

STUDY ON SPECULATION OF THE DETERMINISTIC ODE DENGUE

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ABSTRACT

In this model a lot of four conditions for people and three conditions for mosquitoes has been made. Here, up to 17 parameters and 7 states have been characterized. The model has been additionally improved to partial amounts to take out figuring troubles. The model has been subjectively reached out to numerous locales with legitimate presumptions. Further, the model has been broke down. It has been indicated that there exists an area where the model is epidemiologically and numerically well-presented. The model incorporates a period delay between a T-cell ending up inactively tainted and beneficially contaminated. The model has an infection free and a ceaseless disease balance. It is indicated that the model has Andronov-Hopf bifurcations prompting farthest point cycle conduct in the interminable contamination locale at basic estimations of the time delays.

Keywords: *Mathematical, Model, Dengue, Transmission*

INTRODUCTION

Dengue fever (DF) and Dengue Haemorrhagic Fever (DHF) are significant general medical issues in the tropic and subtropics territories. Dengue infections are transmitted to human by the nibble of *Aedes aegypti* female mosquitoes causing Dengue fever (DF). Successive contamination with dengue fever expands the danger of Dengue Haemorrhagic Fever (DHF). Female *Aedes aegypti* get contamination by taking a blood supper from a tainted human. These contaminated mosquitoes transmit the pathogen to powerless people. Four diverse serotypes that can cause dengue fever (DEN-1-4) can exist together in numerous endemic territories. Infection with one of dengue serotype has been appeared to give long lasting insusceptibility to that serotype however not or just transient protection from the different serotypes.

Mathematical model for dengue transmission

We consider the numerical model for dengue transmission depicted by an arrangement of non-direct customary differential conditions. This model depicts the cooperation between powerless, contaminated and recuperated human populaces together with defenseless and tainted mosquito populaces.

$$\begin{aligned}\frac{dS^h}{dt} &= \lambda N_T - \frac{b\beta_h}{N_T} S^h I^v - \mu_h S^h \\ \frac{dI^h}{dt} &= \frac{b\beta_h}{N_T} S^h I^v - (\mu_h + r) I^h \\ \frac{dR^h}{dt} &= r I^h - \mu_h R^h \\ \frac{dS^v}{dt} &= D - \frac{b\beta_v}{N_T} S^v I^h - \mu_v S^v \\ \frac{dI^v}{dt} &= \frac{b\beta_v}{N_T} S^v I^h - \mu_v I^v\end{aligned}$$

$$\text{with } N_T = S^h + I^h + R^h \text{ and } N_v = S^v + I^v$$

Where

NT – The complete number of human populace

O – The birth pace of the human populace b – the gnawing pace of the mosquitoes

h E – The transmission likelihood of the infection from mosquitoes to people

v E – The transmission likelihood of the infection from people to mosquitoes

P h – the demise pace of the human populace r – the recuperation pace of the human populace

D – the consistent enrollment pace of the mosquito populace

Pv – the demise pace of the mosquito populace and h S – powerless human populace h I – tainted human populace h R – recouped human populace v S – helpless mosquito populace v I – contaminated mosquito populace NT – all out human populace Nv – all out mosquito populace.

Mathematical Model

For effortlessness, just a solitary serotype model is considered in this paper. We depict the elements of dengue in its three parts of transmission: the oceanic mosquitoes (including the eggs, hatchlings and pupae), the grown-up mosquitoes and human hosts. We partition the oceanic mosquitoes into powerless (SA) and tainted amphibian mosquitoes (IA) subgroups. The brooding time frame for grown-up mosquitoes keeps going somewhere in the range of 10 and 12 days for a normal mosquito life expectancies seething from 11 to 20 days, and in this manner, ought not be disregarded in the transmission of dengue. The grown-up mosquitoes are partitioned into powerless (AM), uncovered (EM) and irresistible (IM) subgroups. Like the supposition in the mosquito populace pursues a strategic development. Proof shows that vertical transmission of the infection exists in mosquitoes (Buckner et al. which is portrayed by the term $(1 - v)\mu MIM(1 - NA kA)$). The human populace is separated into helpless (SH), uncovered (EH), irresistible (IH) and recuperated (RH) subpopulations. We accept that individuals are safe after they recoup. We build the model dependent on the dengue endemic circumstance in Guangdong Province of China in 2014. The complete populace of Guangdong Province was 106.44×10^6 out of 2013, the birth rate was 10.71h, and the infant populace in 2013 was 1, 137, 300 (SBGP [39]).

Dengue Model

Considering the model exhibited in [5, 6] and the contemplations of [7, 8], another model progressively adjusted to the dengue the truth is proposed. The documentation utilized in the scientific model incorporates three epidemiological states for humans: —defenseless (people who can get the disease), —contaminated (people fit for transmitting the disease), —safe (people who have obtained insusceptibility). It is accepted that the all out human populace, is steady: whenever. The populace is homogeneous, which implies that each person of a compartment is homogeneously blended with different people. Movement and migration are not considered. Three other state factors, identified with the female mosquitoes, are considered: —amphibian stage (that incorporates the egg, hatchling, and pupa stages), —vulnerable (mosquitoes that can get the disease), —contaminated (mosquitoes fit for transmitting the disease). Note that male mosquitoes are not considered, on the grounds that

they are not fit for transmitting the disease and that there is no safe stage, because of the short life expectancy of mosquitoes.

It is expected homogeneity among host and vector populaces, which implies that every vector has an equivalent likelihood to nibble any host. People and mosquitoes are thought to be brought into the world powerless. The dengue pestilence is displayed by the accompanying nonlinear arrangement of time-shifting ODEs (normal differential conditions):

$$\begin{aligned} S_h(t) &— \\ I_h(t) &— \\ R_h(t) &— \end{aligned}$$

$$\begin{aligned} A_m(t) &— \\ S_m(t) &— \\ I_m(t) &— \end{aligned}$$

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It is accepted homogeneity among host and vector populaces, which implies that every vector has an equivalent likelihood to nibble any host. People and mosquitoes are thought to be brought into the world defenseless. The dengue pestilence is demonstrated by the accompanying nonlinear arrangement of time-fluctuating ODEs (common differential conditions):

$$\begin{aligned} \frac{dS_h}{dt} &= \mu_h N_h - \left(B\beta_{mh} \frac{I_m}{N_h} + \mu_h \right) S_h, \\ \frac{dI_h}{dt} &= B\beta_{mh} \frac{I_m}{N_h} S_h - (\eta_h + \mu_h) I_h, \\ \frac{dR_h}{dt} &= \eta_h I_h - \mu_h R_h, \\ \frac{dA_m}{dt} &= \varphi \left(1 - \frac{A_m}{kN_h} \right) (S_m + I_m) - (\eta_A + \mu_A) A_m, \\ \frac{dS_m}{dt} &= \eta_A A_m - \left(B\beta_{hm} \frac{I_h}{N_h} + \mu_m \right) S_m, \\ \frac{dI_m}{dt} &= B\beta_{hm} \frac{I_h}{N_h} S_m - \mu_m I_m, \end{aligned}$$

Cholera

Cholera has been researched since the introduction of the study of disease transmission, and it is as yet a subject of extreme enthusiasm for cutting edge disease transmission experts. Regardless of much epidemiological investigations of this disease, it is evaluated that 3 –5 million cholera cases and 100000 – 120000 passings because of cholera consistently in everywhere throughout the world (W.H.O, 2014). These insights are gross thinks little of, the same number of cholera pestilence nations don't report cholera to the W.H.O and a high level of underreporting for the W.H.O included cholera nations. It assaults both adolescent and grown-ups everywhere throughout the universes, yet the majority of the cholera cases are packed in immature and creating countries where sufficient water treatment, legitimate sanitation, and reasonable cleanliness are not met.

Since it keeps on being a danger to the significant bits of the world, it is essential to comprehend the disease elements and how communications with en-vironmental and human components add to the plague conduct saw during ebb and flow cholera episodes. Moreover, the huge measure of information benefit capacity of the disease makes it a perfect framework to ponder. Generally, there have been seven recognized cholera pandemics; late flare-ups in Zimbabwe and Haiti are incorporated into the seventh and progressing pandemic.

OBJECTIVES OF THE STUDY

1. To study on Mathematical model for dengue transmission
2. To study on Simplification of SPR_SODE Model

RESEARCH METHODOLOGY

Modeling the Epidemiology

Numerical displaying is a procedure by which a genuine issue is portrayed by a scientific Formulation (D. Murthy, N. Page, and E. Rodin, 1990). Numerical demonstrating can support our comprehension and evaluation of the present and future hazard zones or spread of irresistible malady (Get his et al., 2011). It likewise causes us to assess the techniques on

battling dengue (2008). There are numerous numerical models for different infections. Consider the model Susceptible(S), Infection (I), Recovery (R) known as SIR by Kermack-McKendrick (1927). SIR is one of the most essential epidemiological models. This is the most broadly utilized model for the spread of malady. In 1957 MacDonald improved the model to a two dimensional model with one variable speaking to human and the other one speaking to mosquitoes.

Simplification of SPR_SODE Model

Stream outline, fulfill the accompanying arrangement of Ordinary differential conditions,

which become SODE model for DF. $\left[\frac{dx}{dt} \right]_k = \left[\frac{df}{dt} \right]_k = \left[\frac{rc}{dt} \right]_k = \left[\frac{ex}{dt} \right]_m$, and $\left[\frac{if}{dt} \right]_m$ signify at time 't', the extent of uncovered human contaminated human, recuperated human, uncovered mosquitoes, and tainted mosquitoes separately.

Reproductive Number

Presently, one of the parameters in epidemiological models is the regenerative number R_0 . This R_0 can be characterized as the quantity of diseases that would result from one irresistible individual (Either human or mosquito) over the irresistible period, given that the various people are powerless and that is initially produced for socioeconomics. Presently broadly utilized for irresistible sickness, R_0 is "One of the chief and most important thoughts that numerical reasoning has brought to pestilence hypothesis" (Heesterbeek and Dietz, 1996).

- If $R_0 < 1$, every individual produces, on a normal, short of what one new tainted individual and consequently the ailment ceases to exist
- If $R_0 > 1$, every individual creates more than one new contaminated individual and thus the infection can attack the vulnerable populace
- This enables us to decide the viability of control measures.

Improved SPR_ SODE Model On Dengue Fever

In the event that the populace isn't homogeneously blended and one needs to research the spread of malady, the augmentation is very significant, In the fundamental model a supposition that is made that any individual is similarly liable to contaminate some other individual. For enormous and thick populace, this may be valid however it may not be a decent guess in for all demonstrating circumstances.

Relation with Eigen Values

Evaluating the substitution of the expansions into the Eigen value equation at $O(\varepsilon^1)$, $u(1) = \lambda_1 Lu(1)$. This implies that u is the right Eigenvector of L corresponding to the Eigen value $1/\lambda$. Thus, close to the bifurcation point, the equilibrium point can be approximated by $[ex]_h = \varepsilon\sqrt{A}$ and $[ex]_m = \varepsilon\sqrt{B}$, where ε is arbitrarily small and close to the bifurcation point. Hence the initial direction of the branch of equilibrium points, $u(1)$, near the bifurcation point, $(\lambda_1, 0)$, is equal to the right Eigenvector of L corresponding to the characteristic value $1/\lambda$.

The Jacobean is a matrix of partial derivatives which is created by differentiating every equation with respect to every variable. If there are 7 equations and 7 variables one can have a 7×7 matrix. 1. Calculate the disease-free equilibrium 2. Create the Jacobian matrix 3. Evaluate the Jacobean at the equilibrium 4. Find the Eigen values 5. If all Eigen values are less than zero, it implies stable and if even one Eigen value is greater than zero, it implies unstable 6. Largest Eigen value implies R_0 -like threshold. If $\max\{|\lambda|: \lambda \text{ Eigen value of } K\} = 1$, then we cannot give a statement about the stability of the fixed point by that criterion; the behavior then depends on higher order terms than linear ones. The Jacobean can determine stability of equilibrium. It can also lead us to R_0 -like thresholds. These determine whether an epidemic will persist or die out. The different methods of finding R_0 are given below. Linear stability on the two equilibrium points is conducted at without disease, m , x , and n .

DATA ANALYSIS

Baseline Parameter Values

Benchmark esteems are appeared in Table 8.1 for the parameters. Two arrangements of benchmark esteems are considered: one for zones of high transmission and another for low transmission (as estimated by $0 R$). In Appendix, our purposes behind utilizing these qualities and suitable references any place accessible are depicted. From distributed investigations and nationwide data for some parameter esteems are evaluated. For area explicit parameters, for example, relocation rates, reasonably achievable qualities are picked. For parameters concerning human populaces, values speaking to towns, communities, or little districts are picked. Two critical figures of exactness for every one of the parameters are utilized.]

Low Transmission

For the benchmark parameters at low dengue transmission in Table 1, $0 R = 1.1$ (relating to 1.3 new contaminations in people from one tainted human through the length of the irresistible (and recouped) period). There is just a single locally asymptotically stable endemic harmony point in D,

$$x_{ee} = (0.0029, 0.080, 0.10, 578, 0.024, 0.016, 2425)$$

Figure 1 shows the graphs of a typical solution of the model (A) with respect to the original variables.

Parameters	Dimensions	Baseline high	Baseline low
β_h	Humans x day ⁻¹	0.033	0.041
$[BIR]_h$	day ⁻¹	1.1x10 ⁻⁴	5.5x10 ⁻⁵
$[BIR]_m$	day ⁻¹	0.13	0.13
Φ_m	day ⁻¹	0.50	0.33
Φ_h	day ⁻¹	19	4.3
P_{mh}	1	0.022	0.022
P_{hm}	1	0.48	0.24
\overline{P}_{hm}	1	0.10	0.024
ξ_h	day ⁻¹	0.048	0.10
ξ_m	day ⁻¹	0.10	0.083
θ_h	day ⁻¹	0.091	0.0035
η_h	day ⁻¹	0.0035	1.8x10 ⁻⁵
L_h	day ⁻¹	9.0x10 ⁻⁵	2.7x10 ⁻⁷
$[DID]_h$	day ⁻¹	5.5x10 ⁻⁴	8.8x10 ⁻⁵
$[DDD]_h$	Humans x day ⁻¹	1.6x10 ⁻⁵	2.0x10 ⁻⁷
$[DID]_m$	day ⁻¹	3.0x10 ⁻⁷	0.033
$[DDD]_m$	mosquitoes x day ⁻¹	0.033	4.0x10 ⁻⁵

High transmission

For the standard parameters at high dengue transmission in Table 8.1, $R_0 = 4.4$ (comparing to 20 new contaminations in people from one tainted human through the length of the irresistible (and recuperated) period). There is just a single locally asymptotically stable endemic harmony point in D,

$$x_w = (0.0059, 0.16, 0.77, 490, 0.15, 0.11, 4850)$$

Figure 2 shows the graphs of a typical solution of the model (A) with respect to the original variables.

Sensitivity Analysis

In deciding how best to lessen human mortality and grimness because of dengue, it is important to know the general significance of the various variables liable for its transmission and predominance. Starting sickness transmission is legitimately identified with R_0 , and

malady predominance is straightforwardly identified with the endemic balance point, explicitly to the extents of

$$[ex]_h, [if]_h, [rc]_h, [ex]_m, \text{ and } [if]_m.$$

In deciding how best to decrease human mortality and dismalness because of dengue, it is important to know the general significance of the various components answerable for its transmission and predominance. Introductory malady transmission is straightforwardly identified with 0 R, and sickness commonness is legitimately identified with the endemic balance point, explicitly to the extents of

$[if]_h$ is particularly significant in light of the fact that it speaks to the individuals who might be clinically sick, and is straightforwardly identified with the all out number of dengue passings. The affectability records of the conceptive number 0 R and the endemic balance direct ee x toward the parameters are determined in the model. These records disclose to us how pivotal every parameter is to sickness transmission and commonness. Affectability investigation is regularly used to decide the heartiness of model expectations to parameter esteems (since there are normally blunders in data collection and assumed parameter esteems). Here we use it to find parameters that highly affect 0 R and ee x and ought to be focused by mediation systems.

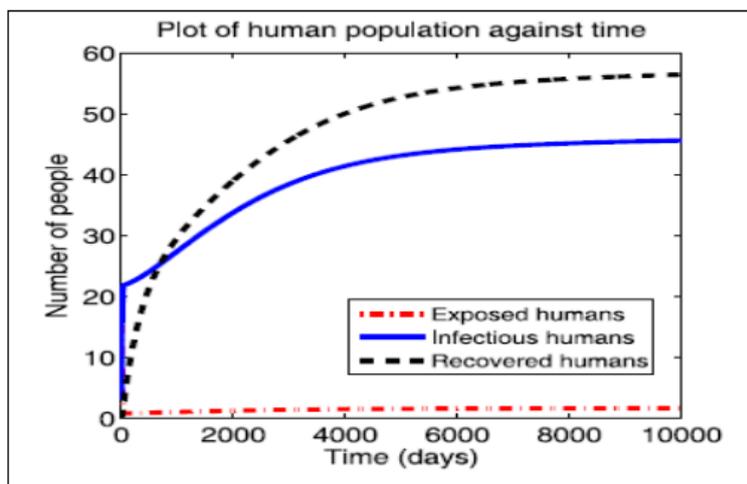


Fig. 3 Plot of Time vs human population – except susceptible for initial condition 3

Further Investigation of Stochastic Behavior

In the previous section the effect of the stochastic approach has been limited to random fluctuations around the trajectories in the deterministic model. This is natural since there exists only one stable equilibrium point for the system when looking at parameter values for a high transmission area. Thus there is only so much "damage" the stochastic approach can do result is shown if initial values for the system between the two trajectories in Figure 8.10 are selected. This is to be noted that the initial values cause the deterministic trajectory to seemingly fall between the two stable equilibria. (This only looks to be the case; however, in fact, it tends to the stable disease free equilibrium.) The stochastic trajectory on the other hand sometimes heads for the endemic equilibrium and sometimes for the disease free equilibrium, while at other times it reaches a value in between. The two stable equilibria can in some sense be said to cause divergence in the stochastic trajectories. This behavior is made apparent in Figure 4. This behavior in the same way as in Figure 8.8 to make the difference more clear is illustrated. This is shown in Figure 4 together with a quantile plot which illuminates the difference in behavior compared to the result in Figure 8.9 compared to the deterministic one. By considering a case where there exist multiple stable equilibria the stochastic approach behaves rather differently. An interesting

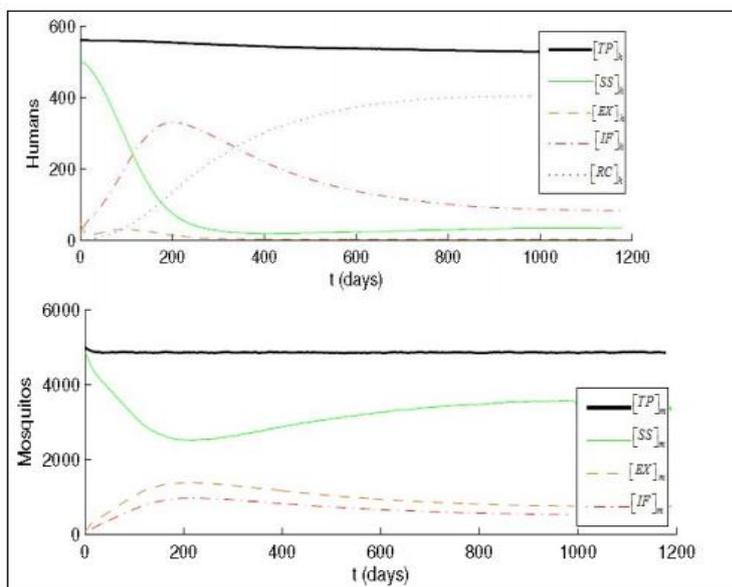


Fig. 4. The average of 100 stochastic trajectories of the dengue model (A) simulated by the Gillespie method with parameter values for an area of high transmission.

CONCLUSION

India has been one of the dengue endemic locales since old days. At present dengue is endemic in India in 23 states/Union Territories. Ngwa and Shu (2000) and Ngwa (2004) proposed a customary differential condition (ODE) compartmental model, a vulnerable uncovered irresistible recuperated powerless (SEIRS) design for people and a helpless uncovered irresistible (SEI) design for mosquitoes. In this work, Ngwa (2004) model has been considered for further advancement. Truth be told, the *Aedes aegypti* mosquitoes, the key vector for the spread of dengue, nibble people as well as winged creatures and creatures. In this theory, just human has been considered. A customary differential condition model called SPR_SODE has been made for the transmission of dengue. In this model a lot of four conditions for people and three conditions for mosquitoes has been made. Here, up to 17 parameters and 7 states have been characterized. The model has been additionally improved to partial amounts to take out figuring troubles. The model has been subjectively reached out to numerous locales with legitimate presumptions. Further, the model has been broke down. It has been indicated that there exists an area where the model is epidemiologically and numerically well-presented. The presence and uniqueness of a harmony point with no sickness (Disease free balance point) x has been demonstrated.

REFERENCES

- [1] Hu, Z., Teng, Z., & Zhang, L. (2014). Stability and bifurcation analysis in a discrete sir epidemic model. *Mathematics and Computers in Simulation*, 97, 80–93.
- [2] Hui, J., & Zhu, D.-m. (2007). Dynamics of seis epidemic models with varying population size. *International Journal of Bifurcation and Chaos*, 17 (05), 1513–1529.
- [3] Huo, H.-F., & Li, W.-T. (2004). Existence and global stability of periodic solutions of a discrete predator–prey system with delays. *Applied Mathematics and Computation*, 153 (2), 337–351.

- [4] Jeschke, J. M., Kopp, M., & Tollrian, R. (2002). Predator functional responses: discriminating between handling and digesting prey. *Ecological Monographs*, 72 (1), 95–112.
- [5] Jin, Z., & Ma, Z. (2006). The stability of an sir epidemic model with time delays. *Mathematical biosciences and engineering: MBE*, 3 (1), 101–109.
- [6] Jing, Z., & Yang, J. (2006). Bifurcation and chaos in discrete-time predator–prey system. *Chaos, Solitons & Fractals*, 27 (1), 259–277.
- [7] Jokinen, S., Osterlund, P., Julkunen, I., & Davidkin, I. (2007). Cellular immunity to mumps virus in young adults 21 years after measles-mumps-rubella vaccination. *The Journal of infectious diseases*, 196 (6), 861–867.
- [8] Kaddar, A., Abta, A., & Alaoui, H. (2010). Stability analysis in a delayed sir epidemic model with a saturated incidence rate. *Nonlinear Analysis: Modelling and Control*, 15 (3), 299–306.
- [9] Kang, H., & Fu, X. (2015). Epidemic spreading and global stability of an sis model with an infective vector on complex networks. *Communications in Nonlinear Science and Numerical Simulation*, 27 (1), 30–39.
- [10] Kaper, J., Rappuoli, R., & Buckley, M. (2005). Vaccine development: current status and future needs.
- [11] Khazeni, N., Hutton, D. W., Garber, A. M., Hupert, N., & Owens, D. K. (2009). Effectiveness and cost-effectiveness of vaccination against pandemic influenza (h1n1) 2009. *Annals of internal medicine*, 151 (12), 829–839.
- [12] Koch, R. (1876). The etiology of anthrax, based on the life history of bacillus anthracis. *Beitr. Biol. Pflanz*, 2, 277–310.
- [13] Kooij, R. E., & Zegeling, A. (1996). A predator–prey model with ivlev’s functional response. *Journal of Mathematical Analysis and Applications*, 198 (2), 473–489.
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