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AN EPIDEMIC MODEL FOR THE TRANSMISSION DYNAMICS OF HIV/AIDS AND ANOTHER INFECTION

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ABSTRACT

A simple deterministic mathematical model is proposed to study the spread of HIV/AIDS in a population with variable size structure. In writing the model, we have divided the population under consideration into five subclasses i.e. Susceptibles S (t), Infectives I (t), Pre-AIDS class P (t), AIDS patients A (t) and of serious HIV positive patients R (t) suffering from other opportunistic infection like tuberculosis. The model is applicable to a population of homosexual males with constant immigration rate and natural mortality rate. The model has been studied qualitatively using stability theory. It is shown that the positive non-trivial 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 community.

INTRODUCTION:

In recent year the human immunodeficiency virus (HIV) infection which can cause acquired immunodeficiency syndrome (AIDS), has shown a very high degree of prevalence in population all over world. The most susceptible individuals at risk of acquiring infection include the persons having sexual contact with HIV infected, homosexuals and bisexuals men intravenous drug abusers and persons transfused with contaminated blood or its products. The infection can also be transmitted vertically to the offspring of infected mothers. The transmission dynamics of HIV infection is affected by various epidemiological factors such as latent period, the infectious period, the portion of infected people in the population having AIDS, the percentage of asymptotic carriers, type of mixing within various high risk groups, their age and large variability in periods of incubation and infectivity, migration, diffusion, population structure etc.

In this direction some investigations have been made by taking into account immigration of the population and other demographic features, Nallaswamy and Shukla [9], May et al. [6,8], Anderson[1], Castillo-Chavez et al. [4], Brauer [2] Busenberg et al. [3], Nokes and Anderson[10], Zhou and Hethcote [13], West and Thompson [12], Greenhalgh et al.[5]. In particular, May et al. [6] proposed a model in which a fraction of infectives developed AIDS while the remaining part was assumed to be non-infectious seropositives. Castillo-Chavez et al. [4] have analyzed a model where the mean rate of acquisition of new partners depends on the size of the sexually active population. Brauer [2] described models for the spread of universally fatal disease like AIDS by incorporating nonlinear contact rates and population dynamics as well as differences in the distribution of infective periods. He has shown that the stability of endemic equilibrium for some kinds of population dynamics may depend on the distribution of infective periods. West and Thompson [12] 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.

In view of the above, in this paper we, therefore, propose a model to study the spread of HIV/AIDS in a population with variable size structure under the assumption that only a fraction of infectives moves to develop AIDS. The population, under consideration, is divided into five subclasses. The dynamics of these classes in assumed to be governed by ordinary differential equations with immigration, non-linear interaction and natural death terms. The interaction between susceptibles and infectives is as usual taken to be of standard mass action type.

MATHEMATICAL MODEL:

Consider a population of size N (t) at time t with constant immigration rate Q_0. The population size N(t) is divided into five subclasses of HIV negatives but susceptibles S(t), HIV positives or infectives I(t)

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(also assumed to be infectious), Pre-AIDS class P(t), AIDS patients A(t) and of serious HIV positive patients R(t) suffering from other opportunistic infections like tuberculosis with natural mortality rate d in all the classes. The susceptibles become infected via sexual contact with infectives and the interaction is of standard mass action type with a constant contact rate β_1. As outlined above, the AIDS patients, once identified, are effectively isolated and treated as sexually inactive and do not contribute to viral transmission so is the case with the individuals in the class R(t). The individuals in this class are also removed from circulation for being easily identifiable due to sufferings from other opportunistic infections. It is also assumed that a fraction $\delta_1 I$ of infectives moves to join Pre-AIDS class and $\delta_2 I$ infectives moves to develop 'full blown' AIDS while the remaining part of infectives i.e.$\delta_3 I$ increases the growth rate of R (t).

With these considerations, the spread of disease is assumed to be governed by the following system of differential equations,

$$\frac{dS}{dt} = Q_0 - \frac{\beta_1 SI}{N} - dS \; ; \; S(0) = S_0 \qquad (1)$$

$$\frac{dI}{dt} = \frac{\beta_1 SI}{N} - (\delta + d) I \; ; \; I(0) = I_0 \qquad (2)$$

$$\frac{dP}{dt} = \delta_1 I - (\alpha_1 + d) P \; ; \qquad P(0) = P_0 \qquad (3)$$

$$\frac{dA}{dt} = \alpha_1 P + \delta_2 I - (\alpha + d) A \; ; \; A(0) = A_0 \qquad (4)$$

$$\frac{dR}{dt} = \delta_3 I - dR \; ; \qquad R(0) = R_0 \qquad (5)$$

Where Q_0 is the rate of immigration to the class of susceptibles, d is the natural mortality rate constant. Since individuals will not usually remain infectious indefinitely, we assume that they leave the infective class at a rate δ so that $1/\delta$ denotes the average incubation period; α is the disease induced death rate constant.

Since N = S+I+P+A+R and $\delta = \delta_1 + \delta_2 + \delta_3$, the above equations, can now be written as,

$$\frac{dN}{dt} = Q_0 - dN - \alpha A \; ; \qquad N(0) = N_0 \qquad (6)$$

$$\frac{dI}{dt} = \frac{\beta_1 (N - I - P - A - R) I}{N} - (\delta + d) I \; ; \; I(0) = I_0 \qquad (7)$$

$$\frac{dP}{dt} = \delta_1 I - (\alpha_1 + d) P \; ; \qquad P(0) = P_0 \qquad (8)$$

$$\frac{dA}{dt} = \alpha_1 P + \delta_2 I - (\alpha + d) A \; ; \qquad A(0) = A_0 \qquad (9)$$

$$\frac{dR}{dt} = \delta_3 I - dR \; ; \qquad R(0) = R_0 \qquad (10)$$

From the model, it is noted that in the absence of infection, the population size approaches the steady state value Q_0/d. During the early stages of the epidemic, if it is assumed that $S \cong N \cong Q_0/d$ then the growth of infectious people I (t) can be approximately governed by the following equation,

$$\frac{dI}{dt} = [\beta_1 - (\delta + d)] I \; ; \qquad I(0) = I_0 \qquad (11)$$

which gives $I(t) = I_0 \exp((D_0 - 1)/T)t$ where $D_0 = \beta_1/(\delta + d)$, the basic reproduction rate, $T = 1/(\delta + d)$, the time during which people remain infective and I_0 is the initial infective population at t = 0. The doubling time t_d of the epidemic can be obtained as

$$t_d = (\ln 2) T / D_0 - 1 \qquad (12)$$

Thus, if $D_0 > 1$, the infection triggers an epidemic otherwise its prevalence is zero i.e. for $D_0 < 1$.

STABILITY ANALYSIS:

The model (6-10) has two non-negative equilibria namely

E_0 (Q_0/d, 0, 0, 0, 0), the disease free, and E*(N*, I*, P*, A*, R*), the endemic equilibrium, where

$$N^* = \frac{1}{d}\left[Q_0 - \frac{\alpha\gamma}{(\alpha + d)} I^* \right]; \qquad P^* = \frac{\delta_1}{(\alpha_1 + d)} I^*$$

$$I^* = \frac{[\beta_1 - (\delta + d)] \frac{Q_0}{d}}{\frac{\alpha\gamma}{d(\alpha + d)} [\beta_1 - (\delta + d)] + \beta_1 \left[1 + \frac{\delta_1}{(\alpha_1 + d)} + \frac{\delta_3}{d} + \frac{\gamma}{\alpha + d} \right]} \; ;$$

$$A^* = \frac{I^* \gamma}{(\alpha + d)}; \qquad R^* = \frac{\delta_3 I^*}{d}$$

Where $\gamma = \frac{\alpha_1 \delta_1}{(\alpha_1 + d)} + \delta_2$;

It is noted that E* is positive only when

$$\beta_1 > (\delta + d) \text{ and } Q_0 > \frac{\alpha\gamma}{(\alpha + d)} I^*.$$

From the above, it is found that the equilibrium

level of infectives I* increases as Q_0 or β_1 increases or δ decreases leading to increase in A* and R*. Further the equilibrium level of AIDS patients A* decreases as disease induced death rate α increases or as γ decreases and R* increases as d decreases. It is also noted that 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.

Theorem:

(i) The equilibrium point E_0 (Q_0/d, 0, 0, 0, 0) is locally asymptotically stable (LAS) if $D_0<1$ otherwise it is unstable and then second equilibrium E*(N*, I*, P*, A*, R*) exists.

(ii) The second equilibrium E*, if it exists, is locally asymptotically stable.

Proof: To determine the local stability of E_0 and E*, we compute the variational matrices M (E_0) and M (E*) corresponding to E_0 (Q_0/d, 0, 0, 0, 0) and E*(N*, I*, P*, A*, R*) as follows:

$$M(E_0)=\begin{bmatrix} -d & 0 & 0 & -\alpha & 0 \\ 0 & [\beta_1-(\delta+d)] & 0 & 0 & 0 \\ 0 & \delta_1 & -(\alpha_1+d) & 0 & 0 \\ 0 & \delta_2 & \alpha_1 & -(\alpha+d) & 0 \\ 0 & \delta_3 & 0 & 0 & -d \end{bmatrix}$$

$$M(E^*)=\begin{bmatrix} -d & 0 & 0 & -\alpha & 0 \\ [\beta_1-(\delta+d)]\frac{I^*}{N^*} & -\frac{\beta_1 I^*}{N^*} & -\frac{\beta_1 I^*}{N^*} & -\frac{\beta_1 I^*}{N^*} & -\frac{\beta_1 I^*}{N^*} \\ 0 & \delta_1 & -(\alpha_1+d) & 0 & 0 \\ 0 & \delta_2 & \alpha_1 & -(\alpha+d) & 0 \\ 0 & \delta_3 & 0 & 0 & -d \end{bmatrix}$$

From M (E_0), it is clear that E_0 is locally asymptotically stable (LAS) provided $\beta_1<\delta+d$ i.e. $D_0<1$, the disease dies out but under this condition the equilibrium E* does not exist as expected. However, if $D_0>1$ the equilibrium point E_0 is a saddle point which is stable in N-P-A-R manifold and unstable in I-direction. In such a case E* exists and the infection is maintained in the population.

The characteristic equation corresponding to M (E*) is given by

$$f(\lambda)=\left(\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4\right)(\lambda+d)=0 \tag{13}$$

Where

$$a_1=\frac{\beta_1 I^*}{N^*}+\alpha+3d+\alpha_1$$

$$a_2=3d^2+2d(\alpha+\alpha_1)+\alpha\alpha_1+\delta\beta_1\frac{I^*}{N^*}+\beta_1\frac{I^*}{N^*}(\alpha+\alpha_1+3d)$$

$$a_3=d^2(d+\alpha+\alpha_1)+\alpha\alpha_1 d$$
$$+\left[3d^2+2d(\alpha+\alpha_1)+\alpha(\alpha_1+\delta_1+\delta_3)+\delta(2d+\alpha_1)\right]\beta_1 I^*/N^*$$

$$a_4=[\delta\alpha_1 d+\delta d^2+\alpha d(\alpha_1+d)+d^2(\alpha_1+d)+(\delta_1+\delta_3)\alpha d+\delta_3\alpha\alpha_1]\frac{\beta_1 I^*}{N^*}$$
$$+\alpha(\delta_1\alpha_1+\delta_2)[\beta_1-(\delta+d)]\frac{I^*}{N^*}$$

Thus by Routh Hurwitz criteria, E* is locally asymptotically stable. As it can be seen for

$$a_1>0, a_2>0, a_3>0, a_4>0, a_1a_2-a_3>0 \text{ and } a_1a_2a_3-a_3^2-a_1^2a_4>0$$

We now show in the following theorem that the endemic equilibrium E*, if it exists, is globally asymptotically stable. A lemma is established first.

Lemma: The region

$$\Omega=\left\{\begin{matrix}(N,I,P,A,R);O\le N\le\frac{Q_0}{d};0\le I(t)\le I_{\max};0\le P(t)\le P_{\max};\\ 0\le A(t)\le\frac{\alpha_1 P_{\max}+\delta_2 I_{\max}}{(\alpha+d)};0\le R(t)\le\frac{\delta_3 I_{\max}}{d}\end{matrix}\right\}$$

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

$$I_{\max}=\frac{Q_0}{d}\left[1-\frac{(\delta+d)}{\beta_1}\right]$$

$$P_{\max}=\frac{\delta_1 I_{\max}}{(\alpha_1+d)}$$

Theorem:

If the endemic equilibrium E* exists, then it is globally asymptotically stable provided the following condition is satisfied in Ω.

$$\frac{\alpha^2}{\alpha+d}<\frac{d^2}{3\delta_2}\quad,\quad\frac{\alpha_1^2}{\alpha_1+d}<\frac{2\delta_2}{3\delta_1}(\alpha+d) \tag{14}$$

Proof: Consider the following positive definite function about E*

$$V = \frac{1}{2}(N - N^*)^2 + k_1\{I - I^* - I^* \ln(I/I^*)\}$$
$$+ \frac{1}{2}k_2(P - P^*)^2 + \frac{1}{2}k_3(A - A^*)^2 + \frac{1}{2}k_4(R - R^*)^2 \quad (15)$$

where the constants k_1, k_2, k_3 and $k_4$can be chosen suitable. The derivative of V along the solution of the system (6-10) can be written as,

$$\begin{aligned}\frac{dV}{dt} &= (N - N^*)[Q_0 - dN - \alpha A] \\ &+ k_1(I - I^*)\left[\frac{\beta_1(N - I - P - A - R)}{N} - (\delta + d)\right] \\ &+ k_2(P - P^*)[\delta_1 I - (\alpha_1 + d)P] \\ &+ k_3(A - A^*)[\alpha_1 P + \delta_2 I - (\alpha + d)A] \\ &+ k_4(R - R^*)[\delta_3 I - dR]\end{aligned}$$

After some algebraic manipulations,It can further be written as the sum of the quadratics as,

$$\begin{aligned}\frac{dV}{dt} = &-\frac{1}{2}a_{11}(N-N^*)^2 + a_{12}(N-N^*)(I-I^*) - \frac{1}{2}a_{22}(I-I^*)^2 \\ &-\frac{1}{2}a_{11}(N-N^*)^2 + a_{14}(N-N^*)(A-A^*) - \frac{1}{2}a_{44}(A-A^*)^2 \\ &-\frac{1}{2}a_{22}(I-I^*)^2 + a_{23}(I-I^*)(P-P^*) - \frac{1}{2}a_{33}(P-P^*)^2 \\ &-\frac{1}{2}a_{22}(I-I^*)^2 + a_{24}(I-I^*)(A-A^*) - \frac{1}{2}a_{44}(A-A^*)^2 \\ &-\frac{1}{2}a_{22}(I-I^*)^2 + a_{25}(I-I^*)(R-R^*) - \frac{1}{2}a_{55}(R-R^*)^2 \\ &-\frac{1}{2}a_{33}(P-P^*)^2 + a_{34}(P-P^*)(A-A^*) - \frac{1}{2}a_{44}(A-A^*)^2\end{aligned} \quad (16)$$

where

$$a_{11} = d, \qquad a_{12} = \beta_1 k_1 (I + P + A + R)/NN^*,$$
$$a_{13} = -\alpha, \qquad a_{22} = \beta_1 k_1 / 2N^*,$$
$$a_{23} = -(\beta_1 k_1 / N^* - k_2\delta_1), \quad a_{33} = k_2(\alpha_1 + d),$$
$$a_{24} = -(\beta_1 k_1 / N^* - \delta_2 k_3), \quad a_{44} = \frac{2}{3}k_3(\alpha + d)$$
$$a_{55} = -(\beta_1 k_1 / N^* - \delta_3 k_4), \quad a_{34} = k_3\alpha_1$$

Thus a sufficient condition for dV/dt to be negative definite is that

$$a_{12}^2 - a_{11}a_{22} < 0 \quad (16i)$$

$$a_{14}^2 - a_{11}a_{44} < 0 \quad (16ii)$$

$$a_{23}^2 - a_{22}a_{33} < 0 \quad (16iii)$$

$$a_{24}^2 - a_{22}a_{44} < 0 \quad (16iv)$$

$$a_{25}^2 - a_{22}a_{55} < 0 \quad (16v)$$

$$a_{34}^2 - a_{33}a_{44} < 0 \quad (16vi)$$

Now choosing

$$k_2 = \frac{\beta_1 k_1}{\delta_1 N^*}, \; k_3 = \frac{\beta_1 k_1}{\delta_2 N^*} \text{ and } k_4 = \frac{\beta_1 k_1}{\delta_3 N^*},$$

the conditions (16i–vi) give

$$\frac{\alpha^2}{\alpha + d} < \frac{d^2}{3\delta_2},$$
$$\frac{\alpha_1^{\,2}}{\alpha_1 + d} < \frac{2\delta_2}{3\delta_1}(\alpha + d)$$

Where k_1 is obtained as $k_1 = \frac{dN^*}{2\beta_1}$.

Hence V is a Liapunov function with respect to E* whose domain contains Ω, proving the theorem.

CONCLUSION:

In the paper, a nonlinear mathematical model is proposed and analyzed to study the spread of HIV/AIDS in a population of varying size with constant recruitment under the assumption that all infectives may not ultimately develop AIDS but some other diseases due to weak immune system. The interaction between susceptibles and infectives is assumed to be standard mass action type. Using the stability theory, some inferences have been drawn regarding the spread of the disease. It is shown that in the system (6-10), as usual, there exists a threshold parameter $D_0 = (\beta_1/(\delta+d))$. It is noted that when $D_0<1$, the disease dies out and when $D_0>1$ the disease becomes endemic. The model has two non-negative equilibria namely $E_0(Q_0/d, 0, 0, 0, 0)$, the disease free equilibrium and E* (N*, I*, P*,A*, R*), the endemic equilibrium for $D_0>1$. It is found that the equilibrium state E_0, corresponding to disappearance of the disease, is locally asymptotically stable if $D_0<1$ and for $D_0>1$ it is unstable and the infection is maintained in the population. Further, the endemic equilibrium E* which exists only when $D_0>1$ is always locally asymptotically stable. This equilibrium is also shown to be globally asymptotically stable if the condition (14) is satisfied. It is noted when disease remain endemic, the disease induced deaths reduce the equilibrium population size from Q_0/d to N*. It is shown that I* increases as rate of recruitment Q_0 or contact rate β_1 increases or as rate of movement δ from infectious class decreases. Thus the

equilibrium of infective population decreases which in turn increases the population of AIDS patients and that of other serious HIV positive patients suffering from opportunistic infections. An increase in the disease induced death rate α is, however, to decrease the AIDS patients population.

Thus analysis of the model suggests, as discussed above, that in order to reduce the growth of the disease to the 'full blown' AIDS, a device in the form of vaccination or drug therapy may be evolved to suppress the infection at an early stage. Analysis also suggests that if the number of sexual partners and the unsafe sexual interaction with infectives is restricted, the number of diseased individuals can be decreased. Also if the migration of the population into a community with infection is restricted, the spread of the disease can be minimized.

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