

A Study on Application of System Biology for Mathematical Modelling of Dengue

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Abstract - : Dengue is a mosquito-borne viral infection that is usually found in tropical and subtropical regions around the world. In recent years, transmission has increased predominantly in urban and semi-urban areas and has become a serious public health concern. . It is transmitted to the man by mosquitoes (*Aedes*) and exists in two forms ie. Dengue Fever and Dengue Haemorrhagic Fever. The disease are often contracted by one among the four different viruses. Mathematical modeling is a strong & reliable tool to find and compare different intervention strategies which may be useful in controlling or eliminating Dengue, which is particularly important in our world of limited resources. The different types mathematical models help us conceptualize the transmission dynamics during a quantitative way also as allow us to check different hypotheses to know their importance. In this paper a comparison and contrast of two different models of Dengue and identify their best features along with their performance for various scenarios has been done.

Key Words: SIR model, DEROUICH model, dengue fever, haemorrhagic, vaccination.

1. INTRODUCTION

Dengue is a mosquito-borne viral infection that is usually found in tropical and subtropical regions around the world. There are four different, but closely related, serotypes of the virus that cause Dengue (DEN-1, DEN-2, DEN-3 and DEN-4)[1]. Recovery from infection by one provides lifelong immunity against that specific serotype. However, cross-immunity to the opposite serotypes after recovery is merely partial and temporary. Subsequent infections by other serotypes increase the danger of developing severe dengue. A person who has been infected by one among the four serotypes will never be infected again by an equivalent serotype (homologous immunity), but he loses immunity to the three other serotypes (heterologous immunity) in about 12 weeks then becomes more vulnerable to developing dengue fever.

The first form is characterized by a sudden fever without respiratory symptoms, amid intense headaches making its nickname "breakbone fever" well deserved. It lasts between three and seven days but it stays benign. The haemorrhagic form (DHF) is additionally characterized by a sudden fever, nausea, vomiting and fainting thanks to low vital sign caused by fluid leakage. It usually lasts between two and three days and may cause death. The case of a second infection has therefore a capital importance due to the likelihood of evolution toward the haemorrhagic sort of the disease.

2. MATHEMATICAL PROCEDURE

Mathematical modelling became a stimulating tool for the understanding of those illnesses and for the proposition of strategies. The formulation of the model and therefore the possibility of a simulation with parameter estimation, all-low tests for sensitivity and comparison of conjunctures.

In this paper we have compared and contrast two different models of Dengue and identify their best features along with their performance for various scenarios using different models.

A. The S – I – R model: The basic model is a model in which a constant population is divided into three compartments of individuals depending on their infection status: susceptible S, infected I and recovered R. This is usually referred to as the S–I –R model. These compartments are explained as follows:

- i. S is used to represent the number of individuals who are susceptible to the disease at time t.
- ii. I denote the number of individuals who have been infected with the disease and are capable of spreading the disease to those in the susceptible category.
- iii. R represents the number of individuals who have been infected and recovered from the disease. Those during this category are resistant to infection and that

they wouldn't transmit the infection to others.

iv. Assumptions:- Each compartment is assumed to be homogeneous. In other word, individuals in each compartment are randomly mixing with each other. This is almost like mass-action principle model in chemistry. The per capita rate of infection and the per capita rate of recovery are assumed to be independent of the length of time the person has spent in each compartment. They are assumed to follow an exponential distribution. The basic S - I - R model is formulated as:

$$\frac{dS}{dt} = -\lambda S, \quad \frac{dI}{dt} = \lambda S - \gamma I$$

$$\frac{dR}{dt} = \gamma I, \quad S + I + R = N$$

Where λ is the force of infection and γ is the mean recovery rate and N is the total population. S-I-R models can have separate formulations, counting on the essential assumptions regarding the force of infection: density-dependent and frequency-dependent.

1. Density-dependent model: The density-dependent model assumes that all members of a population existing in a fixed area come in contact with one another no matter how many individuals are present in the population. Therefore, the force of infection is defined as $\lambda = \beta I$ where β denotes the transmission coefficient (which is that the product of the amount of contact per susceptible person per unit time and therefore the probability of a successful transmission of the infection given the contact). Assuming that β is a constant, the force of infection depends on the number of infected persons in the population, [3].

2. Frequency-dependent models: However it's been shown that for many human infections, the amount of individuals everyone is in touch with per day is fairly Constant across the planet, no matter the population density of the place. That is why an alternate, referred to as the frequency-dependent," formulation of the SIR model is usually wont to model the transmission of human diseases, where the force of infection is defined as $\lambda = \beta (I/N)$. The term I/N is the probability that any random contact that a susceptible person makes will be with someone Infectious, which is equivalent to the proportion of the total population that is infectious, [3].

B. DEROUICH MODEL OF DENGUE FEVER : We first study the model of Dengue fever developed by Derouich et al in [3]. Their model is based on the compartmental diagram shown in Figure1. The host population, N_h , contain susceptibles, S_h , infectives, I_h , and removed, R_h . Its corresponding vector population, N_v , contains susceptibles, S_v and infectives I_v . Mosquitos are a

reservoir host for the four viruses that cause Dengue fever: they are carriers of the virus but not negatively affected by it. Hence, there's not a "removed vector population" to think about. For the human population, the model developed by Derouich et al., [3], takes the shape

$$\frac{dS_h}{dt} = \mu_h N_h - (\mu_h + p + C_{vh} I_v / N_h) S_h$$

$$\frac{dI_h}{dt} = \left(\frac{C_{vh} I_v}{N_h} \right) S_h - (\mu_h + \gamma_h) I_h$$

$$\frac{dR_h}{dt} = (p S_h + \gamma) I_h - \mu_h R_h$$

The parameter values are described in Table 1. One of the key features of the model is the fraction, p, that represents a (random) fraction of the human population that can be permanently immunized against the four serotypes that cause Dengue fever. For the vector population

$$\frac{dS_v}{dt} = \mu_v N_v - (\mu_v C_{hv} I_h / N_h) S_v$$

$$\frac{dI_v}{dt} = \left(\frac{C_{hv} I_h}{N_h} \right) S_v - \mu_v I_v$$

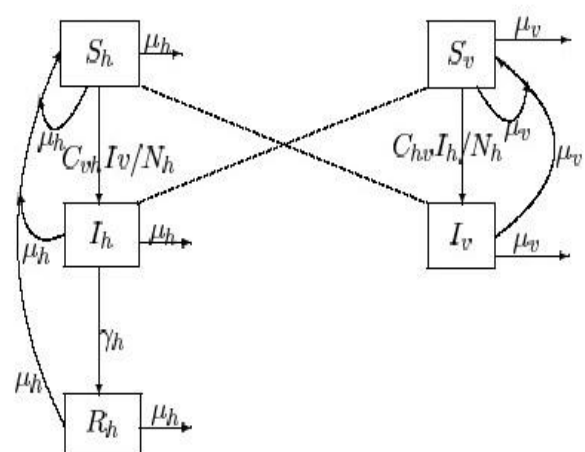


Figure 1: The block diagram used by Derouich et al in [3] in the formulation of their model of Dengue fever.

$$\frac{dS_h}{dt} = \mu_h N_h - (\mu_h + p + C_{vh} I_v / N_h) S_h$$

$$\frac{dI_h}{dt} = \left(\frac{C_{vh} I_v}{N_h} \right) S_h - (\mu_h + \gamma_h) I_h$$

$$\frac{dI_h}{dt} = \left(\frac{C_{vh}I_v}{N_h}\right)(N_v - I_v) - \mu_v I_v$$

The main result of Derouich et al in [3] is that system has two equilibrium points,

$$S_h^* = \frac{N_h(\beta + M)}{\left(1 + \frac{p}{\mu_h}\right)\beta + MR}$$

$$I_h^* = \frac{N_h(R - 1 - p/\mu_h)}{\left(1 + \frac{p}{\mu_h}\right)\beta + MR}$$

$$I_v^* = \frac{\beta N_v(R - 1 - p/\mu_h)}{R(\beta + M)}$$

β , M , and R are given by $\beta = C_{hv}/\mu_v$, $M = (\mu_h + \gamma_h)/\mu_h$, and $R = C_{vh}C_{hv}N_v/(\mu_v(\mu_h + \gamma_h)N_h)$. Analysis of the Jacobian at E_1 and E_2 shows that E_1 is globally asymptotically stable if $R \leq 1 + p/\mu$ and E_2 is locally stable if $R > 1 + p/\mu$. To develop a deeper understanding of the model we conduct several simulations.

Parameter Notation Base Value

Transmission probability of vector to human	
Phv	0:75
Transmission probability of human to vector	
pvh	0.75
Bites per susceptible mosquito per day	bs 0.5
Bites per infectious mosquito per day	bi 1.0
Effective contact rate: human to vector	
Chv=pvhbs	0.375
Effective contact rate: vector to human	
Cvh=pvhbi	0.75
Human life span	
1= h	25000 Days
Vector life span	
1= v	4 days

Table 1: Parameter values used following an equivalent choices as in Derouich et al, [3]

3. RESULT

Our first simulation is done on the variation of Vaccination levels of a whole population. In Fig 2 we numerically show the change in outbreak behavior using four levels of total population vaccinated. From Figure .2. It is seen that if 20% of the population is vaccinated, the outbreak of the epidemic decreases the number of infected hosts

during the outbreak by three times. If half the population is vaccinated, there's almost no outbreak and if 90% of the population is vaccinated there's no outbreak. The second scenario is predicated on the idea that for various environment temperatures the activity level of mosquitoes differs. For this model, our final simulation is predicated on the hypothetical size of mosquito population and its influence on the dimensions of the outbreak within the human population.

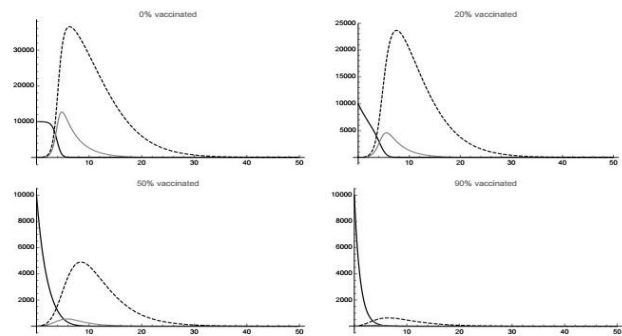


Figure.2: Numerical simulations of the model by Derouich et al., [3] for the different levels of population vaccinated of a total population vaccinated.

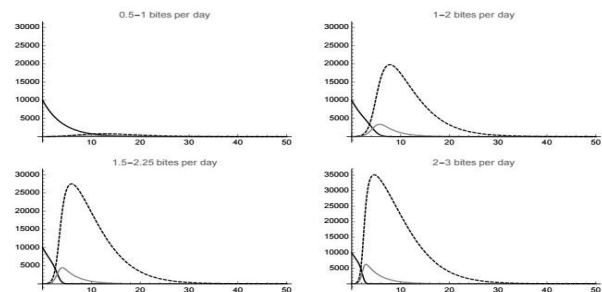


Figure 3: Numerical simulations of the model by Derouich et al., [3], for different levels of mosquito activity bites per susceptible/infectious mosquito per day).

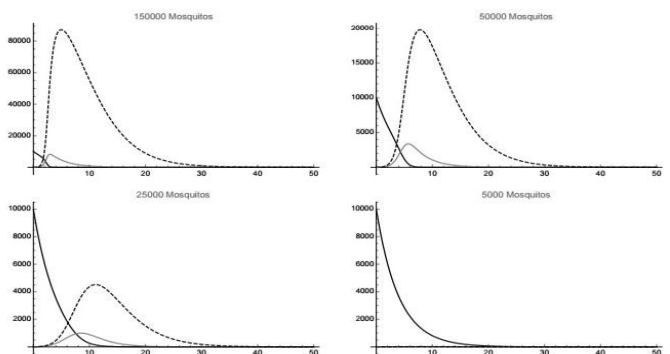


Figure 4: Numerical simulations of the model by Derouich et al., [3] for different levels of mosquito population.

4. CONCLUSION

The last simulation illustrates the importance of various control measures of mosquito population. In Figure 4, we see that a substantial decrease of mosquito population can almost prevent an epidemic of Dengue within the human population. Consistent with these scenarios it's difficult to spot which parameter affects the severity of an epidemic the foremost. However, the amount of mosquitoes and therefore the vaccination level of the susceptible population appear to be of high importance. Despite the very fact that vaccination campaigns are often easily implemented, they're effective as long as only one strain of the virus is present within the environment.

REFERENCES

- [1] Derouich M Modélisation et Simulation de modèles avec et sans structure d'âge: Application au diabète et à la fièvre dengue. Ph.D thesis Faculty of Sciences, Oujda Morocco 2001,
- [2] Sesser S Plague proportion. The Asian Wall Street Journal 2002 Au-gust 30 Septembre 1
- [3] Cummings DAT, Lesser J. Infectious Disease Dynamics. In: Nelson KE, Masters
- [4] M. Derouich, A. Boutayeb, and E.H. Twizell, "A model of Dengue fever," BioMedical Engineering OnLine, 2:4, 2003.
- [5] Newton EA and Reiter P A model of the transmission of dengue fever with an evaluation of the impact of ultra-low volume (ULV) Insecticide applications on dengue epidemics. Am J Trop Med Hyg 1992, 47:709-720
- [6] Esteva L and Vargas C Analysis of a dengue disease transmission model. Mathematical Biosciences 1998, 150:131-151
- [7] Feng Z and Hernández V Competitive exclusion in a vector-host model for the dengue fever. Journal of Mathematical Biology 1997, 35:523-544
- [8] Luenberger DG Introduction to Dynamic systems, Models, and Applications. Theory, Models, and Applications, John Wiley & sons 1997,
- [9] Luenberger DG Introduction to Applied Non linear systems and Chaos. S Wiggins, Springer 1996,
- [10] Dietz K Transmission and control of arbovirus diseases. In Pro-ceedings of the Society for Industrial and Applied Mathematics, Epidemiol-ogys: Philadelphia (Edited by: Ludwing D) Philadelphia 1974, 104-106