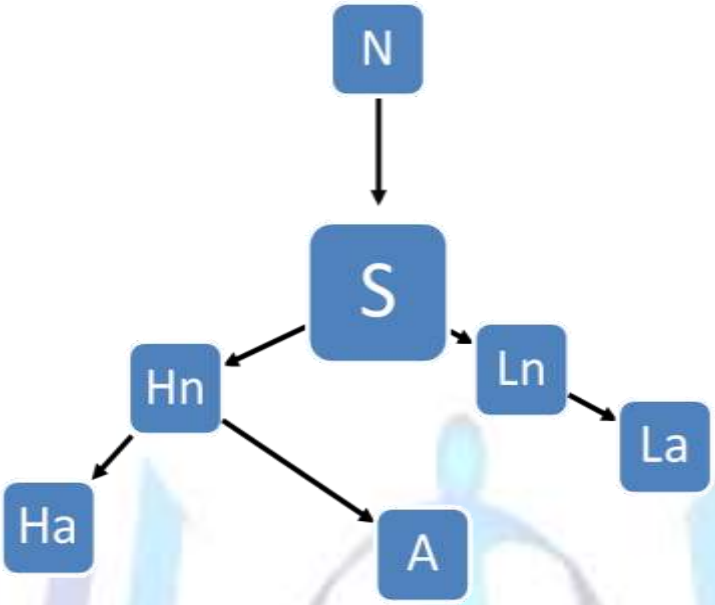


**Figure.1 Transfer diagram of HIV/AIDS**

The state variables and all the parameters are listed below:

$S$: Number of susceptible (i.e. HIV negative)

$H_n$ $(L_n)$: Number of unaware HIV positive persons with high (low) sexual activity

$H_a$ $(L_a)$: Number of highly (less) active persons who are now aware that they are HIV positive

$A$: Number of people who has developed AIDS

$B$: Recruitment rate in susceptible class

$\mu$: Natural death rate

$\delta$: Disease induced death rate

$\beta_1$ $(\beta_2/\beta_3)$: Probability of transmission of infection from an infective $H_n$ $(L_n/H_a)$ to a susceptible per contact per unit time

$c_1$ $(c_2/c_3)$: Number of contacts made by a person from $H_n$ $(L_n/H_a)$ class

$\sigma_1$ $(\sigma_2)$: Rate of unaware infective $H_n$ $(L_n)$ to become aware by screening.

$\alpha$: Rate with which all type of infective develops AIDS.

Here, we have assumed that when a person from $L_n$ class moves to $L_a$ class, it does not spread infection any more. So, the model takes the form as follows:

$$\begin{aligned}
\frac{dS}{dt} &= B-\beta_1 c_1\frac{SH_n}{N}-\beta_2 c_2\frac{SL_n}{N}-\beta_3 c_3\frac{SH_a}{N}-\mu S\\
\frac{dH_n}{dt} &= \beta_1 c_1\frac{SH_n}{N}+\beta_3 c_3\frac{SH_a}{N}-(\mu+\alpha+\sigma_1)H_n\\
\frac{dL_n}{dt} &= \beta_2 c_2\frac{SL_n}{N}-(\mu+\alpha+\sigma_2)L_n\\
\frac{dH_a}{dt} &= \sigma_1 H_n-(\mu+\alpha)H_a\\
\frac{dL_a}{dt} &= \sigma_2 L_n-(\mu+\alpha)L_a\\
\frac{dA}{dt} &= \alpha(H_n+L_n+H_a+L_a)-(\mu+\delta)A
\end{aligned} \qquad (1)$$

with $N = S + H_n + L_n + H_a + L_a + A$

Adding all the above equations, we have

$$\frac{d}{dt}(S + H_n + L_n + H_a + L_a + A) = B - \mu(S + H_n + L_n + H_a + L_a + A) - \delta A$$

or $(S + H_n + L_n + H_a + L_a + A)' \le B - \mu(S + H_n + L_n + H_a + L_a + A)$

or $N' \le B - \mu N$

Then $\limsup_{t\to\infty}(S + H_n + L_n + H_a + L_a + A) \le \frac{B}{\mu}$

So, the feasible region for the system is

$$\Lambda = \left\{(S, H_n, L_n, H_a, L_a, A) : S + H_n + L_n + H_a + L_a + A \le \frac{B}{\mu}, S > 0, H_n > 0, L_n > 0, H_a > 0, L_a \ge 0, A \ge 0\right\}$$

Let $E(\bar{S}, \bar{H}_n, \bar{L}_n, \bar{H}_a, \bar{L}_a, \bar{A})$ be the equilibrium point of the system (1) of equation. Now, we calculate basic reproduction number, $R_0$, as follows:

Since, the recruitment term B can never be zero and population cannot vanish, therefore there is no trivial equilibrium point like $(\bar{S}, \bar{H}_n, \bar{L}_n, \bar{H}_a, \bar{L}_a, \bar{A}) = (0,0,0,0,0,0)$. So, let $E(\bar{S}, \bar{H}_n, \bar{L}_n, \bar{H}_a, \bar{L}_a, \bar{A}) = (\bar{S}, 0,0,0,0,0)$. Then system (1) of equations at this point gives $\bar{S} = \frac{B}{\mu}$.

So, we can see that there is a disease free equilibrium at

$$E_0(\bar{S}, \bar{H}_n, \bar{L}_n, \bar{H}_a, \bar{L}_a, \bar{A}) = \left(\frac{B}{\mu}, 0,0,0,0,0\right)$$

Let $X' = (H_n, L_n, H_a, L_a, A, S)^T$.

Therefore, $X' = \frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X)$

where $\mathcal{F}(X) = \begin{bmatrix} \beta_1 c_1 \frac{SH_n}{N} + \beta_3 c_3 \frac{SH_a}{N} \\ \beta_2 c_2 \frac{SL_n}{N} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$

and $\mathcal{V}(X) = \begin{bmatrix} (\mu + \alpha + \sigma_1) H_n \\ (\mu + \alpha + \sigma_2) L_n \\ -\sigma_1 H_n + (\mu + \alpha) H_a \\ -\sigma_2 L_n + (\mu + \alpha) L_a \\ -\alpha(H_n + L_n + H_a + L_a) + (\mu + \delta) A \\ -B + \beta_1 c_1 \frac{SH_n}{N} + \beta_2 c_2 \frac{SL_n}{N} + \beta_3 c_3 \frac{SH_a}{N} + \mu S \end{bmatrix}$

The derivatives $D\mathcal{F}(E_0)$ and $D\mathcal{V}(E_0)$ at disease free equilibrium point $E_0$, are partitioned as

$$D\mathcal{F}(\mathrm{E}_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix} \quad \text{and} \quad D\mathcal{V}(\mathrm{E}_0) = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix}$$

where *F* and *V* are 3 × 3 matrices given by

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$$F=\begin{bmatrix}\frac{\beta_1 c_1 B}{\mu N} & 0 & \frac{\beta_3 c_3 B}{\mu N}\\ 0 & \frac{\beta_2 c_2 B}{\mu N} & 0\\ 0 & 0 & 0\end{bmatrix} \quad \text{and} \quad V=\begin{bmatrix}(\mu+\alpha+\sigma_1) & 0 & 0\\ 0 & (\mu+\alpha+\sigma_2) & 0\\ -\sigma_1 & 0 & (\mu+\alpha)\end{bmatrix}$$

$$FV^{-1}=\begin{bmatrix}\left(\frac{\beta_1 c_1 B}{\mu N(\mu+\alpha+\sigma_1)}+\frac{\beta_3 c_3 B}{\mu N(\mu+\alpha+\sigma_1)(\mu+\alpha)}\right) & 0 & \frac{\beta_3 c_3 B}{\mu N(\mu+\alpha)}\\ 0 & \frac{\beta_2 c_2 B}{\mu N(\mu+\alpha+\sigma_2)} & 0\\ 0 & 0 & 0\end{bmatrix}$$

Therefore, basic reproduction number

$R_0=\rho\left(FV^{-1}\right)$ = spectral radius of $FV^{-1}$

$$\Rightarrow R_0=\max\left(\frac{\beta_1 c_1(\mu+\alpha)+\beta_3 c_3\sigma_1}{(\mu+\alpha+\sigma_1)(\mu+\alpha)},\frac{\beta_2 c_2}{(\mu+\alpha+\sigma_2)}\right)=\frac{\beta_1 c_1(\mu+\alpha)+\beta_3 c_3\sigma_1}{(\mu+\alpha+\sigma_1)(\mu+\alpha)} \qquad (2)$$

## Stability of Disease Free Equilibrium

T The disease free equilibrium is stable if all the eigen values of the Jacobian matrix of the system (1) have negative real parts. For this, the Jacobian of the system (1) at $E_0\left(\frac{B}{\mu},0,0,0,0,0\right)$ takes the form

$$J=\begin{bmatrix}-\mu & -\frac{\beta_1 c_1 B}{\mu N} & -\frac{\beta_2 c_2 B}{\mu N} & -\frac{\beta_3 c_3 B}{\mu N} & 0 & 0\\ 0 & \frac{\beta_1 c_1 B}{\mu N}-(\mu+\alpha+\sigma_1) & 0 & \frac{\beta_3 c_3 B}{\mu N} & 0 & 0\\ 0 & 0 & \frac{\beta_2 c_2 B}{\mu N}-(\mu+\alpha+\sigma_2) & 0 & 0 & 0\\ 0 & \sigma_1 & 0 & -(\mu+\alpha) & 0 & 0\\ 0 & 0 & \sigma_2 & 0 & -(\mu+\alpha) & 0\\ 0 & \alpha & \alpha & \alpha & \alpha & -(\mu+\delta)\end{bmatrix}$$

Here,

trace ($J$) = $-\left(6\mu+4\alpha+\sigma_1+\sigma_2+\delta\right)+\frac{(\beta_1 c_1+\beta_2 c_2)B}{\mu N}$. Clearly, trace ($J$) < 0.

Now for det ($J$) to be > 0, we proceed as follows:

Expanding det ($J$), we get

$$\det(J)=-\mu(\mu+\delta)(\mu+\alpha)\begin{vmatrix}\frac{\beta_1 c_1 B}{\mu N}-(\mu+\alpha+\sigma_1) & 0 & \frac{\beta_3 c_3 B}{\mu N}\\ 0 & \frac{\beta_2 c_2 B}{\mu N}-(\mu+\alpha+\sigma_2) & 0\\ \sigma_1 & 0 & -(\mu+\alpha)\end{vmatrix}$$

$$\Rightarrow \det(J)=\mu(\mu+\delta)(\mu+\alpha)\left(\frac{\beta_2 c_2 B}{\mu N}-(\mu+\alpha+\sigma_2)\right)\left[(\mu+\alpha)\left(\frac{\beta_1 c_1 B}{\mu N}-(\mu+\alpha+\sigma_1)+\frac{\beta_3 c_3 B\sigma_1}{\mu N}\right)\right]$$

For det ($J$) > 0, after some algebraic simplification, we get the relation that

$$\beta_1 c_1(\mu+\alpha+\sigma_1)(\mu+\alpha)+\beta_3 c_3\sigma_1(\mu+\alpha+\sigma_1)<(\mu+\alpha)(\mu+\alpha+\sigma_1)^2$$

$$\frac{\beta_1 c_1(\mu+\alpha)+\beta_3 c_3\sigma_1}{(\mu+\alpha)(\mu+\alpha+\sigma_1)}<1 \quad \Rightarrow \quad R_0<1 \quad \text{(by (1))}$$

This implies that the disease free equilibrium is locally asymptotically stable if $R_0<1$ otherwise unstable.

## Endemic Equilibrium

Let the endemic equilibrium point be $E_e\left(S^*,H^*_n,L^*_n,H^*_a,L^*_a,A^*\right)\neq(0,0,0,0,0,0)$ with

$$N^*=\frac{(\mu+\alpha+\delta)B}{\mu R_0(\mu+\alpha+\delta)+\alpha\delta(R_0-1)} \tag{3a}$$

$$H^*_n=\frac{(\mu+\alpha)(R_0-1)^2}{R_0(\mu+\alpha+\sigma_1)(\mu+\alpha+\sigma_2-1)} \tag{3b}$$

$$L^*_n=\frac{(\mu+\delta)(\mu+\alpha)B(R_0-1)}{(\mu+\alpha+\sigma_2)\left[\mu R_0(\mu+\alpha+\delta)+\alpha\delta(R_0-1)\right]}-(\mu+\alpha+\sigma_1)H^*_n \tag{3c}$$

$$H^*_a=\frac{\sigma_1}{(\mu+\alpha)}H^*_n \tag{3d}$$

$$L^*_a=\frac{\sigma_2}{(\mu+\alpha)}L^*_n \tag{3e}$$

$$A^*=\frac{B\alpha(R_0-1)}{\mu R_0(\mu+\alpha+\delta)+\alpha\delta(R_0-1)} \tag{3f}$$

with $S^*=\frac{B}{\mu R_0}$. (3g)

## Local Stability of Endemic Equilibrium

For this, the Jacobian of the system (1) at $E_e\left(S^*,H^*_n,L^*_n,H^*_a,L^*_a,A^*\right)$ takes the form

$$J_E=\begin{bmatrix}
-\frac{1}{N}(\beta_1c_1H_n+\beta_2c_2L_n+\beta_3c_3H_a)-\mu & -\frac{\beta_1c_1S}{N} & -\frac{\beta_2c_2S}{N} & -\frac{\beta_3c_3S}{N} & 0 & 0\\
\frac{1}{N}(\beta_1c_1H_n+\beta_3c_3H_a) & \frac{\beta_1c_1S}{N}-(\mu+\alpha+\sigma_1) & 0 & \frac{\beta_3c_3S}{N} & 0 & 0\\
0 & 0 & \frac{\beta_2c_2S}{N}-(\mu+\alpha+\sigma_2) & 0 & 0 & 0\\
0 & \sigma_1 & 0 & -(\mu+\alpha) & 0 & 0\\
0 & 0 & \sigma_2 & 0 & -(\mu+\alpha) & 0\\
0 & \alpha & \alpha & \alpha & \alpha & -(\mu+\delta)
\end{bmatrix}$$

where all the state variables are at endemic equilibrium point given by relations (3a) – (3g).

Here,

trace ($J_E$) = $-\frac{1}{N}(\beta_1c_1H_n+\beta_2c_2L_n+\beta_3c_3H_a)+(\beta_1c_1+\beta_2c_2)\frac{S}{N}-(6\mu+4\alpha+\sigma_1+\sigma_2+\delta)$

Clearly, trace ($J_E$) < 0.

Now for det ($J_E$) to be > 0, we proceed as follows:

Expanding det ($J_E$) and placing it to be greater than zero, we get

$$\frac{\beta_2c_2L^*_n}{\mu N^*}\left(\frac{R_0S^*}{N^*}-1\right)+\frac{R_0S^*}{N^*}-(\mu+\alpha+\sigma_1)\frac{H^*_n}{\mu N^*}R_0>1$$

Substituting values of state variables at endemic equilibrium point and then after some algebraic simplifications we reach the relation

$$\frac{R_0\left[\alpha(\mu+\alpha+\sigma_2)+(\mu+\delta)\right]}{(\mu+\alpha+\sigma_2)(\mu+\alpha+\delta)}+\frac{R_0(R_0-1)}{\beta_2 c_2}<1 \qquad (4)$$

This implies that the endemic equilibrium point $E_e\left(S^*,H_n^*,L_n^*,H_a^*,L_a^*,A^*\right)$ is locally asymptotically stable if the relation (4) holds.

## Global Stability of Endemic Equilibrium

Consider the Lyapunov function

$$V=\frac{1}{2}\left(N-N^*\right)^2+m_1\left(H_n-H_n^*-H_n^*\log\frac{H_n}{H_n^*}\right)+m_2\left(H_a-H_a^*-H_a^*\log\frac{H_a}{H_a^*}\right)+\frac{m_3}{2}\left(L_n-L_n^*\right)^2+\frac{m_4}{2}\left(L_a-L_a^*\right)^2+\frac{m_5}{2}\left(A-A^*\right)^2$$

$$\dot{V}=\left(N-N^*\right)\frac{dN}{dt}+m_1\left(\frac{H_n-H_n^*}{H_n}\right)\frac{dH_n}{dt}+m_2\left(\frac{H_a-H_a^*}{H_a}\right)\frac{dH_a}{dt}+m_3\left(L_n-L_n^*\right)\frac{dL_n}{dt}+m_4\left(L_a-L_a^*\right)\frac{dL_a}{dt}+m_5\left(A-A^*\right)\frac{dA}{dt}$$

Using (1) and simplifying we get

$$\dot{V}=-\left(N-N^*\right)\left[\mu\left(N-N^*\right)-\delta\left(A-A^*\right)\right]-m_2(\mu+\alpha)\left(L_a-L_a^*\right)^2-m_3(\mu+\delta)\left(A-A^*\right)$$
$$-m_4\beta_3 c_3 H_a\left(\frac{H_n-H_n^*}{H_n}\right)\left[\left(N-N^*\right)+\left(H_n-H_n^*\right)+\left(L_n-L_n^*\right)+\left(H_a-H_a^*\right)+\left(L_a-L_a^*\right)\right]-m_5(\mu+\alpha)\frac{\left(H_a-H_a^*\right)^2}{H_a}$$

Hence $\dot{V}$ is negative. Also note that $\dot{V}=0$ if and only if $N=N^*$, $H_n=H_n^*$, $L_n=L_n^*$, $H_a=H_a^*$, $L_a=L_a^*$ and $A=A^*$. Therefore the largest compact invariant set in $\left\{(S,H_n,L_n,H_a,L_a,A)\in\Lambda:\dot{V}=0\right\}$ is the singleton set $\{E_e\}$, where $E_e$ is endemic equilibrium. Therefore, LaSalle's invariant principle implies that $E_e$ is globally asymptotically stable in Lyapunov sense.

## Sensitivity Analysis

Now we evaluate the sensitivity indices of $R_0$ to all the different parameters it depends on. These indices guide us to find the right parameter responsible for disease spread and need to be taken care of.

We use following parameter values: $\beta_1 = \beta_2 = \beta_3 = 0.47$, $\sigma1 = \sigma2 = 0.015$, $\mu = 0.02$, $\delta = 1$, $\alpha = 0.1$, $c_1 = 3$, $c_2 = 0.4$, $c_3 = 0.6$. Most of the values are taken from previous works done in Indian context. Others are derived on the basis of statistics released by some organisations. The analysis results are as follows:

**Table 1: Sensitivity Indices of $R_0$ to the parameters for the HIV/AIDS model**

| Parameter | Sign | Value |
|---|---|---|
| $\beta_1$ | + | 0.9756 |
| $\beta_3$ | + | 0.0244 |
| $c_1$ | + | 0.9756 |
| $c_3$ | + | 0.0244 |
| $M$ | - | 0.1522 |
| $A$ | - | 0.7611 |
| $\sigma_1$ | - | -0.0867 |

These results show that we need to target high activity group. The effective index of rate with which this spread infection turns out to be $\beta_1 \times c_1 = 0.9518$. Also the rate of progression from infectious class to AIDS class is an important parameter having negative impact on disease spread as generally humans after developing AIDS rarely spread infection any more.

## Numerical Simulation

Here, in order to find future trends of the disease we simulated the data with a population sample size of N = 23000. The results obtained are shown in Fig. 2.

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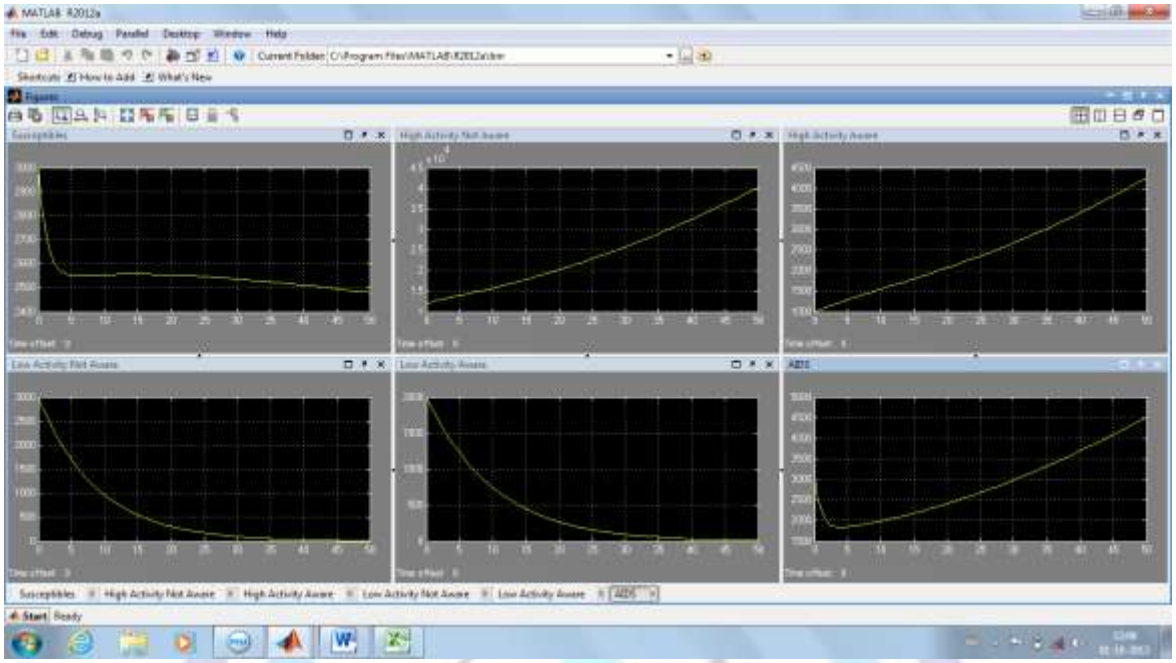

**Figure 2: Population Dynamics in Different compartments**

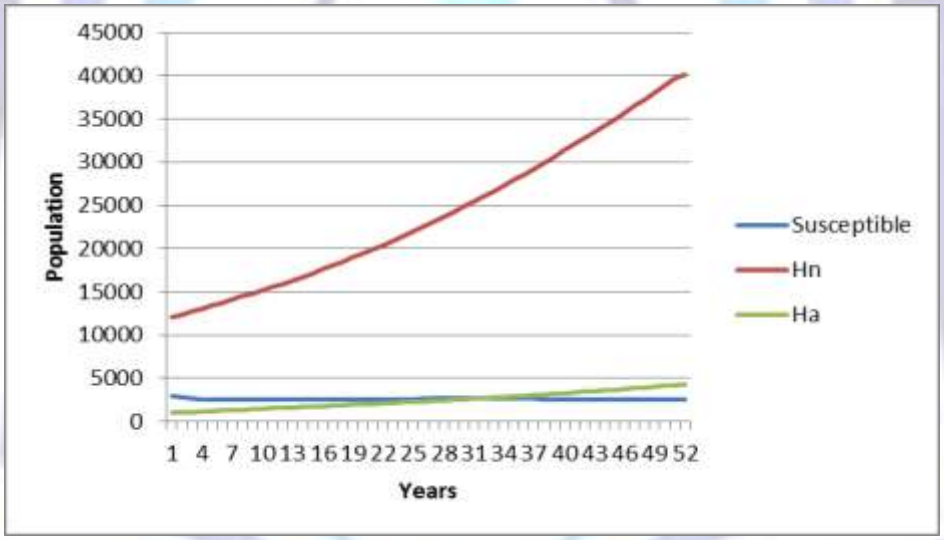


**Figure 3: Population trend in High Activity Group –Not aware verses Aware**

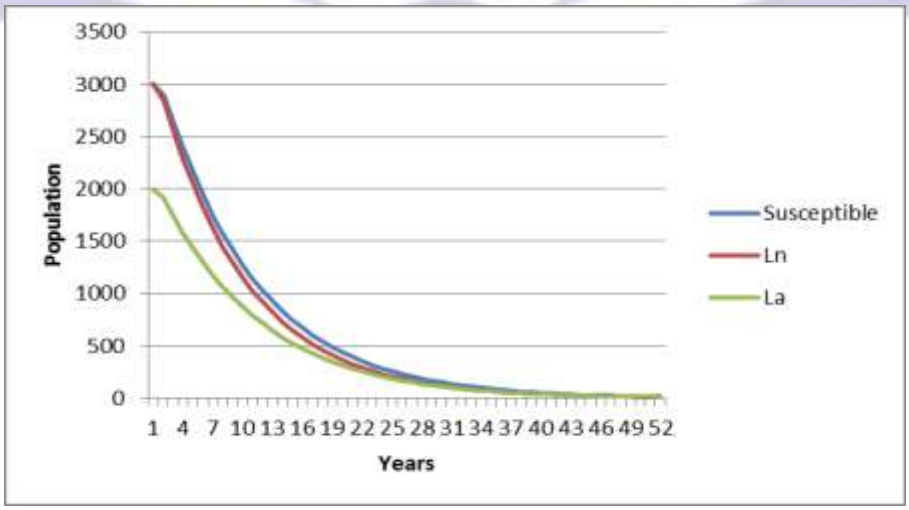


**Figure 4: Population trend in Low Activity Group –Not aware verses Aware**

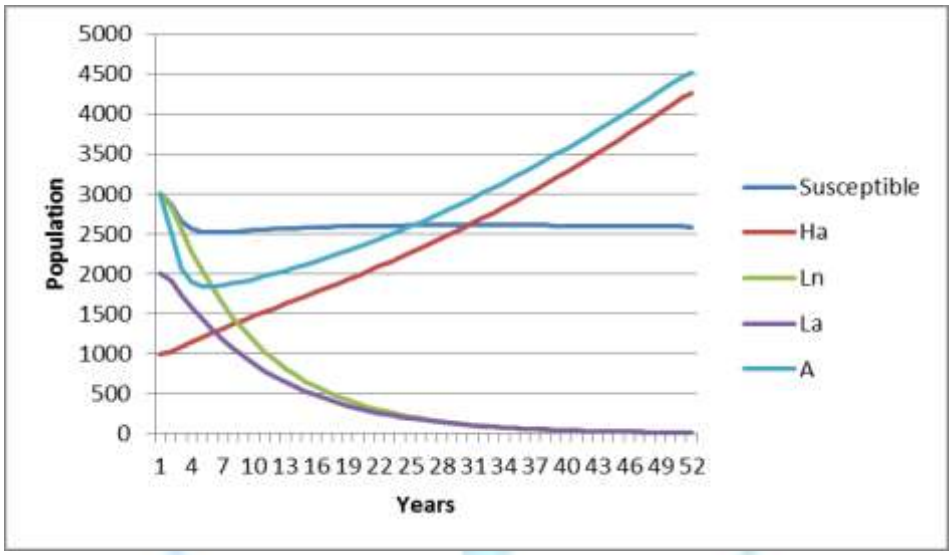


**Figure 5: Population trend in Various Compartments in Next 50 years**

## Result and Discussion

In this study, we developed a mathematical model for HIV/AIDS in order to understand the population dynamics of the disease. We divided the infected class into four groups depending on their activity levels and screening status. For starting the qualitative analysis of the model, a relation for basic reproduction number $R_0$ is established. Then the existence of steady states and their stabilities is analysed. The analysis shows that the disease free equilibrium is locally asymptotically stable if $R_0 < 1$. For local stability of endemic equilibrium we established a relation that should hold. For global stability of it, we defined a Lyapunov function and established the result accordingly. Sensitivity analysis is done using data for India. The results of sensitivity analysis show that we need to target the high activity group (figures (2) – (5)). If we make them aware about the disease and educate them for using some safety measures then it may reduce the spread of disease. The results of numerical simulation confirm the results of sensitivity analysis. The graphs predict the future dynamics of disease prevalence and guide us that we need to control high activity group's risky behaviour.

## Acknowldgements

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