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Mathematical Analysis of an HIV/AIDS Epidemic Model

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Abstract Mathematical model is a very useful tool to understand and analyse the dynamics of diseases. Here we present a non-linear mathematical model which investigates the spread and control of HIV in different populations. Present study fitted the model, which exhibits two equilibrium points namely, the disease free equilibrium and the endemic equilibrium point. The global stability of these equilibrium points is also investigated. The model is analysed by using the basic reproduction number R_0.

Keywords HIV, AIDS, Basic reproduction number, Endemic equilibrium point, Local and global stability

1. Introduction

HIV is a well known disease throughout the world. According to the World Health Organization (WHO), since the beginning of the epidemic, almost 78 million people have been infected with the HIV virus and about 39 million people have died of HIV. Globally, about 35.0 million people were living with HIV at the end of 2013. An estimated 0.8% of adults aged 15-49 years worldwide are living with HIV, although the burden of the epidemic continues to vary considerably between countries and regions. Sub-Saharan Africa remains most severely affected, with nearly 1 in every 20 adults living with HIV and accounting for nearly 71% of the people living with HIV worldwide. So HIV is one of the greatest threats to the human society.

Infection with HIV occurs by the transfer of blood, semen, vaginal fluid, pre-ejaculate or breast milk. Within these body fluids, HIV is present as both free virus particles and virus within infected immune cells [2]. HIV can be transmitted between injecting drug users if the drug equipment (needles, syringes or rinsing water) is contaminated by HIV-infected fluids (usually blood) and is then reused by another person without first sterilizing it.

Mother to child transmission of HIV is also possible. According to the UNICEF, nine out of every 10 children under the age of 15 become infected with HIV every year because their mothers are HIV positive. This is known as "mother to child transmission" of HIV. In 2001 alone, 710,000 children under 15 contracted HIV with while in their mother's womb, during childbirth or through breastfeeding.

After entering into the human body, HIV infects vital cells (especially $CD4^+$ T cells), macrophages and dendritic cells. in the human immune system such as helper T cells HIV infection leads to low levels of $CD4^+$ T cells through a number of mechanisms including: apoptosis of uninfected by stancer cells, direct viral killing of infected cells, and killing of infected $CD4^+$ T cells by CD8 cytotoxic lymphocytes that recognize infected cells. When $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost, and the body becomes progressively more susceptible to opportunistic infections [1].

Without treatment, HIV infection gradually destroys the immune system. Standard HIV treatment (also called antiretroviral therapy or ART) involves taking a combination of HIV medicines from at least two different HIV drugs classes every day. ART is highly effective at preventing HIV from multiplying. Having less HIV in the body protects the immune system and prevents HIV from advancing to AIDS. ART also reduces the risk of HIV drug resistance.

2. Formulation of the Model

The total sexually-active population at time t, is denoted by $N(t)$. This population $N(t)$ is divided into four mutually-exclusive compartments, namely susceptible class S(t), the infected individuals who do not know that they are infected $I_1(t)$, the infected individuals who do know that they are infected $I_2(t)$, and that of the AIDS populations $A(t)$. Hence the total population at time t, $N(t)$ can be written as $N(t) = S(t) + I_1(t) + I_2(t) + A(t)$.

It is assumed that individuals are recruited at a constant rate Π to the susceptible class S(t). Susceptible individuals can be infected with HIV following the contact with infected individuals at a rate λ, where $\lambda = \frac{\beta_1 I_1 + \beta_2 I_2}{N}$, where β_1, β_2 are the transmission rates for HIV. The individuals in the I_2 class are more infectious than those in the I_1 class. Therefore we must have $\beta_1 < \beta_2$. Suppose that the

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individuals of the $I_1(t)$ class enter into the $I_2(t)$ class at a rate ω and into the AIDS class $A(t)$ at a rate δ_1. Again suppose that the individuals of the $I_2(t)$ class progress into the AIDS class at a rate δ_2. Let μ and d denote the natural mortality rate and disease induced death rate respectively. Under the above considerations, the model dynamics is assumed to be governed by the following system of ordinary differential equations

$$\frac{ds}{dt} = \Pi - \lambda S - \mu S,$$
$$\frac{dI_1}{dt} = \lambda S - \omega I_1 - \delta_1 I_1 - \mu I_1$$
$$\frac{dI_2}{dt} = \omega I_1 - \delta_2 I_2 - \mu I_2 \dots\dots\dots\dots\dots\dots\dots\dots\dots\dots\dots (1)$$
$$\frac{dA}{dt} = \delta_1 I_1 + \delta_2 I_2 - (\mu + d)A$$

3. Analysis of the Model

3.1. Invariant Region

All the parameters of the model are assumed to be non-negative. On the other hand, model (1) monitors human population, so the state variables are non-negative for all time $t \geq 0$.

Total population can be written as

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t) \dots\dots\dots (3.1)$$

Here Eq. (2) is changing at a rate

$$\frac{dN}{dt} = \frac{ds}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dA}{dt} = \Pi - \mu N - dA \dots (3.2)$$

In the absence of disease i.e. for $I_1 = I_2 = A = 0$, we have

$$\frac{dN}{dt} \leq \Pi - \mu \dots\dots\dots\dots\dots\dots\dots\dots\dots (3.3)$$

By the separation of variables of differential inequality (3.3) we must have

$$\frac{dN}{\Pi - \mu N} \leq dt$$

Integrating the above equation we have

$$\Pi - \mu N \geq Ce^{-\mu t}$$

where C is a constant which is to be determined. Let at $t = 0,\ N = N_0$. So we have,

$$C = \Pi - \mu N_0.$$

From Eq. (3.4) we have

$$\Pi - \mu N \geq (\Pi - \mu N_0)e^{-\mu t}$$
$$\Rightarrow N(t) \leq \frac{\Pi}{\mu} - \left[\frac{\Pi - \mu N_0}{\mu}\right] e^{-\mu t}$$

As $t \to \infty, 0 \leq N(t) \leq \frac{\Pi}{\mu}$.

Therefore, the feasible solutions set of the system (1) enters the region:

$$\Gamma = (S, I_1, I_2, A) \in \Re_+^4 : N \leq \frac{\Pi}{\mu}.$$

In this case, whenever $N(t) \leq \frac{\Pi}{\mu}$, every solution with initial condition in $\Re_+^4$ remains in that region for $t > 0$. Thus the model is well posed and epidemiologically meaningful in the domain Γ.

3.2. Positivity of Solutions

In this section, we discuss the positivity of the solutions which describes the non-negativity of the solutions of the system (1). Let the initial data be $\{S(0), I_1(0), I_2(0), A(0)\} \in \Gamma$. Then we need to show that the solution set $\{S(t), I_1(t), I_2(t), A(t)\}$ of system (1) is positive for $t > 0$.

From the first equation of the system (1) we have

$$\frac{ds}{dt} = \Pi - \lambda S - \mu S \geq -(\lambda + \mu)S$$
$$\Rightarrow \frac{ds}{dt} \geq -(\lambda + \mu)S$$

Integrating we have s

$$S(t) \geq S(0)e^{\int -(\lambda+\mu)dt} > 0$$

provided $(\lambda + \mu) < \infty$.

Similarly from the 2nd, 3rd and 4th equation of model (1) we have:

$I_1(t) \geq I_1(0)e^{\int -(\omega+\delta_1+\mu)dt} > 0$ provided $(\omega + \delta_1 + \mu) < \infty$,

$I_2(t) \geq I_2(0)e^{\int -(\delta_2+\mu)dt} > 0$ provided $(\delta_2 + \mu) < \infty$ and finally

$A(t) \geq A(0)e^{-\int(\mu+d)dt} > 0$ provided $\mu + d > 0$ respectively.

Thus the solution set $\{S(t), I_1(t), I_2(t), A(t)\}$ of system (1) is positive for all $t > 0$.

4. Disease-Free Equilibrium Point

Disease-free equilibrium (DFE) points of a disease model are its steady-state solutions in the absence of infection or disease. We denote the DFE point by E_0 and define the "diseased" classes that are either exposed or infectious.

For all equilibrium points we have $I_1 = I_2 = A = 0$. The positive disease-free equilibrium point for the model (1) is $N = \Pi/\mu$.

The model (1) has exactly one disease-free equilibrium point and the equilibrium point is $E_0 = \left(\frac{\Pi}{\mu}, 0, 0, 0\right)$. We need to show that the DFE is the only equilibrium point of the model (1). Substituting $E_0 = \left(\frac{\Pi}{\mu}, 0, 0, 0\right)$ in the model (1) shows all derivatives equal to zero; hence DFE is an equilibrium point. From the above lemma, the only equilibrium point for N is $\frac{\Pi}{\mu}$. Thus the only equilibrium

point for the model (1) is DFE [3].

5. Local Stability of DFE Point

The local stability of the model (1) is highly dependent on the basic reproduction number which is denoted by R_0. The associated non-negative matrix F, for the new infection terms and the non-singular M-matrix, V, for the remaining transfer terms, are given, respectively by

$$F = \begin{pmatrix} \frac{\beta_1 \Pi}{N\mu} & \frac{\beta_2 \Pi}{N\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$V = \begin{pmatrix} \omega + \delta_1 + \mu & 0 & 0 \\ -\omega & \delta_2 + \mu & 0 \\ -\delta_1 & -\delta_2 & \mu + d \end{pmatrix}$$

The basic reproduction number R_0, is the spectral radius of the matrix FV^{-1}. The Eigenvalues of the matrix FV^{-1} are

$$R_m = \rho(FV^{-1}) = \begin{pmatrix} 0 \\ 0 \\ \frac{\beta_1 \Pi \delta_2 + \beta_1 \Pi \mu + \beta_2 \Pi \omega}{N\mu(\omega\delta_2 + \omega\mu + \delta_1\delta_2 + \delta_1\mu + \delta_2\mu + \mu^2)} \end{pmatrix}$$

So the basic reproduction number is

$$R_0 = \frac{\beta_1 \Pi \delta_2 + \beta_1 \Pi \mu + \beta_2 \Pi \omega}{N\mu(\omega\delta_2 + \omega\mu + \delta_1\delta_2 + \delta_1\mu + \delta_2\mu + \mu^2)}$$

The disease-free equilibrium point R_0 of the model (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

6. Global Stability of DFE Point

The global asymptotically (GAS) of the disease-free state of the model is investigated using the theorem by Castillo-Chavez [1]. So from the model (1) we have:

$$G^*(X,Z) = \begin{pmatrix} (\beta_1 I_1 + \beta_2 I_2)(1 - \frac{S}{N}) \\ 0 \\ 0 \end{pmatrix} = \begin{pmatrix} G_1^*(X,Z) \\ G_2^*(X,Z) \\ G_3^*(X,Z) \end{pmatrix}$$

Since $G_1^*(X,Z) > 0$, $G_2^*(X,Z) > 0$ and $G_3^*(X,Z) > 0$, so the condition is satisfied. So E_0 is globally asymptotically stable when $R_0 < 1$.

7. Endemic Equilibrium of the Model

The endemic equilibrium of the model is studied by using the Central Manifold Theorem [1]. To apply this theorem we need to change the variables as follows:

$x_1 = S, x_2 = I_1, x_3 = I_2, x_4 = A,$ so that

$N(t) = x_1(t) + x_2(t) + x_3(t) + x_4(t).$

Then the model (1) can be written in the form

$\frac{dX}{dt} = F(x),$ where

$X = (x_1, x_2, x_3, x_4)$ and $F = (f_1, f_2, f_3, f_4)$ as

$$\frac{dx_1}{dt} = \Pi - \lambda x_1 - \mu x_1,$$
$$\frac{dx_2}{dt} = \lambda x_1 - \omega x_2 - \delta_1 x_2 - \mu x_2,$$
$$\frac{dx_3}{dt} = \omega x_2 - \delta_2 x_3 - \mu x_3 \ldots\ldots\ldots\ldots (7.1)$$
$$\frac{dx_4}{dt} = \delta_1 x_2 + \delta_2 x_3 - (\mu + d) x_4$$

where $\lambda = \frac{\beta_1 x_2 + \beta_2 x_3}{N}$.

The Jacobian of the system (7.1) is

$$J = \begin{pmatrix} -\mu & -\beta_1 & -\beta_2 & 0 \\ 0 & \beta_1 - \omega - \delta_1 - \mu & \beta_2 & 0 \\ 0 & \omega & -\delta_2 - \mu & 0 \\ 0 & \delta_1 & \delta_2 & -\mu - d \end{pmatrix}$$

To analyse the dynamics of (7.1), we compute the Eigen values of the Jacobian of (7.1) at the disease free equilibrium point. It can be shown that this Jacobian has a right eigenvector given by:

$V = (v_1, v_2, v_3, v_4)^T$, where

$v_1 = -\left[\frac{\beta_1}{\mu\omega}(\delta_2 + \mu) + \frac{\beta_2}{\mu}\right] v_3,$

$v_2 = \frac{\delta_2 + \mu}{\omega} v_3$ and

$v_4 = \frac{1}{\mu + d}\left[\frac{\delta_1\delta_2 + \delta_1\mu}{\omega} + \delta_2\right] v_3.$

The left eigenvectors are given by

$W = (w_1, w_2, w_3, w_4)^T,$ where

$w_1 = 0,$

$w_2 = \frac{\delta_2 + \mu}{\beta_2} w_3,$

$w_4 = 0.$

Now using (7.1) we have

$$s^* = \frac{2}{\Pi}(w_2 + w_3)(\beta_1 w_2 + \beta_2 w_3)(v_1 - v_2)$$
$$= \frac{2}{\Pi} \times \left(\frac{\delta_2 + \mu}{\beta_2} + 1\right) w_3 \times \left(\beta_1 \frac{\delta_2 + \mu}{\beta_2} + \beta_2\right) w_3 \times \{-[\tfrac{\beta_1}{\mu\omega}(\delta_2 + \mu) + \tfrac{\beta_2}{\mu}] - \tfrac{\delta_2 + \mu}{\omega}\}$$
$$= -\frac{2}{\Pi}\left(\frac{\delta_2 + \mu}{\beta_2} + 1\right) \times \left(\beta_1 \frac{\delta_2 + \mu}{\beta_2} + \beta_2\right) w_3^2 \times \left(\frac{\beta_1\delta_2 + \beta_1\mu + \beta_2\omega + \delta_2\mu + \mu^2}{\mu\omega}\right)$$
$$< 0.$$

and $r^* = \beta_1\beta_2 v_2 v_4 > 0$.

Thus the model (7.1) satisfies the required criteria.

So the model (1) has a unique endemic equilibrium point which is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$ [4].

Table 1. Description of variables

Variables	Description
S(t)	Susceptible class.
$I_1(t)$	Infected individuals who do not know that they are infected.
$I_2(t)$	Infected individuals who know that they are infected.
A(t)	AIDS class.

Table 2. Values of parameter of model

Parameters	Description	Values
Π	Recruitment rate	4000/(1000*365)
β_1	Transmission rate of HIV in $I_1(t)$ class	0.28 [3]
β_2	Transmission rate of HIV in $I_2(t)class$	0.35 [2]
ω	Transfer rate between I_1 and I_2.	0.9 [2]
δ_1	Transfer rate between I_1 and A.	0.7 [4]
δ_2	Transfer rate between I_2 and A.	0.4 [1]
μ	Natural mortality rate	0.000027 [1]
d	Disease induced death tare	60000/(1000*365 *0.000027)

8. Numerical Simulation and Discussions

Here ω is the transmission rate of HIV from $I_1(t)$ to $I_2(t)$ class. The effect of ω is monitored in the figure (1) and (2). From the figure it is observed that if we increase the value of ω, then the value of R_0 decreases. Hence the number of infected population also decreases.

δ_2 is the transmission of the disease from I_2 class to the AIDS class $A(t)$. In the figure (3) and (4), the effect of δ_2 is observed. The increment in the value of δ_2, increases the basic reproduction number R_0. As a result it increases the number of total infected population.

The prevalence of the disease is being monitored in the figure (5) and (6).

In this paper, a non-linear mathematical model is formulated. Sufficient conditions are given ensuring the local and global stability of the disease free equilibrium point and unique endemic equilibrium point. The disease free equilibrium point E_0 is shown locally asymptotically stable when the basic reproduction number R_0 is less than unity. The global stability is also investigated. Finally we have shown that the model has a unique endemic equilibrium point which is locally asymptotically stable.

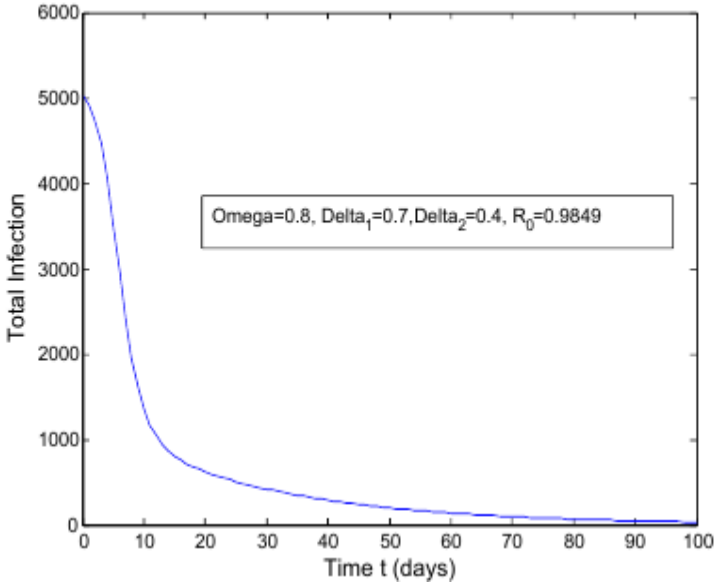

Figure 1. Total infection as a function of time for model (1) with different values of ω, where $\delta_1 = 0.7$, $\delta_2 = 0.4$

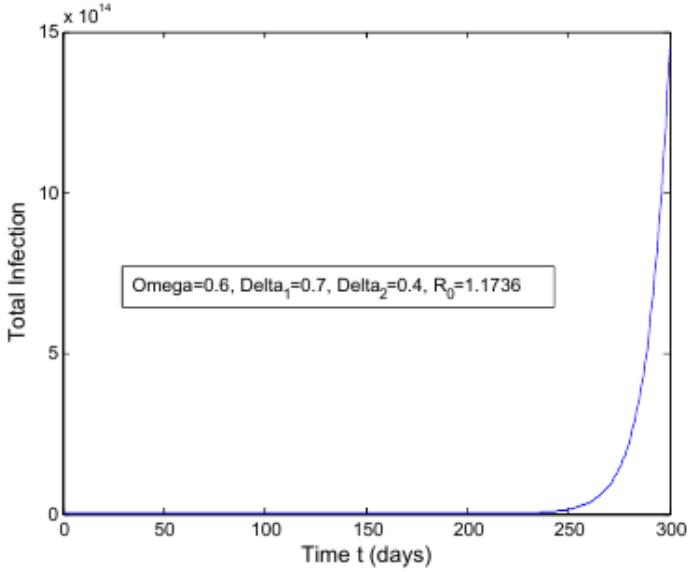

Figure 2. Total infection as a function of time for model (1) with different values of ω, where $\delta_1 = 0.7, \delta_2 = 0.4$

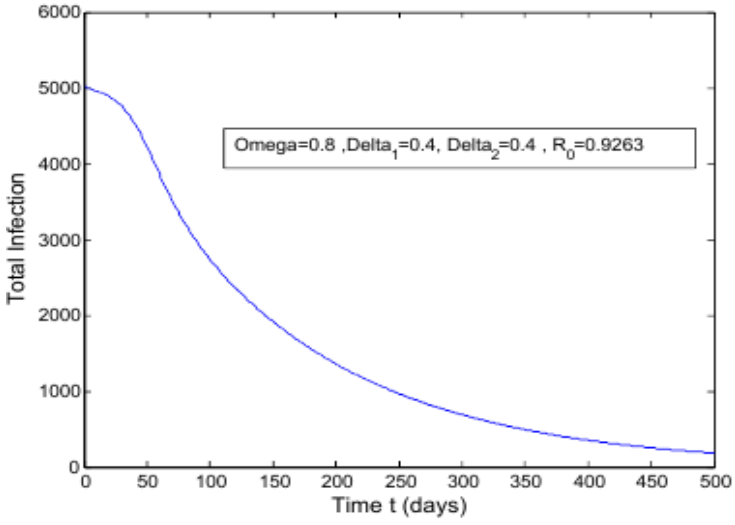

Figure 3. Total infection as a function of time for model (1) with different values of δ_1, where $\omega = 0.8, \delta_2 = 0.4$

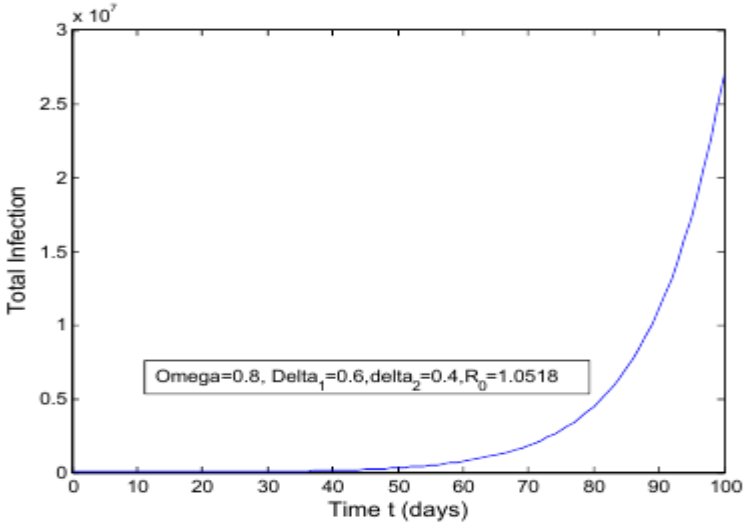

Figure 4. Total infection as a function of time for model (1) with different values of δ_1

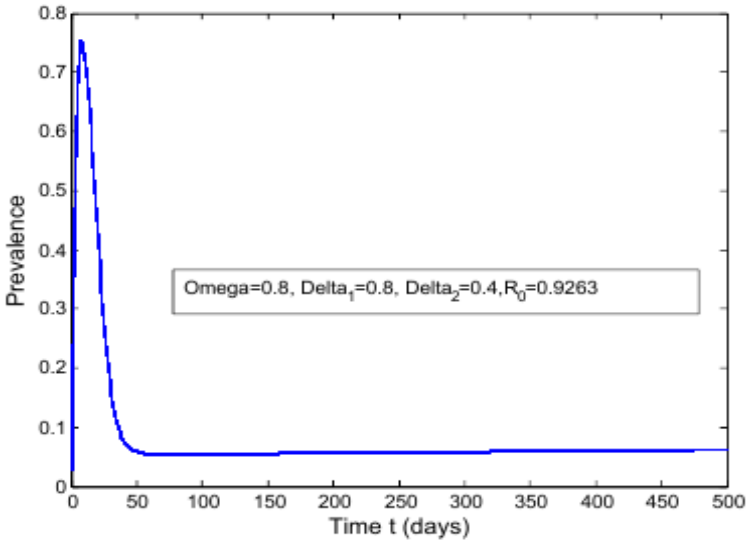

Figure 5. Prevalence as a function of time for model (1) with different values of δ_1, where $\omega = 0.8, \delta_2 = 0.4$

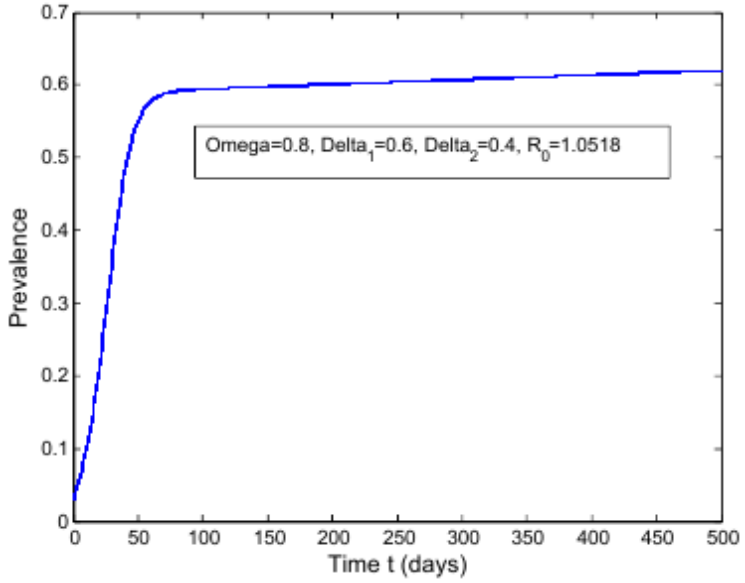

Figure 6. Prevalence as a function of time for model (1) with different values of δ_1, where $\omega = 0.8, \delta_2 = 0.4$

9. Conclusions

In summary, the main findings of the paper are given below:

- We can control the disease burden by controlling the effective contact rate of the infected population.
- Controlling the transfer rate δ_2 of I_2 class to the AIDS class $A(t)$ the disease elimination if feasible.
- The most effective way to lower the incidence rate is to isolate the susceptible population as soon as possible.

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