Mathematical Modelling and Analysis
Volume 10 Number 3, 2005, pages 275–286
 ISSN 1392-6292

MODELLING AND ANALYSIS OF HIV-TB CO-INFECTION IN A VARIABLE SIZE POPULATION

R. NARESH and A. TRIPATHI

Department of Mathematics, Harcourt Butler Technological Institute
Kanpur - 208002, Uttar Pradesh, India
E-mail: ramntripathi@yahoo.com

Received December 31, 2004; revised July 18, 2005

Abstract. In this paper, a nonlinear mathematical model is proposed for the transmission dynamics of HIV and a curable TB pathogen within a population of varying size. In the model, we have divided the population into four sub classes of susceptibles, TB infectives, HIV infectives and that of AIDS patients. The model exhibits four equillibria namely, a disease free, HIV free, TB free and a co-infection equilibrium. The model has been studied qualitatively using stability theory of nonlinear differential equations. It is shown that the positive co-infection equilibrium is always locally stable but it may become globally stable under certain conditions showing that the disease becomes endemic due to constant migration of the population into the habitat. A numerical study of the model is also performed to investigate the influence of certain key parameters on the spread of the disease.

Key words: HIV/AIDS epidemic, TB pathogen, stability, region of attraction, equilibrium points

1. Introduction

There are many infectious diseases which afflict human population around the world and spread by sexual contact between susceptibles and infectives. Recently, the Human Immuno-deficiency virus (HIV) infection, which can lead to Acquired Immuno-deficiency Syndrome (AIDS), has become an important infectious disease in both the developed and developing nations. It is a fatal disease, which breaks down the body's immune system, leaving the victim vulnerable to a host of life threatening opportunistic infections, neurological disorders or unusual malignancies. It causes mortality of millions of people and expenditure of enormous amount of money in health care and disease control. The AIDS epidemic is now spreading rapidly in Asia, where new

infections are increasing faster than anywhere else in the world. Globally, India has the highest estimated number of HIV infected people in any single country, next only to South Africa. India's epidemic is marked by heterogeneity. It is not a single epidemic but made up of distinct epidemics within the same state and continues to be driven strongly by heterosexual transmission. Mathematical models have been used extensively in research into the epidemiology of HIV/AIDS, to help improve our understanding of the major contributing factors in a given epidemic. From the initial models of May and Anderson [1, 2, 14] various refinements have been added into modeling frameworks, and specific issues have been addressed by researchers, see for instance [3, 4, 5, 6, 7, 11, 12, 13, 18, 19, 20]. In particular, Doyle et al. [8] developed a model for the spread of HIV in a heterosexual population taking into account the group contact tracing and carried out equilibrium analysis. Greenhalgh et al. [11] studied the impact of condom use on the sexual transmission of HIV and AIDS amongst a homogeneously mixing male homosexual population. Arazoza and Lounes [3] proposed a nonlinear model for an epidemic with contact tracing and applied it to the Cuban HIV/AIDS epidemic to obtain the size of HIV epidemic. Rao [20] presented a theoretical framework for transmission of HIV/AIDS epidemic in India. Corbett et al. [5] presented a homogeneous mixing population model for HIV transmission, which incorporates an anti-HIV preventive vaccine. Naresh and Omar [18] proposed a simple deterministic model to study the transmission of HIV/AIDS in a population with variable size structure. Hsieh and Chen [13] developed a theoretical model for a community which has the structure of two classes of commercial sex workers and two classes of sexually active male customers with different levels of sexual activity.

TB, caused by *Mycobacterium tuberculosis* is an infectious disease that remains a problem worldwide. TB is such a type of disease which increases due to the environmental factors e.g. discharges of household wastes (garbage, trash etc.) in residential areas, open drainage of sewage in residential areas, open water storage tanks etc. [10, 21]. Once infected with *M. tuberculosis*, a person stays infected for many years, probably for life. The vast majority (90%) of people without HIV infection who are infected with *M. tuberculosis* do not develop tuberculosis disease. HIV is the most powerful risk factor for progression from TB infection to TB disease. An HIV positive person infected with *M. tuberculosis* has a 50% lifetime risk of developing TB whereas an HIV negative person infected with *M. tuberculosis* has only a 10% risk of developing TB. This is especially important in India where it is estimated that almost half of the adult population harbours *M. tuberculosis* [16].

It is seen that TB is the most common serious opportunistic infection occurring among HIV–positive persons and is the first manifestation of AIDS in more than 50% of cases in developing countries. The number of HIV positives in India is estimated to be 3.97 million cases. Amongst the AIDS cases reported so for, nearly 60% had TB [16]. TB shortens the survival of patients with HIV infection. It accelerates the progression of HIV. Therefore, it is essential to study the transmission of HIV-TB co-infection in the population.

Some studies have been made by taking into account HIV-TB co-infection [9, 15, 17, 22, 23]. McLean and Nowak [15] have proposed within host models for the dynamics between HIV and activated CD_4T cells specific to other pathogens. West and Thompson [22] developed models which reflect the transmission dynamics of both TB and HIV and discussed the magnitude and duration of the effect that the HIV epidemic may have on TB. They found the effect that HIV will have on the general population to be dependent on the contact structure between the general population and the HIV risk groups, as well as a possible shift in the dynamics associated with TB transmission.

Our study is focused on the design and analysis of a new population model for the transmission dynamics of HIV and a curable TB pathogen in a population with a variable size structure. In this paper, therefore, a mathematical model is proposed to study the transmission of HIV/AIDS in presence of curable TB pathogen within a given population with varying size including demographic features. Qualitative and numerical analysis of the proposed model are carried out to understand the dynamics of HIV-TB co-infection.

2. The Mathematical Model

We consider the population of size $N(t)$ at time t with constant immigration rate Q_0. The population size $N(t)$ is divided into four subclasses of susceptibles $S(t)$, tuberculosis infectives $I_1(t)$, HIV infectives $I_2(t)$ (also assumed to be infectious) and AIDS patients $A(t)$. The susceptibles become tuberculosis infected following contact with the tuberculosis infectives I_1 at a rate β_1. The population in this class is diminished by HIV infection at a rate β_3. Some members of this class are also cured and recovered at a rate λ and become susceptible again. The population in class I_2 is generated by the HIV infection of both the susceptibles and tuberculosis infected individuals at a rate β_2 and β_3 respectively. It is assumed that the members of I_2 are not susceptible to infection by the tuberculosis. It is further assumed that anti-HIV treatment is not available within the community and therefore, individuals of the I_2 are bound to develop 'full blown' AIDS with a rate δ as shown in the transfer diagram presented in Fig. 1.

Thus, the spread of disease is assumed to be governed by the following system of differential equations,

$$\frac{dS}{dt} = Q_0 - \left(\frac{\beta_1 S I_1 + \beta_2 S I_2}{N}\right) - dS + \lambda I_1; \quad S(0) = S_0,$$

$$\frac{dI_1}{dt} = \frac{\beta_1 S I_1}{N} - \frac{\beta_3 I_1 I_2}{N} - (\lambda + d)\, I_1; \quad I_1(0) = I_{10},$$

$$\frac{dI_2}{dt} = \frac{\beta_2 S I_2}{N} + \frac{\beta_3 I_1 I_2}{N} - (\delta + d)\, I_2; \quad I_2(0) = I_{20},$$

$$\frac{dA}{dt} = \delta I_2 - (\alpha + d)\, A; \quad A(0) = A_0,$$

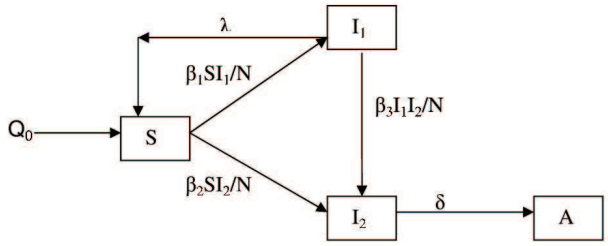

Figure 1. Transfer diagram of the mathematical model.

where d is the natural mortality rate constant and α is disease-induced death rate constant. It is assumed that all the dependent variables and parameters of the model are non-negative.

Since $N(t) = S(t) + I_1(t) + I_2(t) + A(t)$, the above equations can now be written as

$$\frac{dN}{dt} = Q_0 - dN - \alpha A, \tag{2.1}$$

$$\frac{dI_1}{dt} = \frac{\beta_1 (N - I_1 - I_2 - A) I_1}{N} - \frac{\beta_3 I_1 I_2}{N} - (\lambda + d) I_1, \tag{2.2}$$

$$\frac{dI_2}{dt} = \frac{\beta_2 (N - I_1 - I_2 - A) I_2}{N} + \frac{\beta_3 I_1 I_2}{N} - (\delta + d) I_2, \tag{2.3}$$

$$\frac{dA}{dt} = \delta I_2 - (\alpha + d) A. \tag{2.4}$$

From the model, it is noted that in the absence of any infection, the population size approaches the steady state value Q_0/d.

3. Stability Analysis

The model (2.1)–(2.4) has four non-negative equillibria namely,

(i) $E_0(Q_0/d, 0, 0, 0)$ the disease free.

(ii) $E_1\left(\frac{Q_0}{d}, \frac{Q_0[\beta_1 - (\lambda + d)]}{d\beta_1}, 0, 0\right)$ the HIV free, which exists if $\beta_1 > (\lambda + d)$.

(iii) $E_2(\hat{N}, 0, \hat{I}_2, \hat{A})$ the TB pathogen free, which exists if $\beta_2 > (\delta + d)$ and $Q_0 > \frac{\alpha\delta}{\alpha + d}\hat{I}_2$, where

$$\hat{N} = \frac{1}{d}\Big[Q_0 - \frac{\alpha\delta}{\alpha+d}\hat{I}_2\Big], \quad \hat{A} = \frac{\delta}{\alpha+d}\hat{I}_2,$$

$$\hat{I}_2 = \frac{Q_0/d[\beta_2 - (\delta+d)]}{\big[\frac{\beta_2(\alpha+d+\delta)}{\alpha+d} + \frac{\alpha\delta}{d(\alpha+d)}\{\beta_2 - (\delta+d)\}\big]}.$$

(iv) $E^*(N^*, I_1^*, I_2^*, A^*)$ the co-infection equilibrium, where

$$N^* = \frac{1}{d}\left[Q_0 - \frac{\alpha\delta}{\alpha+d}I_2^*\right], \quad A^* = \frac{\delta}{a+d}I_2^*,$$

$$I_1^* = \frac{Q_0/d[\beta_1 - (\lambda+d)] - \left[\frac{\beta_1(\alpha+d+\delta)}{(\alpha+d)} + \beta_3 + \frac{\alpha\delta[\beta_1-(\lambda+d)]}{d(\alpha+d)}\right] I_2^*}{\beta_1},$$

$$I_2^* = \frac{Q_0/d[\{\beta_2 - (\delta+d)\} + \left(\frac{\beta_3-\beta_2}{\beta_1}\right)\Big\{\beta_1 - (\lambda+d)\}]}{[\frac{\beta_3(\alpha+d+\delta)}{\alpha+d} + \frac{\alpha\delta}{d(\alpha+d)}\Big[\Big\{\beta_1 - (\lambda+d)\Big\} + \Big\{\beta_2 - (\delta+d)\Big\}\Big] + \beta_3\Big(\frac{\beta_3-\beta_2}{\beta_1}\Big)\Big]}.$$

It is noted that E^* is positive only when,

$$Q_0 > \frac{\alpha\delta}{\alpha+d}I_2^*, \quad \beta_1 > (\lambda+d), \quad \beta_2 > (\delta+d)$$

and

$$\frac{Q_0[\beta_1 - (\delta+d)]}{d} > \Big[\frac{\beta_1(\alpha+d+\delta)}{a+d} + \beta_3 + \frac{\alpha\delta[\beta_1-(\lambda+d)]}{d(\alpha+d)}\Big] I_2^*.$$

From the above, it is found that the equilibrium level of infectives $I_i^*(i = 1, 2)$ increases as Q_0 or $\beta_i(i = 1, 2)$ increases or λ and δ decreases leading to increase in A^*. Further the equilibrium level of AIDS patients A^* decreases as disease induced death rate α increases. When the disease remain endemic, the disease induced deaths reduce the equilibrium population size from Q_0/d to N^*.

Now we state a theorem for local stability of the above equilibrium points. The proof of the theorem is omitted as it is easy and can be obtained by computing variational matrices corresponding to the equilibrium points.

Theorem 1. (I) *The equilibrium point E_0 is locally asymptotically stable if $\frac{\beta_1}{\lambda+d} < 1$ (i.e. $R_1 < 1$) and $\frac{\beta_2}{\delta+d} < 1$ (i.e. $R_2 < 1$) and the second equilibrium E_1 is unstable. Here R_1 and R_2 are basic reproduction numbers associated with tuberculosis and HIV HIV infection respectively.*

(II) *The third equilibrium E_2 is unstable due to one positive eigenvalue and will be locally asymptotically stable if $\beta_1 < (\lambda+d), \beta_2 < (\delta+d)$ and provided $a_1a_2 - a_3 > 0$, where a_1, a_2 and a_3 all are positive the values of which are given by*

$$a_1 = \alpha + 2d + \beta_2\Big[\frac{2\hat{I}_2}{\hat{N}} + \frac{\hat{A}}{\hat{N}}\Big] - [\beta_2 - (\delta+d)],$$

$$a_2 = d(\alpha+d) + \frac{\beta_2\delta\hat{I}_2}{\hat{N}} + \beta_2(\alpha+d)\Big[\frac{2\hat{I}_2}{\hat{N}} + \frac{\hat{A}}{\hat{N}}\Big] - (\alpha+2d)[\beta_2 - (\delta+d)],$$

$$a_3 = \Big[\delta\big(d+\alpha\big[\frac{\hat{I}_2}{\hat{N}} + \frac{\hat{A}}{\hat{N}}\big]\big)\Big]\frac{\beta_2\hat{I}_2}{\hat{N}} + d(\alpha+d)\beta_2\Big[\frac{2\hat{I}_2}{\hat{N}} + \frac{\hat{A}}{\hat{N}}\Big] - d(\alpha+d)[\beta_2 - (\delta+d)].$$

(III) *The fourth equilibrium E^*, if it exists, is locally asymptotically stable provided $b_i > 0$ (for $i = 1,2,3,4$), $b_1b_2 - b_3 > 0$ and $b_1b_2b_3 - b_3^2 - b_1^2b_4 > 0$, where b_1, b_2, b_3 and b_4 are given by*

$$b_1 = \alpha + 2d + \frac{\beta_1 I_1^*}{N^*} + \frac{\beta_2 I_2^*}{N^*},$$

$$b_2 = \Big[\alpha + d + \delta + d\Big(\alpha + d + \frac{\beta_2 I_2^*}{N^*}\Big)\Big]\frac{\beta_2 I_2^*}{N^*} + \Big(\alpha + 2d + \frac{\beta_2 I_2^*}{N^*}\Big)\frac{\beta_1 I_1^*}{N^*} + (\beta_1+\beta_3)(\beta_3-\beta_2)\frac{I_1^* I_2^*}{N^{*2}},$$

$$b_3 = \Big[\beta_2\Big(\alpha+d+\delta\Big)d + \Big\{\beta_2 - (\delta+d)\Big\}\delta\Big]\frac{I_2^*}{N^*} + \Big[\Big(\alpha+d+\delta+d\Big\{\alpha+d+\frac{\beta_2 I_2^*}{N^*}\Big\}\Big)\beta_1\beta_2\frac{I_1^* I_2^*}{N^{*2}}\Big] + (\beta_3-\beta_2)\Big[\beta_1\delta + (\alpha+2d)(\beta_1+\beta_3)\Big]\frac{I_1^* I_2^*}{N^{*2}},$$

$$b_4 = \Big[\beta_1 - (\lambda+d)\Big]\frac{I_1^*}{N^*} + \Big[d(\alpha+d+\delta)\beta_2 + \Big[\beta_2 - (\delta+d)\Big]\delta\Big]\frac{\beta_1 I_1^* I_2^*}{N^{*2}} + (\beta_3-\beta_2)\frac{\alpha\delta I_2^*}{N^*} + d\Big[(\beta_1+\beta_3)(\beta_3-\beta_2)(\alpha+d) + (\beta_3-\beta_2)\beta_1\delta\Big]\frac{I_1^* I_2^*}{N^{*2}}.$$

It is noted that E_0 is locally asymptotically stable if $R_1 < 1$ and $R_2 < 1$, thus the desease dies out and under this condition the equilibria E_1, E_2 and E^* do not exist. The basic reproduction number is obtained as $R = \max\{R_1, R_2\}$, [6]. Thus, the disease free equilibrium is locally asymptotically stable if $R < 1$ and unstable if $R > 1$. If $R_1 > 1, R_2 > 1$ the equilibrium point E_0 is a saddle point which is stable in $N - A$ manifold and unstable in $I_1 - I_2$ direction. In such a case both the infections are maintained in the population.

Now to show that E^* is globally asymptotically stable, we state the following lemma.

Lemma 1. *The region*

$$\Omega = \Big\{(N, I_1, I_2, A); 0 < N \le \overline{N}; 0 \le I_1 \le \overline{I_1}; 0 \le I_2 \le \overline{I_2}; 0 \le A \le \frac{\delta\overline{I_2}}{(\alpha+d)}\Big\}$$

is a region of attraction for $\beta_1 > (\lambda+d)$, $\beta_2 > (\delta+d)$, where

$$\overline{N} = \frac{Q_0}{d},\ \overline{I_1} = \frac{Q_0}{d}\Big[1 - \frac{\lambda+d}{\beta_1}\Big],\ \overline{I_2} = \frac{Q_0}{d}\Big[(1 - \frac{\lambda+d}{\beta_2}) + \frac{\beta_3}{\beta_2}\Big[\Big(1 - \frac{\lambda+d}{\beta_1}\Big)\Big].$$

Theorem 2. *If the endemic equilibrium E^* exists, then it is globally asymptotically stable provided the following conditions are satisfied in Ω :*

$$\frac{\alpha^2 N^*}{dp_{11}} < k_1 < \min\{q_{11}, q_{12}\}, \quad \beta_1 < p_{11}(\alpha + d),$$

where

$$p_{11} = \frac{4\beta_2(\beta_1 + \beta_3}{9\delta(\beta_3 - \beta_2)}, \quad q_{11} = \frac{4dN^*\beta_1\overline{N}^2}{9[\beta_1(\overline{I}_1 + \overline{I}_2 + \overline{A}) + \beta_3\overline{I}_2]^2},$$

$$q_{12} = \frac{4dN^*(\beta_3 - \beta_2)\overline{N}^2}{9\beta_2[(\overline{I}_1 + \overline{I}_2 + \overline{A})]^2(\beta_1 + \beta_3)}.$$

Proof. Consider the positive definite function defined in the neibourhood of E^*:

$$V = \frac{(N - N^*)^2}{2} + k_1\big(I_1 - I_1^* - I_1^* \ln \frac{I_1}{I_1^*}\big) + k_2\big(I_2 - I_2^* - I_2^* \ln \frac{I_2}{I_2^*}\big) + \frac{1}{2}k_3(A - A^*)^2,$$

where constants k_1, k_2, k_3 can be chosen suitably. It is easy to show that V is a Liapunov function with respect to E^*, whose domain contains Ω. The values of k_2 and k_3 have been found as

$$k_2 = \Big(\frac{\beta_1 + \beta_3}{\beta_3 - \beta_2}\Big)k_1, \quad k_3 = \frac{\beta_2 k_2}{N^*\delta}.$$

■

4. Numerical Analysis and Discussion

In this section we give results of numerical simulation of the equilibrium and stability conditions of the model (2.1)–(2.4). We integrate this system by fourth order Runge–Kutta method using the following set of parameter values:

$$Q_0 = 2000, \ \ d = \frac{1}{50}, \ \ \beta_1 = 0.925, \ \ \lambda = 0.3,$$
$$\beta_2 = 0.365, \ \ \beta_3 = 1.15, \ \ \alpha = 1, \ \ \delta = 0.2$$

and initial values

$$N(0) = 20000, \ \ I_1(0) = 2000, \ \ I_2(0) = 3000, \ \ A(0) = 500.$$

The co-infection equilibrium values are computed as

$$N^* = 39275.6009, \ \ I_1^* = 5349.520, \ \ I_2^* = 6193.2075, \ \ A^* = 1214.4879.$$

Table 1. Effect of λ from equilibrium point E_1.

λ	0	0.2	0.4	0.6	0.8	0.91
R_1	46.25	4.205	2.202	1.492	1.128	0.995
I_1	97387.8	76216.2	54594.6	32973.0	11351.4	0

In Table1 we have shown the effect of recovery rate λ. The higher values of recovery rate λ lead to the reduced TB infection and at $\lambda = 0.91$ we found that $I_1 = 0$ implying that if 91% TB infected people are recovered effectively then the TB pathogen can be eradicated from the population. This condition coincides with the case where both R_1 and R_2 are less than one so that the disease free equilibrium is stable.

In Table 2 we have shown the effect of β_2. As the contact rate decreases, the HIV infected population also decreases. From this table we find that as $\beta_2 \to 0$ then $I_2 \to 0$. We get the expected results given in Theorem 1.

Table 2. Effect of β_2 from equilibrium point E_2.

β_2	1	0.8	0.6	0.4	0.3	0.2
R_2	4.545	3.636	2.727	1.818	1.364	0.909
I_2	8820.6	8731.0	8587.6	8025.0	6999.1	0

The results of numerical simulations are displayed graphically in Fig.2–9.

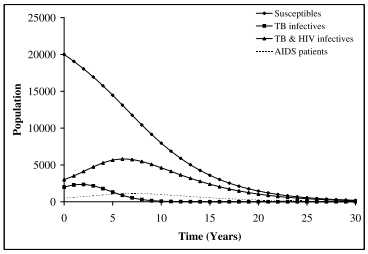

Figure 2. Variation of population in different classes for $Q_0 = 0$, $\lambda = 0, d = 0.02, \alpha = 1, \delta = 0.2, \beta_1 = 0.925, \beta_2 = 0.365$ and $\beta_3 = 1.15$.

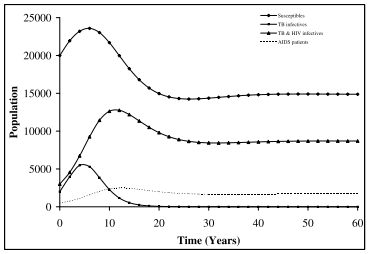

Figure 3. Variation of population in different classes for $Q_0 = 2000, \lambda = 0.3$, $d = 0.02, \alpha = 1, \delta = 0.2, \beta_1 = 0.925, \beta_2 = 0.365$ and $\beta_3 = 1.15$.

In Fig. 2 the distribution of population with time is shown in different classes without migration and recovery rate, i.e. $Q_0 = 0$ and $\lambda = 0$. It is seen that susceptible population decreases continuously and infective population

increases because there is no migration and recovery. Therefore, all infectives ultimately develop AIDS and will die out by disease-induced deaths. Thus the total population, being constant, in this case will be eradicating after some time period.

Fig. 3 shows the variation of population with migration and recovery rate. It is noted that due to recovery rate TB infective population decreases and susceptible population first increases with time and then reaches its equilibrium position. Since due to migration susceptible population increases continuously, therefore, infection becomes more endemic and always persists in the population.

Comparing Fig. 2 and Fig. 3, the role of immigration rate Q_0 can be seen easily on the different populations. In particular, the role of immigration is explicity shown in Fig. 9 and it is seen that the increase in the rate of migration into the community is to increase the AIDS population.

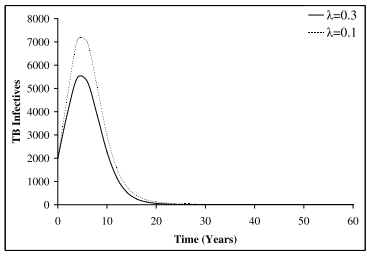

Figure 4. Variation of TB infected population for different values of λ.

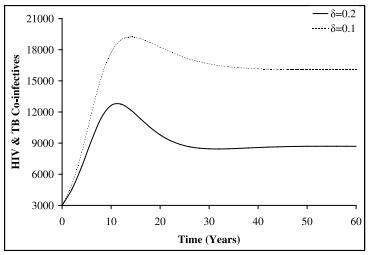

Figure 5. Variation of HIV & TB co-infected population for different values of δ.

Fig. 4 depicts the variation of TB infected population with time for different recovery rates. It is seen that with the increase in the recovery rate λ, the TB infected population decreases which in turn increases the susceptible population. Fig.5 – 6 show the effect of movement rate δ on HIV infected and AIDS population respectively. It is clear that with increase in the value of δ the HIV infected population decreases to increase the full blown AIDS population. This is expected because of shorter incubation period. In Fig.7 the variation of AIDS population for different values of disease-induced death rate is shown. It is observed that with the increase in disease induced death rate α, the AIDS population decreases. The role of β_2 is plotted in Fig.8. It is found that with the increase in contact rate β_2, the AIDS population increases.

From the above discussion, it is observed that if TB infection is treated significantly then acceleration to HIV infection can be kept under control.

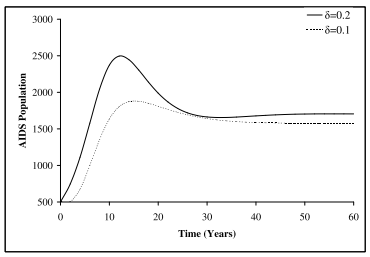

Figure 6. Variation of AIDS population for different values of δ.

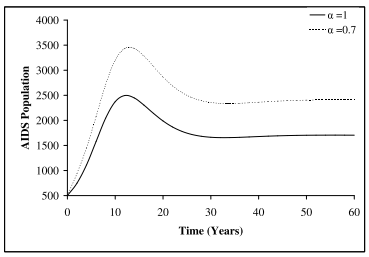

Figure 7. Variation of AIDS population for different values of α.

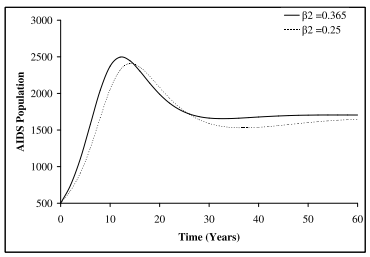

Figure 8. Variation of AIDS population for different values of β_2.

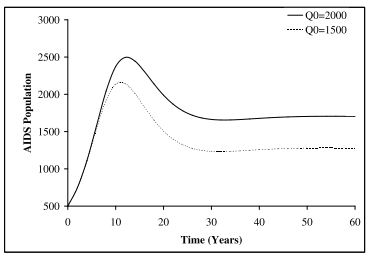

Figure 9. Variation of AIDS population for different values of Q_0.

5. Conclusion

In this paper, a nonlinear mathematical model is proposed and analysed to study the transmission of HIV/AIDS and a curable TB pathogen within a population of variable size structure. It is shown that there exist threshold parameters R_1 and R_2. It is noted that when $R_1 < 1$ and $R_2 < 1$ then both the infections die out and when $R_1 > 1$ and $R_2 > 1$ the co-infection is maintained in the population. The model has four non-negative equillibria namely E_0, the disease free, E_1 the HIV free, E_2 TB pathogen free and E^* the co-infection equilibrium. It is found that the equilibrium state E_0 corresponding to disappearance of disease, is locally asymptotically stable if $R_1 < 1$ and $R_2 < 1$ and for $R_1 > 1$ and $R_2 > 1$ it is unstable and both the infections are maintained in the population. The co-infection equilibrium E^* is always locally asymptotically stable. For this equilibrium we have found a Liapunov function and shown that this equilibrium is globally asymptotically stable if the conditions in Theorem 2 are satisfied. It is also found that these infectious diseases become more endemic due to immigration. Thus if the migration of the population into susceptible community is restricted, the spread of the dis-

ease can be kept under control. The number of HIV infected individuals in population increases rapidly due to presence of another disease that is tuberculosis. Thus if tuberculosis in the population is effectively treated, the spread of HIV can be slowed down. The model analysis suggests that TB will be eradicated from the population if approximately above than 90% people are recovered due to effective treatment. The effect of an increase in disease-induced death rate is, however, to decrease the AIDS patients population.

From the analysis, it may be speculated that if the HIV infection is suppressed at an early stage by way of drug therapy or other control mechanism, the progression to the AIDS can be slowed down and the life span of HIV infectives can be increased.

References

[1] R.M. Anderson. The role of mathematical models in the study of HIV transmission and the epidemiology of AIDS. *Journal of AIDS*, **1**, 241 – 256, 1988.

[2] R.M. Anderson, G. F. Medly, R. M. May and A. M. Johnson. A preliminary study of the transmission dynamics of the Human Immunodeficiency virus (HIV), the causative agent of AIDS. *IMA J. Math. Appl. Med. Biol.*, **3**, 229 – 263, 1986.

[3] H.D. Arazoza and R. Lounes. A nonlinear model for sexually transmitted disease with contact tracing. *IMA J. Math. Appl. Med. Biol.*, **19**, 221 – 234, 2002.

[4] S. Busenberg, K. Cooke and H. Ying-Hen. A model for HIV in Asia. *Math. Biosc*, **128**(1,2), 185 – 210, 1995.

[5] B.D. Corbett, S.M. Moghadas and A.B. Gumel. Subthreshold domain of bistable equilibria for a model of HIV epidemiology. *Int. J. Math. & Math. Sc.*, **58**, 3679 – 3698, 2003.

[6] O. Diekmann, P.J.A. Heesterbeek and J.A.J. Metz. On the definition and the basic reproduction ratio r_0 in models for infectious diseases in heterogeneous populations. *J. Math. Biol.*, **28**, 365 – 382, 1990.

[7] K. Dietz. On the transmission dynamics of HIV. *Math. Biosci.*, **90**, 397 – 414, 1988.

[8] M. Doyle, D. Greenhalgh and S. Blythe. Equilibrium analysis of a mathematical model for the spread of AIDS in a two sex population with mixing constraints. *J. Biol. Sys.*, **6**(2), 159 – 185, 1998.

[9] L. Esteva and M. Matias. A model for vector transmitted disease with saturation incidence. *J. Biol. Sys.*, **9**(4), 235 – 245, 2001.

[10] M. Ghosh, J.B. Shukla, P. Chandra and P. Sinha. An epidemiological model for carrier dependent infectious disease with environmental effect. *Int. J. Appl. Sci.& Comp.*, **7**(3), 188–204, 2000.

[11] D. Greenhalgh, M. Doyle and F. Lewis. A mathematical treatment of AIDS and condom use. *IMA J. Math. Appl. Med. Biol.*, **18**, 225 – 262, 2001.

[12] Y. H. Heieh and S. P. Shen. The effect of density dependent treatment and behavior change on the dynamics of HIV transmission. *J. Math. Biol.*, **43**, 69 – 80, 2001.

[13] Ying-Hen Hsieh and Chien Hsun Chen. Modeling the social dynamics of a sex industry: Its implications for spread of HIV/AIDS. *Bull. Math. Biol.*, **66**, 143 – 166, 2004.

[14] R.M. May and R.M. Anderson. Transmission dynamics of HIV infection. *Nature*, **326**, 137 – 142, 1987.
[15] A. R. McLean and M. A. Nowak. Models of interactions between HIV and other pathogens. *J.Theor.Biol.*, **155**, 69 – 86, 1992.
[16] S.M. Moghadas and M.E. Alexander. Exogenous reinfection and resurgence of tuberculosis: A theoretical framework. *J.Biol. Sys.*, **12**(2), 231 – 247, 2004.
[17] B.M. Murphy, B. H. Singer and D. Kirschner. On treatment of tuberculosis in heterogeneous populations. *J.Theor. Biol.*, **223**(4), 391 – 404, 2003.
[18] R. Naresh and S. Omar. Analysis of a nonlinear AIDS epidemic model with standard incidence. *Ultra Science (Int. J. of Physical Sciences)*, **15**(1M), 63–70, 2003.
[19] A.S. Perelson and P.W. Nelson. Mathematical analysis of HIV-1 dynamics in vivo. *SIAM Rev.*, **41**, 3 – 44, 1999.
[20] A.S.R. Srinivasa Rao. Mathematical modeling of AIDS epidemic in India. *Current Science*, **84**(9), 1192 – 1197, 2003.
[21] S. Singh, P. Chandra and J.B. Shukla. Modeling and analysis of the spread of carrier dependent infectious disease with environmental effects. *J. Biol.Sys.*, **11**(3), 325 – 335, 2003.
[22] R.W. West and A.R. Thompson. Modeling the impact of HIV on the spread of tuberculosis in the United States. *Bull. Math. Biol.*, **59**, 35 – 60, 1996.
[23] B.G. Williams and C. Dye. Antiretroviral drugs for tuberculosis control in the era of HIV/AIDS. *Science*, **301**, 1535 – 1537, 2003.

ŽIV ir TB infekcijų sąveikos kintamo dydžio populiacijoje modeliavimas ir analizė

R. Naresh, A. Tripathi

Šiame darbe pateikiamas netiesinis matematinis modelis, skirtas aprašyti ŽIV ir išgydomo TB patogeno plitimui kintamo dydžio populiacijoje. Modelyje populiacija dalinama į keturias klases – galintys užsikrėsti, TB infekuoti, ŽIV infekuoti ir AIDS pacientai. Modelis turi keturias pusiausvyros padėtis: nesergantys, nesergantys ŽIV, nesergantys TB ir sergantys abiem ligom. Modelis analizuojamas kokybiniu požiūriu, naudojant netiesinių diferencialinių lygčių stabilumo teoriją. Įrodyta, kad teigiama dviejų infekcijų pusiausvyros padėtis visada yra lokaliai stabili, be to, esant tam tikroms sąlygoms, ta padėtis taip pat būna ir globaliai stabili. Tai reiškia, kad esant pastoviai migracijai į arealą, liga tampa endemine. Atlikta modelio skaitinė analizė, skirta nagrinėti kai kurių svarbiausių parametrų įtaką AIDS ir TB ligų plitimui.