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ORIGINAL ARTICLE

A mathematical model on Acquired Immunodeficiency Syndrome

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Abstract A mathematical model SEIA (susceptible-exposed-infectious-AIDS infected) with vertical transmission of AIDS epidemic is formulated. AIDS is one of the largest health problems, the world is currently facing. Even with anti-retroviral therapies (ART), many resource-constrained countries are unable to meet the treatment needs of their infected populations. We consider a function of number of AIDS cases in a community with an inverse relation. A stated theorem with proof and an example to illustrate it, is given to find the equilibrium points of the model. The disease-free equilibrium of the model is investigated by finding next generation matrix and basic reproduction number $\Re_0$ of the model. The disease-free equilibrium of the AIDS model system is locally asymptotically stable if $\Re_0 \leqslant 1$ and unstable if $\Re_0 > 1$. Finally, numerical simulations are presented to illustrate the results.

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1. Introduction

AIDS was first identified as a distinct new disease in 1981. In 1983 HIV was identified at the causative agent for AIDS. The mean time from HIV infection to AIDS is approximately 10 years. There is no effective medicine to cure it and the infected individuals do not recover: that is, they continue to be infectious throughout their lives. HIV infection is a complex mix of diverse epidemics within and between countries and regions of the world, and is undoubtedly the defining public health crisis of our time. The three known modes of transmission of HIV are

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sexual contact, direct contact with HIV-infected blood or fluids, and perinatal transmission from an infected mother to child [1,2].

The complexity of the complete formalization of mathematical models is probably responsible for the discomfort with which biologists treat mathematical research literature on medicine and biology. They also appear to have reservations about the convergence between epidemiological and their mathematical models. However, there are instances of success in constructing models capable of predicting the outcome of infections. Such instances inspire our confidence in proposing realistic models. Hence present modeling attempts should be aimed at determining HIV spread mechanisms through testing the sensitivity of the assumptions [3].

Anderson and May [3] present more models of infections including HIV with illustrations. Models of HIV spread specific to the type of transmission have also appeared in the literature. Mathematical models have also been developed recently for other sexually transmitted diseases (STDs). Garnett [4] has presented a simple and useful discussion on various mathematical models for STDs. Mukherji [5] represents one of the earliest Indian attempts at modelling data on AIDS. This model used annualized south Asian regional data and extrapolated to AIDS in future. Basu [6] attempt to model the spread of AIDS in a comprehensive manner with limited data. The incubation period of AIDS in India, estimated through deconvoluting HIV epidemic density and reported AIDS cases, is between 8 and 12 years. Quantitative information on commercial sex activity and female commercial sex workers (FSWs) number in India are available in various sources [7].

In the last decades, many mathematical models have been developed to describe the immunological response to infection with human immunodeficiency virus (HIV), for example, [8–12] and so on. These models have been used to explain different phenomena. For more references and some detailed mathematical analysis on such models, we refer to the survey papers by Kirschner [13] and Perelson and Nelson [14]. Waziri [15] developed a mathematical model of AIDS dynamics with treatment and vertical transmission and recently Miron & Smith [16] use mathematical modeling to describe the interaction between T cells, HIV-1 and protease inhibitors.

In this paper, we develop a mathematical model SEIA (susceptible- exposed-infectious-AIDS infected) of AIDS epidemic with vertical transmission. A method for finding the equilibrium point given the transmission term η(A) was provided through a proof of a theorem. An example to illustrate use of the theorem is also given. The disease-free equilibrium of the model is investigated by finding next generation matrix and basic reproduction number $\Re_0$ of the model.

The paper is organized as follows: Introduction is given in Section 1, the basic assumptions and parameters of the model is discussed in Section 2, the epidemic model is developed in section 3, Section 4 establishes the stability of the system developed, numerical simulations is given in Section 5, and finally conclusion in Section 6.

2. Model parameter and assumptions

We formulate an AIDS model with vertical transmission by considering the adult population in one group. At time t, there are $S(t)$ adult susceptibles, $E(t)$ latent or exposed phase, during which the individual is said to be infected but not infectious, $I(t)$ infectives who are the infected and infectious individuals that have not yet developed AIDS symptoms and $A(t)$ AIDS cases who are infected and with AIDS symptoms. Susceptibles have sexual contacts at a rate with a probability of transmission at one sexual encounter denoted by β. A proportion of these sexual contacts are with infectives. Assume that this proportion is equal to the prevalence of infectives in the population. The model upholds the common assumption of assuming no sexual contacts with AIDS cases though the role of sexual contacts with persons with AIDS symptoms may become important with advances in medical interventions. Sexual contacts within susceptibles do not result in any transmission and thus do not feature in the model. Also, sexual contacts within infectives which gives rise to issues about the role of reinfection are ignored. Denote the probability of transmission β and the contact rate c at the onset of the epidemic as β_0 and c_0, respectively. Assume that, at future points in time differs from $\beta_0 c_0$ at the beginning of the epidemic and that there is an inverse relationship between βc and the number of AIDS cases, i.e. βc decreases with an increasing number of AIDS cases and increases with decreasing numbers of AIDS cases. The decrease in with increases in number of AIDS cases is attributed to awareness and behaviour change. The increase in βc with decreasing numbers of AIDS cases is attributed to complacency. Our model allows for a generalized form of βc, which as per the above assumptions is a function of the number of AIDS cases denoted by $\eta(A)$. The objective is to investigate the implication of the dependence of the risk of transmission of HIV on the number of persons with AIDS symptoms in a community [17,18].

3. Model equations

In this section the SEIA model including vertical transmission is explained along with an exploration of the differential equations describing the flow from one compartment to another. The flow of this model is depicted in Fig. 1, and the system of equations as per our assumption is as follows:

$$\frac{dS}{dt} = \Lambda(t) - \eta(A)SI/N - \mu S \tag{I}$$

$$\frac{dE}{dt} = \eta(A)SI/N - \mu E - \delta E \tag{II}$$

$$\frac{dI}{dt} = \delta E + \varepsilon I - (\mu + \gamma + \xi)I \tag{III}$$

$$\frac{dA}{dt} = \xi I - (\mu + \sigma)A \tag{IV}$$

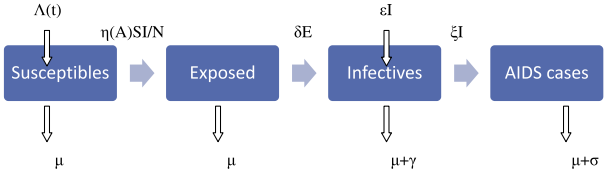

Figure 1 Schematic diagram for the flow of AIDS in the population.

In this model the host population (N) is taken into four compartments: susceptible, exposed, HIV infectious, and AIDS infected, with the numbers of individuals in a compartment, or their densities denoted respectively by $S(t)$, $E(t)$, $I(t)$, $A(t)$, that is $N = S(t) + E(t) + I(t) + A(t)$.

3.1. Susceptible ($S(t)$)

Consider a variable recruitment rate $\Lambda(t)$ to the susceptible population per unit time. Recruited individuals consist of maturing young person's joining the sexually active age group at a predetermined age. The recruitment term can be rewritten in terms of birth rates, maturation rates and rates of mother-to-child transmission with time lags.

Susceptibles are removed through latent period or by natural death. We let μ be the natural death rate for the sexually active adults. The removal rate of susceptibles through latent period is the number of new HIV infections per unit time. This rate is important in calculating HIV incidence which by definition is the number of new infected persons in a specified time period divided by the number of uninfected persons that were exposed for this same time period.

3.2. Exposed ($E(t)$)

Let each susceptible have c sexual contacts per unit time. Assume that a proportion of these contacts are with infectives and at each of these sexual contacts with infectives, a susceptible has a probability β of getting infected. Let βc be a function of the number of AIDS cases given by η(A), then the total probability of one susceptible getting HIV infected from any of their sex contacts per unit time is $\eta(A(t))I/N$. This is the expression for the force of infection. The force of infection is the probability that a susceptible will get an HIV exposed per unit time. Therefore in a population of susceptibles, the number of new HIV exposed per unit time is given by $\eta(A(t))IS/N$.

3.3. Infectives ($I(t)$)

Infectives are recruited through new HIV infections from exposed compartment at rate δ and from vertical transmission at rate ε. It is removed through progression to AIDS at rate ξ and through natural death at rate μ and death rate γ due to HIV infectives. Hence, $1/\delta$ is the duration spent in the infective stage and $1/\mu$ is the life expectancy of the adult population. Both of these rates are assumed constant in the model.

3.4. AIDS cases ($A(t)$)

AIDS cases are recruited through progression from the infective stage to the AIDS stage and removed through AIDS accelerated deaths at rate $\sigma + \mu$ where $1/\delta$ is the average duration spent in the AIDS stage if natural deaths are assumed constant in the model.

Analysis of the model: Let us focus on the equilibrium state of the system and its stability. Suppose that at the equilibrium state in (I, A) the number of susceptibles S continues to increase and hence both S and N vary.

Setting the derivative in equation (IV) to zero, we get

$$A^* = \frac{\xi}{(\mu+\sigma)} I^* \tag{1}$$

$$Or, \quad I^* = \frac{(\mu+\sigma)}{\xi} A^* \tag{2}$$

Similarly, setting the derivative in equation (III) to zero, we get

$$E^* = \frac{(\mu+\gamma+\xi-\varepsilon)}{\delta} I^* \tag{3}$$

And setting the derivative in equation (II) to zero, we get

$$E^* = \frac{\eta(A^*)SI^*}{(\mu+\delta)N} \tag{4}$$

From Eqs. (3) and (4), we get

$$\frac{S}{N} = \frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta(A^*)} \tag{5}$$

But

$$\frac{S}{N} + \frac{I^*}{N} + \frac{E^*}{N} = 1 \tag{6}$$

Using Eqs. (1)–(6), we get

$$\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta(A^*)} + \frac{(\delta+\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)A^*}{\delta\xi N} = 1 \tag{7}$$

Theorem 1. *Assuming for equations (I, II, III, IV), $S(t) \to \infty$ as $t \to \infty$ Further suppose that $\eta(A)$ has an inverse, η^{-1} on (0,1). Then there exists $t_A > 0$ and $\varepsilon_A > 0$ such that for all $t > t_A$,*

$$|A(t) - A(t_A)| < \varepsilon_A, |I(t) - I(t_A)| < \varepsilon_A \text{ and } |E(t) - E(t_A)| < \varepsilon_A.$$

Moreover $S(t) \to \infty$ as $t \to t_A$ [18].

Proof. By our assumption, $S(t) \to \infty$ *as* $t \to \infty$, then from (6)

$$S/N \to 1 (and\ E/N \to 0, I/N \to 0, A/N \to 0).$$

From (5), it follows that $\eta(A(t)) \to (\mu + \sigma)(\mu + \gamma + \xi - \varepsilon)/\delta = \eta(A^*)$ as $t \to \infty$.

Hence $A^* = \eta^{-1}(\mu + \sigma)(\mu + \gamma + \xi - \varepsilon)/\delta$ and for t_A sufficiently large, there exists an $\varepsilon_1 > 0$ such that for all $t > tA$, $|A(t) - A(t_A)| < \varepsilon_1$.

But $I^* = ((\mu + \sigma)/\xi)A^* = ((\mu + \sigma)/\xi)\eta^{-1}(\mu + \sigma)(\mu + \gamma + \xi - \varepsilon)/\delta$

Hence for sufficiently large t_A, for all $t > t_A$,

$|I(t) - I(t_A)| < ((\mu + \sigma)/\xi)\varepsilon_1 = \varepsilon_2$ (say)

Now from Eq. (3) $E^* = ((\mu + \gamma + \xi - \varepsilon)/\delta)I^*$,

Hence for sufficiently large t_A, for all $t > t_A$

$|E(t) - E(t_A)| < ((\mu + \gamma + \xi - \varepsilon)/\delta)\varepsilon_2 = \varepsilon_3$ (say)

Choose $\varepsilon_3 = \varepsilon_A$ since $\varepsilon_3 > \varepsilon_2 > \varepsilon_1$.

Hence there exists $t_A > 0$ and $\varepsilon_A > 0$ such that for all $t > t_A$,

$$|A(t) - A(t_A)| < \varepsilon_A, |I(t) - I(t_A)| < \varepsilon_A \text{ and } |E(t) - E(t_A)| < \varepsilon_A.$$

This completes the proof of first part of the theorem.

Now, from Eq. (6)

$$\frac{S}{(S+I^*+E^*)}=\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta(A^*)}$$

On simplifying it, we get

$$S=\frac{(E^*+I^*)(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta(A^*)-(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}$$

Now since $\eta(A(t))\to(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)/\delta=\eta(A^*)$ *as* $t_A\to\infty$, then

$S(t)\to\infty$ *as* $t\to t_A$.

This completes the proof of second part of the theorem. □

It follows from the above theorem that equilibrium points are

$$A^*=\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$
$$I^*=\left(\frac{\mu+\sigma}{\xi}\right)\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$
$$E^*=\frac{(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$

Example 1. Let $\eta(A)=\eta_0-\eta_1A$ be a linear function of A, where η_0 & η_1 are any constants, then its inverse function is $\eta^{-1}=\frac{\eta_0}{\eta_1}-\frac{\eta}{\eta_1}$.

From the above theorem,

$$A^*=\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$
$$=\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}$$
$$I^*=\left(\frac{\mu+\sigma}{\xi}\right)\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$
$$=\left(\frac{\mu+\sigma}{\xi}\right)\left(\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}\right)$$
$$E^*=\frac{(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)$$
$$=\frac{(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\left(\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}\right).$$

This result can alternatively be derived by setting $\frac{dE}{dt}=0,\frac{dI}{dt}=0$ and $\frac{dA}{dt}=0$ simultaneously with the assumption that $S/N\to1$ as $t\to\infty$, as shown below:

Rewrite equations for E, I and A as:

$$\frac{dE}{dt}=\eta(A)SI/N-\mu E-\delta E \tag{8}$$
$$\frac{dI}{dt}=\delta E+\varepsilon I-(\mu+\gamma+\xi)I \tag{9}$$
$$\frac{dA}{dt}=\xi I-(\mu+\sigma)A \tag{10}$$

Setting $\frac{dA}{dt}=0$ in Eq. (10), we get

$$I=\frac{(\mu+\sigma)}{\xi}A \tag{11}$$

Setting $\frac{dI}{dt}=0$ in Eq. (9), we get

$$E=\frac{(\mu+\gamma+\xi-\varepsilon)}{\delta}I=\frac{(\mu+\gamma+\xi-\varepsilon)}{\delta}\frac{(\mu+\sigma)}{\xi}A \tag{12}$$

Setting $\frac{dE}{dt}=0$ in Eq. (8), we get

$$\frac{\eta(A)SI}{N}-\mu E-\delta E=0$$

Now, putting the value of E from Eq. (12), we get

$$\frac{\eta(A)SI}{N}=\frac{(\mu+\delta)(\mu+\gamma+\xi-\varepsilon)}{\delta}I$$

Putting $N=S+I+E$, $A=0$(*Initially*), we get

$$\eta(A)S=\frac{(\mu+\delta)(\mu+\gamma+\xi-\varepsilon)(S+I+E)}{\delta}$$

Putting the values of I and E from Eqs. (11) and (12), we get

$$\eta(A)S=\frac{(\mu+\delta)(\mu+\gamma+\xi-\varepsilon)}{\delta}\times\left[S+\left(\frac{(\mu+\sigma)}{\xi}+\frac{(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\right)A\right]$$

On simplifying it, we get

$$A=\frac{\left[\eta_0-\frac{(\mu+\delta)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right]S}{\eta_1S+\frac{(\mu+\delta)(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\left[1+\frac{(\mu+\gamma+\xi-\varepsilon)}{\delta}\right]}$$

On dividing top and bottom by S and taking $S\to\infty$, we get

$$A^*=\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}$$
$$I^*=\left(\frac{\mu+\sigma}{\xi}\right)\left(\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}\right)$$
$$E^*=\frac{(\mu+\gamma+\xi-\varepsilon)(\mu+\sigma)}{\delta\xi}\left(\frac{\eta_0}{\eta_1}-\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta\eta_1}\right)$$

Thus, the (A^*, I^*) obtained by this direct method is the same as solutions got by using above theorem.

4. Stability of the Model

Consider the region $\emptyset=\{(S,E,I,A)\in\mathbb{R}^4_+ : N\leqslant\Lambda/\mu\}$.

The AIDS model has a disease-free-equilibrium given by $\varepsilon_0=(\Lambda/\mu,0,0,0)$. It is obvious that the ε_0 attracts the region $\emptyset_0=\{(S,E,I,A)\in\emptyset: E=I=A=0\}$.

The linear stability of ε_0 is governed by the basic reproduction number $\Re_0$. The stability of this equilibrium will be investigated using the next generation matrix operator [19].

Following Diekmann [19], we call FV^{-1} the next generation matrix for the model and we shall set R_0 as equal to the spectral radius $\rho(FV^{-1})$ i.e,

For our model system, the matrices F and V, for the new infection terms and the remaining transfer terms are respectively given by

$$F=\begin{bmatrix}0 & \eta(\mathrm{A}) & \eta'(\mathrm{A})\\ 0 & 0 & 0\\ 0 & 0 & 0\end{bmatrix}\quad\text{and}$$

$$V=\begin{bmatrix}\mu+\delta & 0 & 0\\ -\delta & \mu+\gamma+\xi-\varepsilon & 0\\ 0 & -\chi & \mu+\sigma\end{bmatrix}.$$

Here $\eta(A) = \eta_0 - \eta_1 A$ and at disease-free equilibrium $\eta(A) = \eta_0$ and $\eta'(A) = 0$.

It follows that the basic reproduction number, denoted by R_0 is given by

$$R_0 = \rho(FV^{-1}) = \eta_0/(\mu + \gamma + \xi - \varepsilon).$$

Using Theorem 2 in [20], the following result is established.

Lemma 1. *The disease-free equilibrium of the AIDS model system is locally asymptotically stable if $R_0 \leqslant 1$ and unstable if $R_0 > 1$.*

According to above Lemma 1, our model will be locally asymptotically stable if

$$\eta_0 \leqslant (\mu + \gamma + \xi - \varepsilon).$$

Now, except for a disease-free equilibrium $\varepsilon_0 = (\Lambda/\mu, 0, 0, 0)$, by straightforward computation, our model has unique positive equilibrium $\varepsilon_e = (S^*, E^*, I^*, A^*)$ for $R_0 > 1$, where

$$A^* = \frac{\eta_0}{\eta_1}\left[1 - \frac{(\mu+\sigma)}{\Re_0 \delta}\right],$$

$$I^* = \frac{(\mu+\sigma)}{\xi}\frac{\eta_0}{\eta_1}\left[1 - \frac{(\mu+\sigma)}{\Re_0 \delta}\right],$$

$$E^* = \frac{(\mu+\sigma)\eta_0^2}{\Re_0 \xi \delta \eta_1}\left[1 - \frac{(\mu+\sigma)}{\Re_0 \delta}\right],$$

$$S^* = \frac{\Lambda}{\mu} - \frac{(\mu+\delta)(\mu+\sigma)\eta_0^2}{\Re_0 \mu \xi \delta \eta_1}\left[1 - \frac{(\mu+\sigma)}{\Re_0 \delta}\right].$$

The Jacobian matrix of our model is

$$J(\varepsilon_0) = \begin{bmatrix} -\mu & 0 & -\eta(A) & \eta'(A) \\ 0 & -(\mu+\delta) & \eta(A) & \eta'(A) \\ 0 & \delta & -(\mu+\gamma+\xi-\varepsilon) & 0 \\ 0 & 0 & \xi & -(\mu+\sigma) \end{bmatrix},$$

where $\eta(A) = \eta_0 - \eta_1 A$.

Here, all diagonal elements of the Jacobian matrix are negative. So, all eigenvalues of above Jacobian matrix have negative real part.

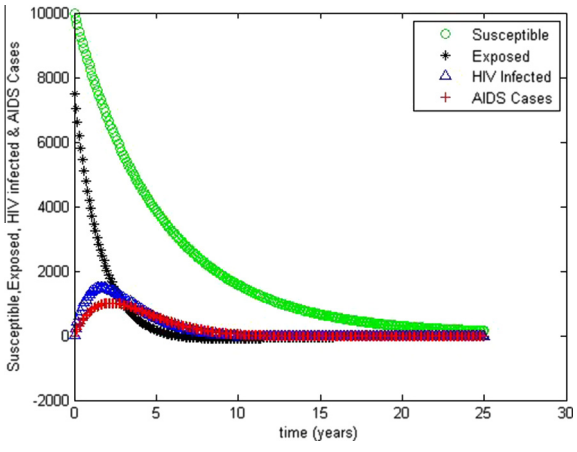

Figure 2 Time series population of different classes.

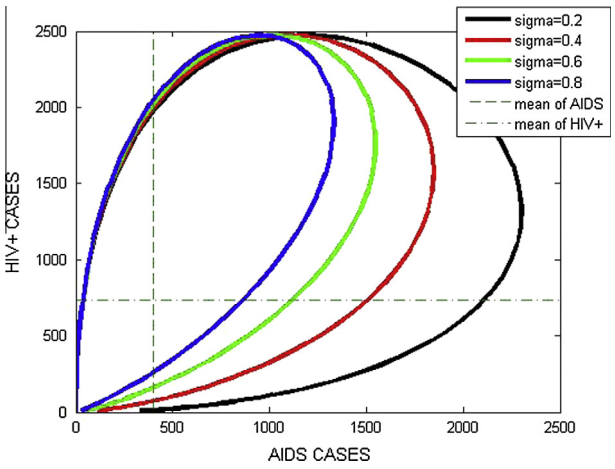

Figure 3 HIV+ versus AIDS cases when $\sigma = 0.2$, 0.4, 0.6 and 0.8.

Hence, the endemic equilibrium of our AIDS model is locally stable [21,22].

5. Numerical simulations and results

Consider the total population size to be $N = 100{,}000$ and the recruitment rate per unit time be $\Lambda(t) = 10$, then initially the susceptible cases is $S(0) = 10{,}000$, exposed cases are $E(0) = 7500$, HIV+ cases is $I(0) = 1$ and AIDS cases is $A(0) = 0$.

Now consider $\eta(A) = 0.827 - 0.0001 * A$, $\mu = 0.2$, $\delta = 0.8$, $\xi = 0.9$, $\sigma = 0.8$, $\varepsilon = 0.2$, $\gamma = 0.01$ and simulating it by Runga Kutta method of order 4 using MATLAB 7.10.0, we get the result as shown in Fig. 2.

We simulate between AIDS cases Versus HIV when $\sigma = 0.2$, $\sigma = 0.4$, $\sigma = 0.6$ and $\sigma = 0.8$ as shown in Fig. 3. The effect of varying the rate of removal of AIDS cases on the oscillations in number of HIV infectives and number of AIDS cases in a community was investigated by studying the $I(t)$ time trends and the $I(t) - A(t)$ phase portraits for varying values of the mean life time spent in the AIDS stage at 2 years ($\sigma = 0.8$), 3 years ($\sigma = 0.6$), 4 years ($\sigma = 0.4$) and 8 years ($\sigma = 0.2$). Stability analysis done by studying the signs of dI/dt and dA/dt in the two regions formed by the I-isocline and A-isocline shows that the resulting trajectory in the $(A - I)$ plane is a clockwise steady spiral toward the equilibrium point (A^*, I^*).

In this paper, we extend the model of Baryarama et al. [17,18] by introducing a new exposed class and vertical transmission on it and also establish the stability of the system developed. The models in [15,23] with vertical transmission did not take into account awareness, behavioral changes and complacency of AIDS cases, whereas, in our model assumption, there is an inverse relationship between βc and the number of AIDS cases. The decrease in βc with increases in number of AIDS cases is attributed to awareness and behavioral changes. The increase in βc with decreasing numbers of AIDS cases is attributed to complacency. Our model allows for a generalized form of $\beta c = \eta(A)$. The objective is to investigate the implication of the dependence of the risk of transmission of HIV on the number of persons with AIDS symptoms in a community.

6. Conclusion

In this paper, a mathematical model SEIA (susceptible-exposed-infectious-AIDS infected) with vertical transmission of AIDS epidemic in adult population is formulated. A stated theorem with proof and example to illustrate is given to find the equilibrium points in terms of invertible transmission function $\eta(A)$. The equilibrium point in terms of η^{-1} is $\left(\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right), \left(\frac{\mu+\sigma}{\xi}\right)\eta^{-1}\left(\frac{(\mu+\sigma)(\mu+\gamma+\xi-\varepsilon)}{\delta}\right)\right)$ in the (A, I) plane. Hence, the equilibrium point is readily found for any invertible $\eta(A)$ as we have shown for linear $\eta(A) = \eta_0 - \eta_1 A$. The disease-free equilibrium of the model is investigated by finding next generation matrix and basic reproduction number R_0 of the model. The disease-free equilibrium is locally asymptotically stable if $\Re_0 \leqslant 1$ and unstable if $\Re_0 > 1$. Stability analysis shows that (A^*, I^*) approaches through a clockwise steady spiral. This is verified by numerical results.

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