Modelling Dengue Transmission Dynamics using a Two-Time Delay Differential Equation

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Abstract
This study proposes an autonomous dynamical system to model the transmission dynamics of dengue epidemic, using a two – time delay differential equation. The delay due to extrinsic and intrinsic incubation period for the host (humans) and vector (mosquitoes) was considered. The existence of the equilibrium points was investigated and its stability studied analytically. The endemic equilibrium was found to be stable if the delay is under certain conditions. Numerical simulations were performed to obtain numerical results for the basic reproduction number with both time delays for a range of parameter values, to illustrate the theoretical results.

Keywords: *Dengue transmission, Delay differential equation, Basic reproduction number, Stability*

1. Introduction

Dengue is a vector –borne disease caused by four stereotypes of the dengue virus (DEN-1, DEN-2, DEN-3, DEN-4). Dengue haemorrhagic fever (DHF) is a major complication of the dengue disease. The principal vector of dengue virus is the Aedes aegypti mosquito (Massawe & Makinde, 2015). This mosquito prefers taking blood-meals from humans and typically bites during the day. The virus is usually transmitted during this process from an infectious mosquito to a susceptible human or from an infectious human to a susceptible mosquito. Recovery from infection by one virus provides lifelong immunity against that virus but confers only partial and transient protection against subsequent infection by the other three viruses (Wearing, 2006), unfortunately, there is no specific effective treatment for Dengue.

Ordinary differential equation (ODE) models of vector-borne diseases like dengue have a long history and, in most cases, studies focus mainly on vector-control (Kochar, 2009, Mendis, 2001, Uwakwe, Emerenini & Inyama, 2020). These studies range from field and laboratory research to mathematical models. Although, these models are capable of predicting long term dynamics, more than one hundred years later, vector-borne diseases such like dengue continue to pose a significant burden worldwide (Gubler,1998). The development of mosquito resistance to insecticides, changes in public health programs, climate change, changes in agricultural practices, the increased mobility of humans, and urban growth are all factors that contribute to the difficulty in controlling and eliminating dengue (Siriyasatien, Phuwee, Ongruk &Jampachaisri, 2016). In a study by Chanprasopchai, Tang & Pongsumpun (2017), the SEIR model was used to describe the transmission of dengue virus, considering the role of rainfall in Thailand. The impact of climate on transmission of dengue virus was also considered by Polwiang (2015). The value of the seasonal reproduction number was calculated to evaluate the potential, severity and persistence of dengue with results showing

that a very high or regular temperature reduces the risk of the dengue infection.
Delays occur naturally in dengue just like in every other vector borne disease (Sakdanupaph& Elvin, 2009). This is because steps in the development of vectors like mosquito take a significant amount of time, particularly compared to the lifespan of the mosquito. This makes delay differential equations a natural choice for modelling vector-borne diseases like dengue. Haito, Dan & Chunhua (2020), carried out a work on delay differential equation of dengue. In their study they formulated a dengue virus transmission model with maturation delay for mosquito production and seasonality. The result revealed that the temperature change causes the periodic oscillations of dengue fever cases. The roles of maturation delay vaccination. Others include works by Zheng, Chen & Sun (2019), Nie & Xue (2017) and Kesorn (2015).
In this work a two-time delay differential equation which incorporates the delays related to the extrinsic and intrinsic incubation periods are considered in modelling the dengue disease.

2. Model formulation

2.1.The ODE model

The transmission involves the vectors which is mosquito and the host (humans). The susceptible mosquitoes are denoted by S_m and the infected mosquitoes I_m. A susceptible mosquito becomes infected upon biting an infected human I_h with a biting rate $\boldsymbol{a}$ and probability of transmission of the disease given by $\boldsymbol{p}$. the susceptible humans S_h becomes infected when bitten by an infectious mosquito. We assume the infected mosquitoes bite at the same rate as susceptible mosquitoes **a**, with the probability of transmission $\boldsymbol{q}$. The model takes the form;

$$\begin{aligned} S_m^I &= \Lambda_m - PS_mI_h - \alpha S_m \\ I_m^I &= PaS_mI_h - a\alpha I_m \\ S_h^I &= \Lambda_h - qaS_hI_m - \mu S_h \\ I_h^I &= qaS_hI_m - (\mu + c + d)I_h \\ R_h^I &= cI_h - \mu R_h \end{aligned} \qquad (2.1)$$

Here Λ_m and Λ_h are the recruitment rates of the mosquitoes and humans respectively. $\boldsymbol{\alpha}$ and $\boldsymbol{\mu}$ are the respective death rates for the mosquitoes and humans, while d is the disease induced death rates of humans and $\boldsymbol{c}$ is the recovery rate of humans. The total population size of the mosquitoes and humans is given by the equations.

$$N_m = S_m + I_m \text{ and } N_h = S_h + I_h + R_h$$

Reducing the system by replacing the susceptible humans and mosquitoes with

$$S_m = N_m - I_m \quad \text{and} \quad S_h = N_h - I_h - R_h$$

The system of infectious mosquitoes and human becomes;

$$\begin{aligned} I_m^I &= Pa(N_m - I_m)I_h - \alpha I_m \\ I_h^I &= qa(N_h - I_h - R_h)I_m - (\mu + c + d)I_h \end{aligned} \qquad (2.2)$$

2.2 The delay equation

We consider two delays; delays related to the extrinsic and intrinsic incubation periods. When the pathogen enters the body of the vector, the time which elapses before the vector becomes infectious is called the extrinsic incubation period. The incubation period of the pathogen within the host is the intrinsic incubation period. Incorporating the delays the model takes the form;

$$\begin{aligned} S_m^I &= \Lambda_m - Pae^{-\alpha\tau_1}S_mI_h(t-\tau_1) - \alpha S_m \\ I_m^I &= Pae^{-\alpha\tau_1}S_mI_h(t-\tau_1) - \alpha I_m \\ S_h^I &= \Lambda_h - qae^{-\alpha\tau_2}S_hI_m(t-\tau_2) - \mu S_h \\ I_h^I &= qae^{-\alpha\tau_2}S_hI_m(t-\tau_2) - (\mu + c + d)I_h \end{aligned} \qquad (2.3)$$

$$R_h^I = cI_h - \mu R_h$$

Where τ_1 *and* τ_2 *are* the delays induced by the extrinsic and intrinsic incubation periods respectively. A mosquito or human that becomes infectious at time *t* was infected at time t - τ_1 and t - τ_2 respectively, $e^{-\alpha\tau_1}$ and $e^{-\alpha\tau_2}$ are the probabilities of survival of the mosquitoes and human when infectious.

Modifying equation (2.3) using the limiting system as in (2.2), we have

$$I_m^I = Pae^{-\alpha\tau_1}(N_m - I_m(t-\tau_1))I_h(t-\tau_1) - \alpha I_m \qquad (2.4)$$
$$I_h^I = qae^{-\alpha\tau_2}(N_h - I_h(t-\tau_2))I_m(t-\tau_2) - (\mu + c + d)I_h$$

3. Model Analysis

Using the next generation matrix method, we obtain the basic reproduction number

$$R_0 = \frac{Pqa^2N_mN_he^{-\alpha\tau_1}e^{-c\tau_2}}{\alpha(\mu + c + d)}$$

At equilibrium we have $I_m^I = I_h^I = 0$

If we assume that the incubation periods are approximately equal that is,

$\tau_1 = \tau_2 = \tau$, the model with two delays becomes;

$$I_m^I = Pae^{-\alpha\tau}(N_m - I_m(t-\tau))I_h(t-\tau) - \alpha I_m \qquad (*) \qquad (3.1)$$
$$I_h^I = qae^{-\alpha\tau}(N_h - I_h(t-\tau))I_m(t-\tau) - (\mu + c + d)I_h \qquad (**)$$

From (*) and (**) in (3.1)

$$I_m^* = \frac{Pae^{-\alpha\tau}N_mI_h^*(t-\tau)}{Pae^{-\alpha\tau}I_h^*(t-\tau)+\alpha}$$
$$I_h^* = \frac{Pqa^2e^{-\alpha\tau_1}e^{-c\tau_2}N_m(N_h-(t-\tau)-R_h)-\alpha(\mu+c+d)}{Pae^{-\alpha\tau}(t-\tau)(N_m(t-\tau)+(\mu+c+d))}$$

3.1.Stability of equilibrium

The Disease –free equilibrium (DFE) is gotten when D = (I_m, I_h) = (0, 0). Hence, the Jacobian of (2.4) at the DFE is given by;

$$J(D) = \begin{pmatrix} -\alpha & N_mPae^{-\alpha\tau_1} \\ N_hqae^{-\alpha\tau_2} & -(\mu + c + d) \end{pmatrix}$$

Calculating the eigenvalues of the Jacobian at the DFE;

$\lambda^2 + (\alpha + k_3)\lambda + \alpha k_3(1 - R_0) = 0$

Where $k_3 = (\mu + c + d)$

By Routh - Horwith Criterion

$a_0 = 1 > 0$, $a_1 = (\alpha + k_3) > 0$, $a_3 = \alpha k_3(1 - R_0) > 0$ *if and only if* $R_0 < 1$

The endemic equilibrium (EE) is given by E = (I_m^*, I_h^*)

Linearizing system (3.1) at the EE we have,

$$J(E) = \begin{pmatrix} -k_1I_h^*(t-\tau) - \alpha & k_1(t-\tau)(N_m - I_m^*(t-\tau)) \\ k_2(N_h - I_h^*(t-\tau) - R_h) & -k_2I_m^*(t-\tau) - k_3 \end{pmatrix}$$

Calculating the eigenvalues of the Jacobian at the EE;

$\lambda^2 + (k_1z_1^2 + \alpha + k_2z_2 + k_3)\lambda + \alpha k_3(t-\tau)(1 - R_0) + k_1k_2[(z_1 + R_h) + z_2(N_h + R_h)]] + k_1k_3z_1^2 + \alpha k_2z_2 = 0$ (3.2)

By Routh- Horwith Criterion, from (3.2),

$a_0 > 0$, $a_1 > 0$ while $a_3 > 0$ if the following is established

(i)$R_0 < 1$ and $(t - \tau) > 0$

(ii) $R_0 > 1$ and $(t - \tau) < 0$

When $R_0 < 1$ the DFE will coexist with a stable and an unstable endemic equilibrium. This is basic evidence of the existence of the backward bifurcation phenomena. Hence, the delays

(τ), (incubation periods) play a major role in the transmission of the disease.

4. Numerical Simulation

In this section, we illustrate the analytical results of the study by carrying out numerical simulations of the model with respective to the basic reproduction number (R_0). Parameter values are obtained from different literatures, some are estimated to vary within realistic means and given as; p = 0.1, q = 0.1, a = 0.2, N_m = 0.02, N_h = 0.01, c = 0.4, d = 0.3, τ = 0.5, α = 0.14 and μ = 0.4.

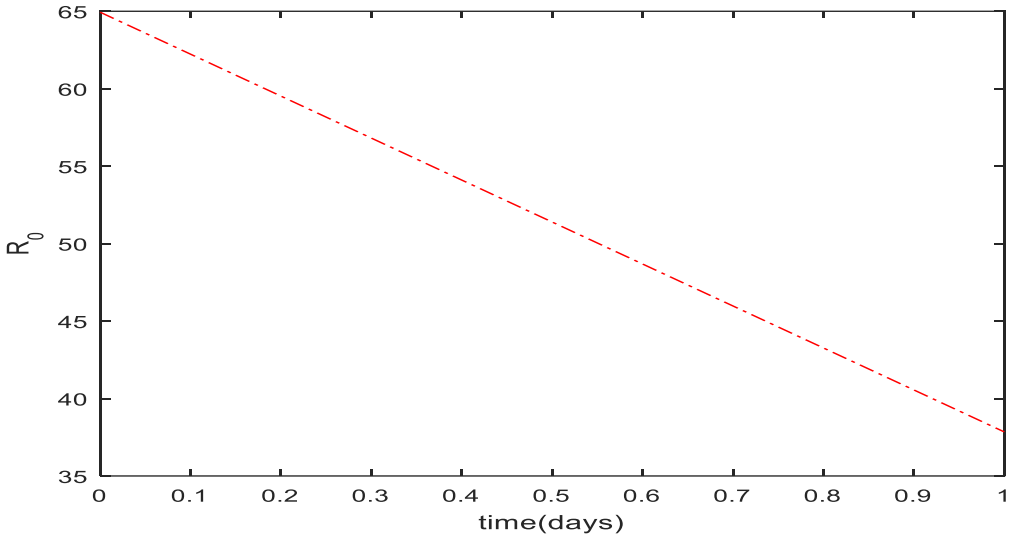

Figure 1. *Simulations of model system showing the average number of infected humans in days with both delays.*

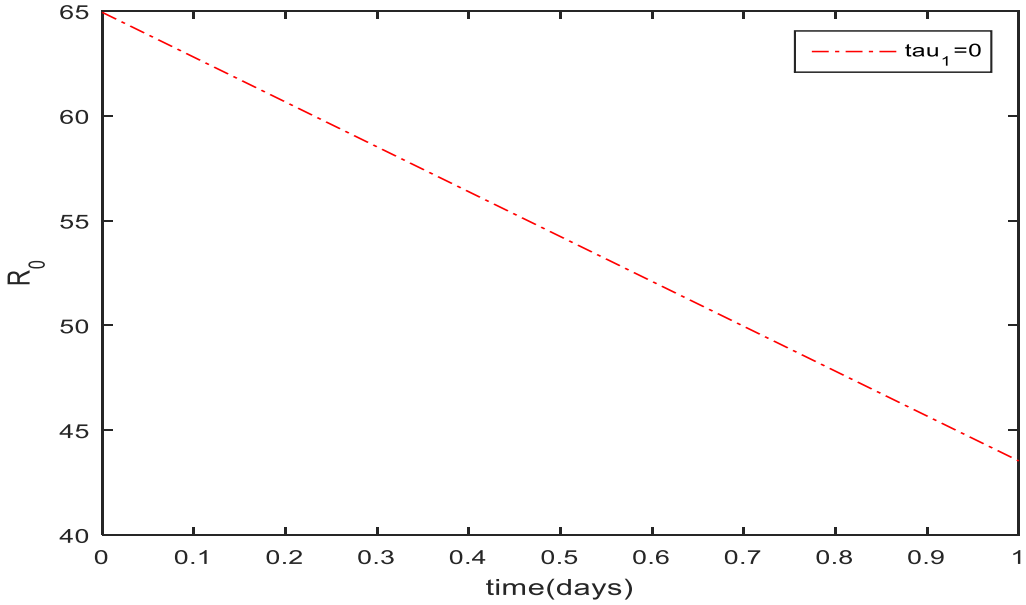

Figure 2. *Simulations of model system showing the average number of infected humans in days without the extrinsic delay*

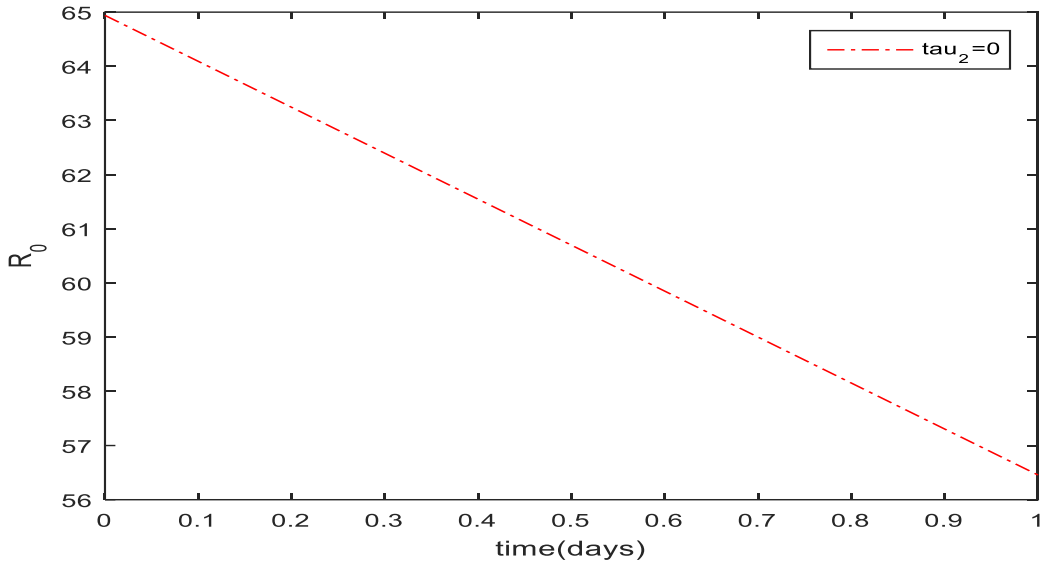

Figure 3. *Simulations of model system showing the average number of infected humans in days without the intrinsic delay*

The figures show the effect of the delays on R_0, the average number of infected humans. From figure 1, the average number of infected humans reduces to 37 in 10days, when both the extrinsic and intrinsic delays are incorporated.
From the figures 2 and 3, it can be observed that both the extrinsic and intrinsic delays affect the number of infected humans as the days goes by. In 10 days, the basic reproduction number (R_0) is below 57 with only the intrinsic delay while with only the extrinsic delay R_0 is below 45. This implies that the extrinsic delay which is the time which elapses before the vector becomes infectious causes a reduction in the number of infections, hence, should be put into consideration when introducing intervention measures,

5. Conclusion

Results from the stability analysis shows that changes in the incubation periods may alter the dynamics of the disease, causing a stable transmission region to become unstable, or vice versa. From the numerical simulation, when only the extrinsic delay is considered, the average number of infected humans at 10days is lower than when only the intrinsic delay is considered. It becomes even lower, when both delays are considered. Consequently, understanding if and when these transitions are likely to occur may be very important in determining the effects of intervention strategies or climate change that prolong incubation periods.

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