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Analysis of a dengue disease transmission model with vaccination

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ABSTRACT

Soewono and Supriatna [9] studied a simple SIR dengue disease transmission model with vaccination. In the present paper we have modified the model with assumption that a random fraction of the recovered host population can loses the immunity and becomes susceptible again. The dynamics of the disease is studied by a compartmental model involving ordinary differential equations for the human and the mosquito populations. Restricting the dynamics for the constant host and vector populations, the model is reduced to a three-dimensional planar equation. Two states of equilibrium are studied, one disease-free and other endemic. The basic reproduction number $\Re_0$ *is obtained. In this model the disease-free equilibrium state is stable if* $\Re_0 \leq 1$ *and if* $\Re_0 > 1$*, the stable endemic equilibrium appears. Numerical simulation and graphical presentation are also provided to justify the stability.*

Keywords: Epidemiology, Dengue disease, Vector-host model, Stability, Reproduction number, Vaccination.
AMS Classification: *92D30.*

INTRODUCTION

Dengue fever is high on the list of mosquito-borne diseases that may worsen with global warming. It is a globally reemerging viral disease transmitted to humans by the bite of an infected Aedes Aegypti mosquitoe and exists in two forms: the Dengue Fever (DF) and the Dengue Haemorrhagic Fever (DHF). The symptoms of the disease include high fever, rash, and severe headache with aching bones, joints, and muscles. Dengue and its deadly complications, dengue hemorrhagic fever and dengue shock syndrome, have increased over the past several decades. Global warming could substantially increase the number of people at risk of dengue epidemics, as warmer temperatures and changing rainfall conditions expand both the area suitable for the mosquito vectors and the length of the dengue transmission season in temperate areas.

Dengue fever is caused by a member of the same family of viruses that cause yellow fever, West Nile and Japanese encephalitis. It is possible to become infected by dengue multiple times because the virus has four different serotypes known as DEN1, DEN2, DEN3 and DEN4. A person infected by one of the four serotypes will never be infected again by the same serotype, but he loses immunity to the other three serotypes in about 12 weeks and then becomes more susceptible to developing dengue haemorrhagic fever. The strategies of mosquito control by insecticides or similar techniques proved to be inefficient.

A standard program used in many countries to control the spread of the disease is the control of the main disease vector by fuming or fogging. Many studies show that this program was not fully effective. A simple SIR model for dengue disease transmission has been studied by many researchers [2, 3, 4, 6, 12]. Now a day's researchers are going on towards the invention of vaccine for dengue disease. The effects of vaccination on the transmission of infectious disease are studied by some of the researchers [1, 9, 10].

In the recent communication Soewono and Supriatna [9] considered two types of vaccination in a host transmission model for dengue fever. In the model considered by them it has been assumed that the vaccine prevents vaccinated people by all types of dengue viruses but it is not perfect. Even after vaccination the host may suffer from the disease with certain probability. In the present paper we have modified the model of Soewono and Supriatna [9] with assumption that a random fraction of the recovered host population can loses the immunity and becomes susceptible again.

2. Formulation of the Model for Dengue Disease Transmission

Let H and V be the host and vector population sizes, respectively. It is assumed that the host and vector population has constant size with birth and death rate equal to μ_H and μ_V. The host population is subdivided into the susceptible S_H, the infective I_H and the recovered (immune) R_H. The vector population, due to a short life period, is subdivided into the susceptible S_v and the infective I_v. We consider here two types of vaccination in a host-vector model for the dengue disease transmission. One is being administered to a portion of new born host and another one is being administered to a portion of susceptible host.

Let a portion ρ, $0 \le \rho \le 1$, of newborn host be vaccinated. Assume that the vaccine is not perfect and let the effectiveness of the vaccine is s, then $(1-\rho s)\mu_H H$ newborns remain susceptible, and $\rho s \mu_H H$ directly being removed to R_H. On the other hand when a portion $\sigma, 0 \le \sigma \le 1$, of susceptible S_H are vaccinated, then the dynamics of both S_H and I_H are affected. Another assumption for this model is that a random fraction of the recovered host population can loses the immunity and becomes susceptible again. The interaction model is governed by the following mathematical equations.

For human population the equations are

$$\begin{aligned}
\frac{dS_H}{dt} &= \mu_H (1-\rho s) H - \frac{b p_H I_v S_H (1-\sigma s)}{H} - \mu_H S_H + \phi_H R_H \\
\frac{dI_H}{dt} &= \frac{b p_H I_v S_H (1-\sigma s)}{H} - \mu_H I_H - \gamma I_H \\
\frac{dR_H}{dt} &= \mu_H \rho s H + \gamma I_H - \mu_H R_H - \phi_H R_H
\end{aligned} \tag{2.1}$$

and for vector population

$$\begin{aligned}
\frac{dS_v}{dt} &= \mu_v V - \frac{b p_v I_H S_V}{H} - \mu_v S_v \\
\frac{dI_v}{dt} &= \frac{b p_v I_H S_V}{H} - \mu_v I_v
\end{aligned} \tag{2.2}$$

where b is the biting rate of the vector, p_H is the transmission rate from infected vector to susceptible host, p_v is the transmission rate from infected host to susceptible vector, γ is the recovery rate of the host population, Φ_H is the loss of immunity rate in the host population. Using $S_H + I_H + R_H = H$ and $S_v + I_v = V$, the systems (2.1) and (2.2) become

$$\begin{aligned}
\frac{dS_H}{dt} &= \mu_H (1-\rho s) H - \frac{b p_H I_v S_H (1-\sigma s)}{H} - \mu_H S_H + \phi_H (H - S_H - I_H) \\
\frac{dI_H}{dt} &= \frac{b p_H I_v S_H (1-\sigma s)}{H} - (\mu_H + \gamma) I_H \\
\frac{dI_v}{dt} &= \frac{b p_v I_H (V - I_v)}{H} - \mu_v I_v
\end{aligned} \tag{2.3}$$

Writing the dynamics (2.3) in population proportion

$S_h = \frac{S_H}{H}$, $I_h = \frac{I_H}{H}$ and $I_v = \frac{I_v}{V}$, we have

$$\frac{dS_h}{dt} = \mu_H(1-\rho s) - bp_H \nu I_v S_h(1-\sigma s) - \mu_H S_h + \phi_H - \phi_H(S_h + I_h)$$

$$\frac{dI_h}{dt} = bp_H \nu I_v S_h(1-\sigma s) - (\mu_H + \gamma) I_h \tag{2.4}$$

$$\frac{dI_v}{dt} = bp_v I_h(1-I_v) - \mu_v I_v$$

where $\nu = \frac{V}{H}$ is the ratio of host and vector population.

Setting $S_h = x$, $I_h = y$, $I_v = z$, and rescaling t by bp_v; these equations can be simplified to

$$\frac{dx}{dt} = \mu(1-r) - \alpha x - \eta xz + \phi - \phi y$$

$$\frac{dI_h}{dt} = \eta xz - \beta y \tag{2.5}$$

$$\frac{dI_v}{dt} = y(1-z) - \delta z$$

where $\mu = \frac{\mu_H}{bp_v}$, $r = \rho s$, $\phi = \frac{\phi_H}{bp_v}$, $\alpha = \frac{\mu_H + \phi_H}{bp_v}$, $\eta = \frac{p_H \nu(1-\sigma s)}{p_v}$, $\beta = \frac{\mu_H + \gamma}{bp_v}$ and $\delta = \frac{\mu_v}{bp_v}$.

3. Stability Analysis of the Equilibrium Points

Equilibrium points of system (2.5) are obtained by setting time derivatives of x, y, z to zero. The system of equations (2.3) possess two equilibrium points; one is disease-free equilibrium $E_1 = \left(\frac{\mu(1-r)+\phi}{\alpha}, 0, 0\right)$ and other is endemic equilibrium $E_2 = (x_e, y_e, z_e)$ where

$$x_e = \frac{\beta[\mu(1-r)+\phi+(\beta+\phi)\delta]}{\eta(\beta+\phi)+\alpha\beta} = \frac{(\mu_H+\gamma)[bp_v\{\mu_H(1-\rho s)+\phi_H\}+(\mu_H+\gamma+\phi_H)\mu_v]}{bp_v[bp_H\nu(1-\sigma s)(\mu_H+\gamma+\phi_H)+(\mu_H+\phi_H)(\mu_H+\gamma)]},$$

$$y_e = \frac{\eta[\mu(1-r)+\phi]-\alpha\beta\delta}{\eta(\beta+\phi)+\alpha\beta} = \frac{b^2p_Hp_v\nu(1-\sigma s)[\mu_H(1-\rho s)+\phi_H]-(\mu_H+\phi_H)(\mu_H+\gamma)\mu_v}{bp_v[bp_H\nu(1-\sigma s)(\mu_H+\gamma+\phi_H)+(\mu_H+\phi_H)(\mu_H+\gamma)]},$$

$$z_e = \frac{\eta[\mu(1-r)+\phi]-\alpha\beta\delta}{\eta[\mu(1-r)+\phi+(\beta+\phi)\delta]} = \frac{b^2p_Hp_v\nu(1-\sigma s)[\mu_H(1-\rho s)+\phi_H]-(\mu_H+\phi_H)(\mu_H+\gamma)\mu_v}{bp_H\nu(1-\sigma s)[bp_v\{\mu_H(1-\rho s)+\phi_H\}+(\mu_H+\gamma+\phi_H)\mu_v]}.$$

This provides a reproduction number $\Re_0 = \frac{b^2p_Hp_v\nu(1-\sigma s)[\mu_H(1-\rho s)+\phi_H]}{(\mu_H+\phi_H)(\mu_H+\gamma)\mu_v}$.

The endemic equilibrium E_2 is stable when $\eta[\mu(1-r)+\phi]-\alpha\beta\delta > 0$

i.e. $\Re_0 = \frac{b^2p_Hp_v\nu(1-\sigma s)[\mu_H(1-\rho s)+\phi_H]}{(\mu_H+\phi_H)(\mu_H+\gamma)\mu_v} > 1.$

Now we shall discuss the local stability of the equilibrium points.

The variation matrix of the system (2.5) is given by

$$J=\begin{pmatrix} -\alpha-\eta z & -\phi & -\eta x \\ \eta z & -\beta & \eta x \\ 0 & 1-z & -y-\delta \end{pmatrix}.$$

For the disease-free equilibrium point $E_1=\left(\frac{\mu(1-r)+\phi}{\alpha},0,0\right)$, the variation matrix will be

$$J(E_1)=\begin{pmatrix} -\alpha & -\phi & -\frac{\eta\left[\mu(1-r)+\phi\right]}{\alpha} \\ 0 & -\beta & \frac{\eta\left[\mu(1-r)+\phi\right]}{\alpha} \\ 0 & 1 & -\delta \end{pmatrix}.$$

Its characteristic equation will be

$$(\lambda+\alpha)\left[\lambda^2+(\beta+\delta)\lambda+\beta\delta-\frac{\eta\{\mu(1-r)+\phi\}}{\alpha}\right].$$

By looking at eigen values, one can easily seen that disease-free equilibrium E_1 is locally stable if $\beta\delta-\frac{\eta\{\mu(1-r)+\phi\}}{\alpha}>0$ ie $\Re_0<1$. Now we can turn to an endemic equilibrium and study about its stability.

For the endemic equilibrium point $E_2=(x_e, y_e, z_e)$, the variation matrix will be

$$J(E_2)=\begin{pmatrix} -\alpha-\eta z_e & -\phi & -\eta x_e \\ \eta z_e & -\beta & \eta x_e \\ 0 & 1-z_e & -y_e-\delta \end{pmatrix},$$

$$J(E_2)=\begin{pmatrix} -\frac{(\eta+\alpha)\left[\mu(1-r)+\phi\right]-\alpha\delta\phi}{\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]} & -\phi & -\frac{\eta\beta\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]}{\eta(\beta+\phi)+\alpha\beta} \\ \frac{\eta\left[\mu(1-r)+\phi\right]-\alpha\beta\delta}{\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]} & -\beta & \frac{\eta\beta\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]}{\eta(\beta+\phi)+\alpha\beta} \\ 0 & \frac{\eta(\beta+\phi)\delta+\alpha\beta\delta}{\eta\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]} & -\frac{\eta\left[\mu(1-r)+\phi+(\beta+\phi)\delta\right]}{\eta(\beta+\phi)+\alpha\beta} \end{pmatrix}.$$

Its characteristic equation will be

$$a_3\lambda^3+a_2\lambda^2+a_1\lambda+a_0=0,$$

where

$$a_3=\left[\beta(\eta+\alpha)+\eta\phi\right]\left[\mu(1-r)+\phi(1+\delta)+\beta\delta\right]>0,$$

$$\begin{aligned} a_2=&\left[\beta\{\beta(\eta+\alpha)+\eta\phi\}+\eta\{\mu(1-r)+\phi(1+\delta)+\beta\delta\}\right]\left[\mu(1-r)+\phi(1+\delta)+\beta\delta\right] \\ &+\left[\beta(\eta+\alpha)+\eta\phi\right]\left[(\eta+\alpha)\{\mu(1-r)+\phi\}+\alpha\delta\phi\right] \\ &>0, \end{aligned}$$

$$a_1 = \left[\eta\{\mu(1-r)+\phi\}-\alpha\beta\delta\right]\left[\beta\{\mu(1-r)+\phi(1+\delta)+\beta\delta\}+\phi\{\beta(\eta+\alpha)+\eta\phi\}\right]+$$
$$+\left[(\eta+\alpha)\{\mu(1-r)+\phi\}+\alpha\delta\phi\right]\left[\beta\{\beta(\eta+\alpha)+\eta\phi\}+\eta\{\mu(1-r)+\phi(1+\delta)+\beta\delta\}\right],$$
$$a_0 = \left[\eta\{\mu(1-r)+\phi\}-\alpha\beta\delta\right]$$
$$*\left[\beta(\eta+\alpha)\{\mu(1-r)+\phi\}+\beta\alpha\delta\phi+\eta\phi\{\mu(1-r)+\phi(1+\delta)+2\beta\delta\}+\beta^2\delta(\eta+\alpha)\right].$$

We see that $a_1, a_0 > 0$ if $\left[\eta\{\mu(1-r)+\phi\}-\alpha\beta\delta\right] > 0$ i.e. $\Re_0 > 1$. Hence, the endemic equilibrium point E_2 is locally stable if $\Re_0 > 1$. We conclude this in the following theorem:

Theorem 3.1: If $\Re_0 < 1$, then the disease-free equilibrium E_1 is locally stable; if $\Re_0 = 1$, E_1 is stable and if $\Re_0 > 1$, the stable endemic equilibrium E_2 will appears.

4. Numerical simulation

In this section, we give an example to illustrate the main theoretical results presented above. In system (2.5), let $\mu = 0.0016$, $r = 0.08$, $\alpha = 1.0016$, $\eta = 0.276$, $\phi = 0.8$, $\beta = 0.0136$ and $\delta = 1$. Computation gives the following value for the basic reproduction number $\Re_0 = 16.23918 > 1$ and system (2.5) has a unique endemic equilibrium point $E_2 = (x = 0.09222, y = 0.871562, z = 0.465687)$. By theorem 3.1, we see that endemic equilibrium E_2 of system (2.5) is globally asymptotically stable if $\Re_0 > 1$. Numerical simulation illustrates the above result (see figure 1).

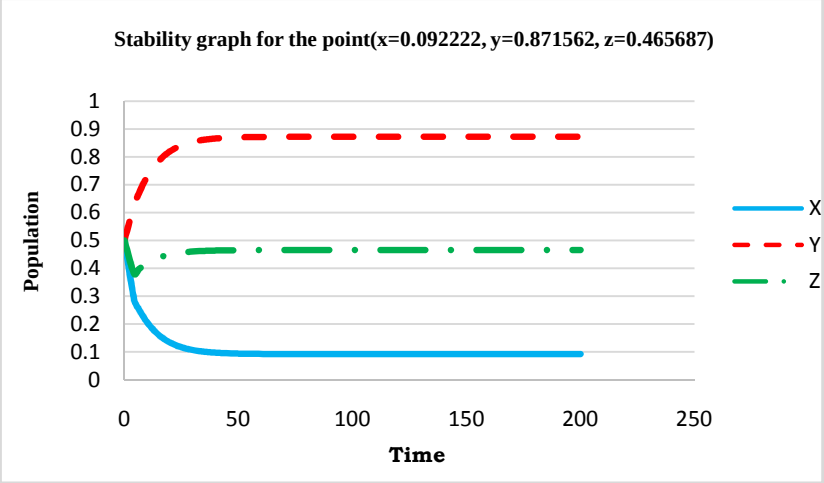

Figure 1

CONCLUSION

In this paper we have discussed the effects of vaccination strategies on the dynamic of the dengue disease transmission model with assumption that a random fraction of the recovered host population can loses the immunity and becomes susceptible again. Dynamic of the model is completely determines by the basic reproduction number $\Re_0$. We have proved that the model has a disease-free equilibrium E_1 if the reproduction number $\Re_0$ is less than or equal one and has a unique positive endemic equilibrium E_2 if the reproduction number $\Re_0$ is greater than one. Stability conditions are given which would be a useful tool for the disease control strategies. For $\phi_H = 0$, the model coincides with that of Soewono and Supriatna [9].

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