Local Stability of AIDS Epidemic Model Through Treatment and Vertical Transmission with Time Delay

To cite this article: Cascarilla Novi W and Dwi Lestari 2016 J. Phys.: Conf. Ser. 693 012010

View the article online for updates and enhancements.
Local Stability of AIDS Epidemic Model Through Treatment and Vertical Transmission with Time Delay

Cascarilla Novi W¹ and Dwi Lestari²
Department of Mathematics Education, Yogyakarta State University, Yogyakarta, Indonesia
E-mail: ¹cascarillanw@gmail.com ²dwilestari@uny.ac.id

Abstract. This study aims to explain stability of the spread of AIDS through treatment and vertical transmission model. Human with HIV need a time to positively suffer AIDS. The existence of a time, human with HIV until positively suffer AIDS can be delayed for a time so that the model acquired is the model with time delay. The model form is a nonlinear differential equation with time delay, SIPTA (susceptible-infected-pre AIDS-treatment-AIDS). Based on SIPTA model analysis results the disease free equilibrium point and the endemic equilibrium point. The disease free equilibrium point with and without time delay are local asymptotically stable if the basic reproduction number is less than one. The endemic equilibrium point will be local asymptotically stable if the time delay is less than the critical value of delay, unstable if the time delay is more than the critical value of delay, and bifurcation occurs if the time delay is equal to the critical value of delay.

1. Introduction
AIDS (Acquired Immune Deficiency Syndrome) is symptoms of diseases caused by decreasing immune system because of HIV (Human Immunodeficiency Virus). HIV/AIDS in Indonesia was discovered firstly in year 1987. It can be transmitted many ways which can be classified into vertical and horizontal. HIV/AIDS transmission horizontally is direct contact with individuals HIV infected through sexual contact or using injection together. On the other hand, vertical transmission can result from direct transfer of HIV/AIDS from an infected mother to an unborn or newborn offspring [1].

Statistical data of AIDS epidemic in Indonesia according to its transmission factor shows that the most HIV infections occur through sexual as many as 35,671 cases, injecting drug users as many as 8,462 cases, and vertical transmission (mother to child) as many as 1,506 cases. In 2003, there were 2,685 HIV cases have been reported. Approximately 90,000-130,000 Indonesian people living with HIV can be estimated that there are 2,250-3,250 babies born infected with HIV. Kementrian Kesehatan Indonesia in 2012 data shows that from 43,624 pregnant women tested for HIV, there are 1,329 (3.01%) of pregnant women suffer positively HIV [2]. Accordingly, the discussion about the AIDS epidemic through vertical transmission need to be considered.

Study about mathematical model of HIV/AIDS transmission from mother to child or vertical transmission with SIPTA (susceptible - Infection - Treatment – Pre AIDS - AIDS) model by [3] and [4]. Individuals with HIV and AIDS can be treated with treatment to strengthen the immune system so that [5] use SITA (Susceptible-Infected-Treatment-AIDS) model at their paper. Other study [6] analyses...
about vertical transmission and gives the assumption of a time delay in humans infected by HIV who need time to positively suffer AIDS.

Along with the development and based on previous study, the AIDS epidemic through treatment and vertical transmission with time delay in human infected by HIV to suffer AIDS need to be considered. Therefore, this study will establish the AIDS epidemic model through treatment and vertical transmission with a time delay. Then, analyze the stability of the equilibrium point at the free population of HIV-AIDS infection and endemic.

2. Discussion

1.1. Model Formulation

AIDS is caused by HIV. HIV-AIDS transmissions can through horizontally or vertically transmission. WHO distinguished the symptoms of human with HIV-AIDS positively become major and minor symptoms. Human who has suffered major and minor symptoms can be called Pre-AIDS or ARC (AIDS Related Complex). ARC is a disease syndrome caused by HIV but has not been included in severe AIDS. Furthermore, the immune system decreases until suffer positively AIDS. That is characterized by suffer serious disease [7]. Until now, there is no medicine that can cure AIDS [8], so there are still individual deaths due to AIDS. Treatment for human infected by HIV-AIDS is a treatment which aims to strengthen the immune system.

In this study, Mathematical model of the AIDS epidemic the population at time $t$ is divided into five compartments. They are susceptible, infected, treatment, pre-AIDS, and AIDS. Susceptible is free infected HIV-AIDS populations. Infected is HIV-infected population. Individuals who are included in infected are susceptible individuals who has horizontal transmission by infected individuals in a population of treatment, pre-AIDS, or AIDS. Individuals who were born from mothers infected HIV-AIDS (vertical transmission) and live included in Infected. Treatment population is HIV-AIDS population who are doing the treatment. Pre-AIDS is an HIV-infected population who has symptoms of AIDS before suffer positively AIDS. While AIDS is a population that suffer positively AIDS.

Then, the following assumptions were used to simplify the model:

- Population is constant and closed. It means the increase or decrease of population occurred only because of birth and death.
- The population is homogeneous, it means that everyone has the same risk to be infected by HIV.
- Individual who was born from mothers infected HIV-AIDS and die was ignored.
- Transmission of HIV-AIDS through sexual contact occurs in individuals in susceptible to individuals in Infected, treatment, pre-AIDS, or AIDS.
- Transmission of HIV-AIDS by using inject together was ignored.
- The death occurred due to natural mortality in each class and death due to AIDS.
- No individual is cured of AIDS.
- Individuals in pre-AIDS and AIDS class were isolated, it means that each individual in the class limit themselves to have not had sexual contact with other individual classes.

The variables and parameters which were used in this study:

- $S(t)$: The number of individuals Susceptible
- $I(t)$: The number of individuals Infected
- $P(t)$: The number of individuals Pre-AIDS
- $T(t)$: The number of individual Treatment
- $A(t)$: The number of AIDS positive individuals
- $N(t)$: Total population.
- $\pi$: The rate of babies born healthy.
\( \mu \): The rate of natural mortality.

\( \lambda \): Infection rate

\( c \): The average frequency of sexual contacts per unit time.

\( \beta \): The rate of sexual contact.

\( 1 - \varepsilon \): The proportion of babies infected HIV and life after birth.

\( \theta \): The rate birth of babies infected HIV.

\( \delta \): The rate of individuals transfer from I

\( \sigma_1 \): The proportion of individuals transfer from population I to P

\( \sigma_2 \): The proportion of individuals transfer from population I to T

\( 1 - \sigma_1 - \sigma_2 \): The proportion of individuals transfer from population I to A

\( \gamma \): The rate of individuals transfer from P

\( m \): The proportion of individuals transfer from population P to T

\( 1 - m \): The proportion of individuals transfer from population P to A

\( k \): The rate of individuals transfer from T to A

\( \nu \): The rate of individuals transfer from A to T

\( \alpha \): The rate of deaths due to AIDS

\( \tau \): Time delay for human with HIV to suffer positively AIDS.

with \( S(t), I(t), P(t), T(t), A(t), N(t) > 0 \) and \( 0 < \pi, \mu, \lambda, \beta, \varepsilon, \theta, \delta, \sigma_1, \sigma_2, \gamma, m, k, \nu, \alpha, \tau < 1 \).

Based on the real problems and the assumptions, the AIDS epidemic could be described in the following flow diagram:

![Flow diagram of the model](image)

**Figure 1.** Flow diagram of the model

In the assumption, individuals in pre-AIDS and AIDS class were isolated. So that, from the Figure 1 and the assumptions, we can get the following system of differential equations:

\[
\begin{align*}
\frac{dS}{dt} &= \pi N - \mu S - \lambda S \\
\frac{dI}{dt} &= \lambda S + (1 - \varepsilon) \theta I - (\mu + \delta) I \\
\frac{dP}{dt} &= \sigma_1 \delta I - (\mu + \gamma) P \\
\frac{dT}{dt} &= \sigma_2 \delta I + m \gamma P + \nu A - (\mu + k) T \\
\frac{dA}{dt} &= (1 - \sigma_1 - \sigma_2) \delta I + (1 - m) \gamma P + kT - (\mu + \alpha + \nu) A
\end{align*}
\]

(1)

By the infection rate per unit time is

\[ \lambda = \frac{c_1 \beta I}{N} + \frac{c_2 \beta T}{N}. \]
Individual transfer from the Infected (I) population to AIDS (A) population was not only affected by the time \( t \), but also influenced by the time delay \( \tau \) so the system (1) in proportion form became:

\[
\begin{align*}
\frac{ds}{dt} &= \pi - \lambda(t)s(t) - (\pi - \alpha a(t) + (1 - \varepsilon)\theta(t))s(t) \\
\frac{di}{dt} &= \lambda(t)s(t) + (1 - \varepsilon)\theta(t) - \left(\sigma_{1} + \sigma_{2}\right)\delta i(t) - (1 - \sigma_{1} - \sigma_{2})\delta i(t - \tau) \\
\frac{dp}{dt} &= \sigma_{1}\delta i(t) - (\pi + \gamma - \alpha a(t) + (1 - \varepsilon)\theta(t))p(t) \\
\frac{dh}{dt} &= \sigma_{1}\delta i(t) + my p(t) + v d(t) - \left(\pi + k - \alpha a(t) + (1 - \varepsilon)\theta(t)\right)h(t) \\
\frac{da}{dt} &= (1 - \sigma_{1} - \sigma_{2})\delta i(t - \tau) + (1 - m)\gamma p(t) + k h(t) - a(t)\left(\pi + v + \alpha - \alpha a(t) + (1 - \varepsilon)\theta(t)\right) \\
\end{align*}
\]

by the infection rate per unit time is \( \lambda(t) = c_{1}\beta_{i}i(t) + c_{2}\beta_{a}h(t) \) and \( s + i + p + h + a = 1 \)

### 1.2. Equilibrium Point

The following are the equilibrium points obtained from the system (2).

**Theorem 1.**

i. If \( i = 0 \), then the system (2) has a disease-free equilibrium point \( E_{0} = (s, i, p, h, a) = (1, 0, 0, 0, 0) \).

ii. If \( i \neq 0 \), the system (2) has an endemic equilibrium point \( E_{1} = (s^{*}, i^{*}, p^{*}, h^{*}, a^{*}) \), those are

\[
\begin{align*}
\pi - \lambda s - (\pi - \alpha a + (1 - \varepsilon)\theta) s &= 0 \\
\lambda s + (1 - \varepsilon)\theta - (\pi + \delta - \alpha a + (1 - \varepsilon)\theta) i &= 0 \\
\sigma_{1}\delta i - (\pi + \gamma - \alpha a + (1 - \varepsilon)\theta) p &= 0 \\
\sigma_{1}\delta i + my p + v d - (\pi + k - \alpha a + (1 - \varepsilon)\theta) h &= 0 \\
(1 - \sigma_{1} - \sigma_{2})\delta i + (1 - m)\gamma p + k h - (\pi + v + \alpha - \alpha a + (1 - \varepsilon)\theta) a &= 0 \\
\end{align*}
\]

by \( \lambda = c_{1}\beta_{i}i + c_{2}\beta_{a}h \).

i. If \( i = 0 \), substitute into (2a) to (2e), we get \( h = 0, \ s = 1, \ p = 0, \) and \( a = 0 \) hence it proved that the disease-free equilibrium point is \( E_{0} = (1, 0, 0, 0, 0) \).
ii. If \( i \neq 0 \) (then can be denoted by \( i^* \)), from (2a) we can get \( \pi - (\pi - \alpha a^* + (1 - \varepsilon)\theta i^*)s^* = \lambda s^* \) and then

\[ \lambda s^* = (\pi + \delta - \alpha a^* + (1 - \varepsilon)\theta i^*)i^* - (1 - \varepsilon)\theta i^* \] was obtained by (2b). With substitute that equations, then we can get \( s^* = \pi + (1 - \varepsilon)\theta i^* - (\pi + \delta - \alpha a^* + (1 - \varepsilon)\theta i^*)i^* \). After that, from (2c) and (2d) can be obtained

\[ p^* = \frac{\sigma_i \delta i^*}{(\pi + \gamma - \alpha a^* + (1 - \varepsilon)\theta i^*)} \quad \text{and} \quad h^* = \frac{l}{\psi \phi} \] with \( l = \sigma_i \delta i^* \phi + m \gamma \sigma_i \delta i^* + va \phi \), \( \psi = (\pi + k - \alpha a^* + (1 - \varepsilon)\theta i^*) \), and \( \phi = (\pi + \gamma - \alpha a^* + (1 - \varepsilon)\theta i^*) \). Then, \( a^* = \frac{1}{\omega \phi \psi} \left[ (1 - \sigma_i - \sigma_j) \delta i^* \phi \psi + (1 - m) \gamma \sigma_i \delta i^* \psi \right] \) was obtained by (2e) equation. So, it is proved that the system (2) had an endemic equilibrium point \( E_1 = (s^*, i^*, p^*, h^*, a^*) \).

1.3. Basic Reproduction Number

The basic reproduction number was determine during next generation matrix. In this model, the infected class is \( i, p, h, a \) class, so the differential equations are used:

\[
\begin{align*}
\frac{di}{dt} &= \lambda s + (1 - \varepsilon)\theta i - (\pi + \delta - \alpha a + (1 - \varepsilon)\theta i)i \\
\frac{dp}{dt} &= \sigma_i \delta i - (\pi + \gamma - \alpha a + (1 - \varepsilon)\theta i) p \\
\frac{dh}{dt} &= \sigma_i \delta i + m\gamma p + va - (\pi + k - \alpha a + (1 - \varepsilon)\theta i)h \\
\frac{da}{dt} &= (1 - \sigma_i - \sigma_j) \delta i + (1 - m)\gamma p + kh - \left( \pi + v + \alpha - \alpha a + (1 - \varepsilon)\theta i \right)a 
\end{align*}
\]

with \( \lambda = c_1 \beta_i + c_2 \beta_j h \), so that

\[
\begin{bmatrix} c_1 \beta_i s + c_2 \beta_j h s + (1 - \varepsilon)\theta i \\ 0 \\ 0 \\ 0 \end{bmatrix}
\]

and \( v = \begin{bmatrix} (\pi + \delta - \alpha a + (1 - \varepsilon)\theta i)i \\ -\sigma_i \delta i + (\pi + \gamma - \alpha a + (1 - \varepsilon)\theta i) p \\ -\sigma_i \delta i - m\gamma p + va + (\pi + k - \alpha a + (1 - \varepsilon)\theta i)h \\ -(1 - \sigma_i - \sigma_j) \delta i + (1 - m)\gamma p + kh + \left( \pi + v + \alpha - \alpha a + (1 - \varepsilon)\theta i \right)a \end{bmatrix} \).

Then \( f \) and \( v \) were linearized and obtained

\[
F = \begin{bmatrix} c_1 \beta_i + (1 - \varepsilon)\theta & 0 & c_2 \beta_j & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \pi + \delta & 0 & 0 & 0 \\ -\sigma_i \delta & \pi + \gamma & 0 & 0 \\ -\sigma_j \delta & -m\gamma & \pi + k & -v \\ -(1 - \sigma_i - \sigma_j) \delta & (m - 1)\gamma & -k & \pi + v + \alpha \end{bmatrix} .
\]

After finding \( V^{-1} \) and then can be obtained matrix \( K \) by multiplication matrix \( FV^{-1} \). Basic reproduction number was obtained from the largest Eigen value so \( f \) matrix \( K \). Thus, the basic reproduction number is

\[
R_0 = \frac{c_1 \beta_i + (1 - \varepsilon)\theta}{\pi + \delta} + \frac{c_2 \beta_j \delta \left( (\pi \gamma + m\gamma a - v\gamma) + \sigma_i (\pi \gamma + m\gamma a + \gamma a) + \pi \gamma \right)}{(\pi + \delta)(\pi + \gamma)(\pi + v + \alpha)(\pi + k) - kv} .
\]
1.4. Stability of Equilibrium Point

Theorem 2.
If \( R_0 < 1 \), then the disease-free equilibrium point \( E_0 = (s, i, p, h, a) = (1, 0, 0, 0, 0) \) was local asymptotically stable for each \( \tau \geq 0 \).

Based on Routh Hurwitz criteria, we get zero makers from characteristic equation of Jacobian disease-free equilibrium \( J(E_0) \) are negative. So, the disease-free equilibrium point \( E_0 = (s, i, p, h, a) = (1, 0, 0, 0, 0) \) was local asymptotically stable for each \( \tau \geq 0 \).

Furthermore, based in [9] and [10], the endemic equilibrium point \( E_1 = (s^*, i^*, p^*, h^*, a^*) \) was local asymptotically stable if it fulfilled Theorem 3.

Theorem 3.
The endemic equilibrium point \( E_1 = (s^*, i^*, p^*, h^*, a^*) \) was local asymptotically stable for each \( \tau \geq 0 \) if qualify necessary and sufficient conditions as follows,
- Real part of each roots from \( \Delta(g, \tau) \) are negative.
- For each real \( \sigma > 0 \), \( \Delta(i\sigma, \tau) \neq 0 \) where \( i = \sqrt{-1} \).

The stability of the equilibrium point system (2) with the time delay \( \tau \geq 0 \) can be determined by determining the characteristic of
\[
\text{det}(J_0 - J e^{-\tau g} - gI) = 0
\]
with \( J_0 \) is the Jacobian matrix for parameters without delay and \( J \) is the Jacobian matrix for the delay parameter. So can be obtained,
\[
N(g) + Q(g) e^{-\tau g} = 0
\]
by \( N(g) = g^5 + N_1 g^4 + N_2 g^3 + N_3 g^2 + N_4 g + N_5 \) and \( Q(g) = Q_1 g^4 + Q_2 g^3 + Q_3 g^2 + Q_4 g + Q_5 \).

Furthermore, it will be indicated that no Eigen values of the linear system at the point had real part zero (complex Eigen values). Suppose the characteristic of linear system at \( E_1 \) has complex Eigen values \( g = i\sigma \) with \( \sigma > 0 \), it can be obtained
\[
\left(\sigma^5 + N_1 \sigma^4 - N_1 \sigma^2 + N_4 \sigma i + N_5\right) + \left(Q_1 \sigma^4 - Q_2 \sigma^3 i - Q_3 \sigma^2 + Q_4 \sigma i + Q_5\right) e^{i\sigma \tau} = 0
\]
\[
\Leftrightarrow \left(\sigma^5 + N_1 \sigma^4 - N_1 \sigma^2 + N_4 \sigma i + N_5\right) + \left(Q_1 \sigma^4 - Q_2 \sigma^3 i - Q_3 \sigma^2 + Q_4 \sigma i + Q_5\right) e^{i\sigma \tau} = 0 \quad (3)
\]
Then, separation between the real and imaginary parts in equation(3), the equations are squared and summed so that,
\[
\sigma^{10} + R_1 \sigma^8 + R_2 \sigma^6 + R_3 \sigma^4 + R_4 \sigma^2 + R_5 = 0
\]
\[
\Leftrightarrow \bar{u}^{10} + R_1 \bar{u}^8 + R_2 \bar{u}^6 + R_3 \bar{u}^4 + R_4 \bar{u}^2 + R_5 = 0 \quad (4)
\]
with
\[
\bar{u} = \sigma^2; \quad R_1 = N_1^2 - 2N_2 + Q_1^2; \quad R_2 = N_1^2 - 2N_2N_4 + 2N_4 + 2Q_1Q_2 - Q_3^2; \quad R_3 = 2N_1N_3 + N_1^2 - 2N_2N_4 - Q_2^2 - 2Q_1Q_3 + 2Q_2Q_4; \quad R_4 = N_1^2 - 2N_2N_4 + 2Q_1Q_2 - Q_3^2; \quad R_5 = A^2 - Q_5^2.
\]
Based on the Routh Hurwitz criteria, zero-makers from the equation (4) is negative if there are no change at the sign of the first column in the Routh table. Thus, the endemic equilibrium point \( E_1 \) is local asymptotically stable.
Theorem 4.
If $\Delta(\delta, \tau)$ has a positive real roots $\sigma_0$, so that can be obtained the critical value of delay $\tau^*$, then the endemic equilibrium $E_1 = (s^*, i^*, p^*, h^*, a^*)$ is local asymptotically stable for $\tau < \tau^*$, unstable for $\tau > \tau^*$ and the bifurcation will occur for $\tau = \tau^*$.

When there was a value of the first column of The Routh table changing the sign, there exists a zero maker in equation $(4)$ had positive value as a sign of changes that occurred in the first column of the table and equation $(4)$ had imaginary roots. Thus the delay can be found below the critical value,

$$\tau^* = \frac{1}{\sigma_0} \arccos \left( \frac{(N_i \sigma_0^4 - N_i \sigma_0^2 - N_i \sigma_0^4 - N_i \sigma_0^2)(Q_j \sigma_0^4 - Q_j \sigma_0^2 - Q_j \sigma_0^4 - Q_j \sigma_0^2)}{(Q_j \sigma_0^4 - Q_j \sigma_0^2 + Q_j)^2 - (Q_j \sigma_0^4 - Q_j \sigma_0^2 + Q_j)^2} \right) + \frac{2n\pi}{\sigma_0}$$

with $n = 0, 1, 2, 3, ...$

3. Numerical simulation of the model
Model simulations carried out using software Maple and Matlab. The data was obtained based on the number of AIDS patients in Daerah Istimewa Yogyakarta (DIY). In year 2012, there found 941 cases of HIV-AIDS \cite{11} (Dinkes DIY, 2013). According to the Badan Komisi Penanggulangan AIDS Yogyakarta, the most cases are found in the city of Yogyakarta with 458 HIV and 219 AIDS. The total population of the city of Yogyakarta at that time is 427,592. Based on these problems, we could obtain the starting value for the proportion of $S(0) = 0.98143; I(0) = 0.0011; P(0) = 0.00022; T(0) = 0.000017, A(0) = 0.00052$.

With the parameters $\theta = 0.2; \alpha = 1; \epsilon = 0.2; \nu = 0.1; \beta_1 = 0.4; \beta_2 = 0.05; \sigma_1 = 0.2; \sigma_2 = 0.01; \gamma = 0.9; \pi = 0.9; k = 0.08; c_1 = 3; c_2 = 1; m = 0.4; \delta = 0.6$; are obtained for the simulation shown in Figure 2.(a) and 2.(b) as follows,

![Figure 2.(a)](image1)

![Figure 2.(b)](image2)

Figure 2.(a). Simulation system (2) $R_0 < 1, \tau = 0$

Figure 2.(b). Simulation system (2) $R_0 < 1, \tau \neq 0$

We get $R_0 = 0.9085621585 < 1$ from those parameters. Figure 2.(a) shows the behavior of the solution toward the free equilibrium point $E_0$. Figure 2.(b) with an arbitrary value of $\tau$ and $\tau \neq 0$ shows the same behaviour as in Figure 2.(a) with $\tau = 0$. This shows that the time delay did not effect on the stability of the free equilibrium point so that any given time delay will always provide a solution toward to the free equilibrium point. The behavior of solutions toward to the free equilibrium point at $R_0 < 1$. It means that the AIDS epidemic will disappear from the population.

For $R_0 > 1$ and $\tau = 0$ with parameters $\epsilon = 0.2; \nu = 0.1; \beta_1 = 0.4; \beta_2 = 0.05; \sigma_1 = 0.2; \sigma_2 = 0.01; k = 0.08; c_1 = 3;
$c_2 = 1, m = 0.4, \delta = 0.6, \alpha = 1, \gamma = 0.9, \pi = 0.1$; and $\theta$ values that were not fixed, are shown in the following figure,

**Figure 3.(a).** Simulation system (2), $R_0 = 1.98163; \theta = 0.2$

**Figure 3.(b).** Simulation system (2), $R_0 = 2.2102; \theta = 0.4$

**Figure 3.(c).** Simulation system (2), $R_0 = 2.66735; \theta = 0.8$

Based on Figure 3.(a), Figure 3.(b), and Figure 3.(c) show that the proportion of S decreased while the proportion of I increase continually due to changes the value of the birth rate of individuals infected by HIV because of a vertical transmission ($\theta$). The proportion increasing of I population influence the proportion increasing of A population. It means the birth of individuals infected by HIV affects many as proportion of AIDS positive population. In addition, the growing value of the parameter $\theta$ also indicated that the solution of the system (2) toward to the endemic equilibrium point is equals to the enlarged of the basic reproduction number. It means that if the birth rate of HIV-infected individuals because of vertical transmission was growing, the rate of AIDS epidemic would be even greater. For $R_0 > 1$ and $\tau \neq 0$ are shown in figures below,

**Figure 4.(a).** $\tau = 0$

**Figure 4.(b).** $\tau = 1$
From the simulation of $R_0 = 1.98163; \theta = 0.2$ with time delay, the value of delay ($\tau$) was in between $0 < \tau \leq 1.98$ for these parameters. The existence of time delay in the model of AIDS epidemic did not affect the stability of the endemic equilibrium point, but it only affects the behaviour of solutions in towards the endemic equilibrium point. The behaviour of the solution is shown in Figure 4.(a), (b), (c), and (d) that towards to the endemic equilibrium point after about $t = 25, t = 30, t = 50, \text{ and } t = 100$. This shows the effect of time delay only make the behaviour of the solutions slow towards to the endemic equilibrium point. In other words, the time delay only make slow down the AIDS positive symptoms from individuals infected HIV to be suffer positively AIDS.

4. Conclusions
Based on these discussions, the model form of AIDS epidemic model through treatment and vertical transmission with time delay is non-linear differential equation with time delay. Based on the model analysis gives the result of the disease free equilibrium point and the endemic equilibrium point.

Basic reproduction number as an indicator epidemic of AIDS shows if $R_0 < 1$, AIDS does not attack the population, whereas if $R_0 > 1$ AIDS. On the $R_0 < 1$ and $\tau \geq 0$ condition, the disease-free equilibrium point local asymptotically stable. In the other hand, the endemic equilibrium point will be local asymptotically stable if the time delay is less than the critical value of delay, unstable if the time delay is more than the critical value of delay, and bifurcation occurs if the time delay is equal to the critical value of delay.

5. References

