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Dynamic of a Multi-serotype Epidemic Model with Distributed Delay

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Motivated by the growing discussion about dengue fever, we proposed a model to study a multi-serotype infectious disease, for example, dengue. The main purpose is to include and analyze the effect of the general time delay on the model describing the length of the cross immunity protection and the effect of ADE-Antibody Dependent Enhancement, both characteristic of Dengue fever [2]. As an illustration, in this work, we restrict ourselves to two serotypes.

Let $N(t)$ be the total host population in a region. We divided the total population in classes according to the status, susceptible for all serotypes, infected by serotype i, temporarily immune for all serotypes, recovered for serotype i but susceptible to the others and, recovered for all serotypes, represented at time t, respectively by $S(t)$, $I_i(t)$, $C_i(t)$, $R_i(t)$ e $R(t)$. We represent the reinfection by serotype j, after being infected by serotype i, by $I_{ij}(t)$. We assume the Ricker's function for births in susceptible class, let d be the natural mortality, β_i the average number of effective contacts per infected individual, per unit time, by serotype i, and α_j the probability of been infected again by different serotype. We will not consider the mosquito population explicitly in the model. Moreover, β_i and α_j will denote the average number of bite and the probability of a susceptible individual being bitten by an infected mosquito with serotype i and, respectively, the probability of an individual recovered from the serotype i being bitten again by an infected mosquito with serotype j. Moreover, let $P^i(t)$ be the function that describes the immunity period, that is, the time that an individual remain in the class C_i, t units of time after entering. We assume that $P^i(0) = 1$, $P^i(\infty) = 0$ and, $P^i(t)$ is non-increasing. The individuals recovers at a constant rate γ. The epidemiological effects of ADE is described through the constant rate ϕ, that represents the degree of enhancement, where it assumed that previously exposure to one serotype results in increasing probability of being reinfected and, therefore, an increase of the transmission rate.

For this model, we found the equilibriums of the limiting system in the invariant region. The local stability for the Disease Free equilibrium and for the boundary endemic equilibriums were proved. Using the theory found in Brauer [1] we have the results about

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the stability of the solutions of the system, that is completely determined by the Basic Reproduction Number and by the Invasion Reproduction Number, defined mathematically, as a threshold value for stability [3]. The global dynamics is investigated, by constructing Lyapunov functionals. The model is described by

$$\frac{dS(t)}{dt} = rN(t)e^{-kN(t)} - dS(t) - \beta_1\frac{S(t)}{N(t)}(I_1(t) + I_{21}(t)) - \beta_2\frac{S(t)}{N(t)}(I_2(t) + I_{12}(t)) \quad (1)$$

$$\frac{dI_1(t)}{dt} = -(d+\gamma)I_1(t) + \beta_1\frac{S(t)}{N(t)}(I_1(t) + I_{21}(t)) \quad (2)$$

$$\frac{dI_2(t)}{dt} = -(d+\gamma)I_2(t) + \beta_2\frac{S(t)}{N(t)}(I_2(t) + I_{12}(t)) \quad (3)$$

$$C_1(t) = \int_0^t \gamma I_1(s)P^1(t-s)e^{-d(t-s)}ds \quad (4)$$

$$C_2(t) = \int_0^t \gamma I_1(s)P^2(t-s)e^{-d(t-s)}ds \quad (5)$$

$$\frac{dR_1(t)}{dt} = -dR_1(t) - \alpha_2\phi\frac{R_1(t)}{N(t)}(I_{12}(t) + I_2(t)) - \int_0^t \gamma I_1(s)P_t^1(t-s)e^{-d(t-s)}ds \quad (6)$$

$$\frac{dR_2(t)}{dt} = -dR_2(t) - \alpha_1\phi\frac{R_2(t)}{N(t)}(I_{21}(t) + I_1(t)) - \int_0^t \gamma I_2(s)P_t^2(t-s)e^{-d(t-s)}ds \quad (7)$$

$$\frac{dI_{12}(t)}{dt} = -(d+\gamma)I_{12}(t) + \alpha_2\phi\frac{R_1(t)}{N(t)}(I_2(t) + I_{12}(t)) \quad (8)$$

$$\frac{dI_{21}(t)}{dt} = -(d+\gamma)I_{21}(t) + \alpha_1\phi\frac{R_2(t)}{N(t)}(I_1(t) + I_{21}(t)) \quad (9)$$

$$\frac{dR(t)}{dt} = -dR(t) + \gamma(I_{12}(t) + I_{21}(t)) \quad (10)$$

$$\frac{dN(t)}{dt} = rN(t)e^{-kN(t)} - dN(t). \quad (11)$$

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