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RESEARCH ARTICLE

Mathematical Modeling and its Stability Analysis of an SEIR Model to Control Dengue by Segregating the Infective: An Approach for Efficient Resource Allocation

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Abstract

Objectives: Many research deals study on modeling the spread of dengue virus disease. The study dealing mathematical model to influence the concept of segregating infective is very important as it makes an organised practice of treatment which enlightens civilization. This article focuses on the same and frames a model with a proper analysis. **Methods:** BhirkoffRhota theorem helps in proving the boundedness of the model. By using the Next-Generation matrix the expression for Reproduction number (R_0) is determined. The Routh-Hurwitz stability criterion is used to reveal the Local Stability of the proposed model. The aid of Lyapunov-LaSalle's principle proves the Global Stability. **Findings:** The proposed model is found to be positive and also bounded. Moreover, the equilibrium points exists and stable locally and globally. The numerical simulation shows that $R_0 < 1$ at the disease-free equilibrium and $R_0 \geq 1$ at the endemic equilibrium proving the stability of the model numerically. The comparative analysis of the model results in revealing the fact that the proposed model controls the infective better than the usual model. **Novelty:** The article differs from the usual model by incorporating the idea of categorizing the infected population into 3 phase namely febrile, critical and convalescent concluding that the use of proposed model helps in effective treatment and in efficient management of hospital resources in a systematic way.

Keywords: Epidemic; Treatment Class; SEIR model; Jacobian matrix; Sensitivity index

1 Introduction

The dengue viral fever is an Aedes mosquito-borne disease and is a significant seasonal public health concern. The dense populated countries, stands at the forefront of vulnerability to this disease. The dengue virus, belonging to the Flaviviridae family, manifests in multiple serotypes, causing a spectrum of symptoms from mild fever to

severe complications, such as haemorrhagic fever and shock syndrome. The European Centre for Disease Prevention and Control[1] states, "In 2023, over six million dengue cases and over 6000 dengue-related deaths were reported from 92 countries/territories". Also, Tamil Nadu Health Minister said that the dengue cases are expected to raise due to the northeast monsoon. This might extend for the successive months, as there is a report that there is a lag in monsoon climate.

While usual approaches to epidemic dynamics focus on the outbreak transmission, this article approaches the complexity of the dengue virus's dynamics along with it focuses towards the hospital resource management. By integrating the concept of stratifying infected individuals into those exhibiting minimal and severe symptoms, the model endeavours to optimize the allocation of hospital resources, particularly for patients experiencing critical illness. Therefore, the proposed model not only seeks to solve the complexities of disease transmission but also promotes a strategic approach to allocate healthcare resources.

Current research analysing an optimal dengue model[2] and a regional study on dengue dynamics with potential control strategies[3] is notable and focus on controlling the outbreak with cost efficiency is inspiring. Particularly, the comprehensive 10-year study[4] of how various mathematical models signifies in controlling dengue providing a clear view of current models and techniques. Along with, there are studies constructing novel models to learn the effect of temperature[5] and the impact of climate change[6] in spreading dengue outbreak. Further, the study on cyclical patterns of dengue dynamics[7] contributed in understanding the recurrent behaviour of the disease.

The study of risk factors involved in a disease is necessary in order to analyse the outbreak[8]. Especially regarding dengue transmission, it is imperative to comprehend the dynamics of vector-borne diseases[9]. To address this need, a study conducted in Bangladesh[10] introduced a model that accounts for both host and vector population involved in dengue transmission. Similarly, research employing a two-staged structure model[11] within human population aids in elucidating the nuances of dengue transmission dynamics. Furthermore, the development of complex models has efficiently managed the risk of co-infection with COVID19 and dengue[12], which was faced during the period of 2020s. Notably, the utilization of fuzzy logic-based techniques[13] for dengue transmission has been surprising, demonstrating enhanced reliability in implementing control measures.

Therefore, it is imperative to develop mathematical models that incorporates control measures to prevent epidemic outbreaks. This entails analysing the impact of vaccination[14], management measures[15], incidence rates[16] and immunity basis[17]. It is notable that most of the analysis typically focuses on reducing infected individuals which fails in planning for hospital resource management during the outbreak. Hence, there is a critical need to study on constructing a model addressing on both the foresaid factors. Failure to do so could result insignificant drawback during outbreaks. Thus, it is time to be concerned after experiencing it once during the time of COVID-19.

Therefore, the article proposes a mathematical model that incorporates demographic considerations and taking into consideration and inspiring the ideology used in study[18]. Considering the infected individuals into different categories of infected individuals, the article commences with model formulation followed by the presentation of analytical results pertaining to positivity and boundedness. Subsequently, it demonstrates the existence of equilibrium points and derives the expression for R_0. Further, the article performs both local and global stability analyses, and examines the normalised sensitivity index of R_0. Finally, numerical simulations are provided in the results and discussion section to supplement findings. Overall, the article highlights the efficacy of the fact, considering categorized infected individuals facilitates in efficient treatment strategy.

2 Methodology

2.1 Mathematical Formulation

The following is the dynamics of the proposed SEIR model with segregated treatment classes with $S(0) = S_0 \geq 0$, $E(0) = E_0 \geq 0$, $I(0) = I_0 \geq 0$, $T_s(0) = T_{s0} \geq 0$, $T_h(0) = T_{h0} \geq 0$ and $R(0) = R_0 \geq 0$ as its initial conditions.

$$\begin{aligned}
\frac{dS}{dt} &= (1-m)\Lambda - \beta S(E+I) - \mu S + \grave{o}R \\
\frac{dE}{dt} &= \beta S(E+I) - (\gamma + \omega + \mu)E \\
\frac{dI}{dt} &= \gamma E - (\eta_1 + \eta_2 + \delta_1 + k_1 + \mu)I \\
\frac{dT_s}{dt} &= \eta_1 I - \delta_2 T_s - \mu T_s \\
\frac{dT_h}{dt} &= \eta_2 I - (\delta_3 + \mu)T_h \\
\frac{dR}{dt} &= m\Lambda + \omega E + \delta_1 I + \delta_2 T_s + \delta_3 T_h - (\mu + \grave{o})R
\end{aligned} \tag{1}$$

The proposed model is a modified SEIR model with treatment classes developed from general SEIR model. The treatment classes are $T_s(t)$ and $T_h(t)$ which resembles the individuals who take treatment at home and individuals who take medication at hospital respectively. Here, $T_s(t)$ contains infected individuals falls in the febrile phase, whereas $T_h(t)$ includes the infective who falls in critical phase. The other compartments are the usual classes namely Susceptible $S(t)$, Exposed $E(t)$, Infective $I(t)$ and Recovered $R(t)$. In the foresaid system of ordinary nonlinear differential equations, Λ represents the recruitment rate and μ represents the natural mortality rate respectively. The individuals who prevent themselves from Aedes aegypti mosquito are removed from the susceptible and move to recovered class with the rate m. Now, β is the transmission rate of dengue virus from susceptible population. The notations δ_1, δ_2, and δ_3 are the recovery rate of the infected class, Self-Treatment class and Hospitalised treatment class. Also, ω is the recovery rate of the exposed individual due to self-immune system. The rate at which the exposed individuals move to infective class is given by γ. The notation η_1 is the rate at which infected individuals takes self-treatment whereas η_2 is the rate at which infective take hospitalised treatment. Here, $\eta_1 I$ are the individuals who are at the initial stage of infection and $\eta_2 I$ are the individuals infected severely. k_1 is the diseased caused mortality rate. Finally, ò is the rate at which recovered individual move to the susceptible category. The proposed model highlights the ideology of managing the hospital resource for the prior infective and also helps to understand the spreading nature of dengue disease.

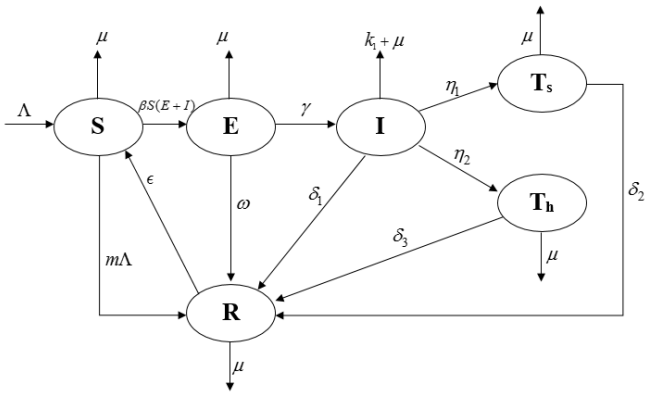

Fig 1. Transmission diagram of the proposed model

2.2 Analytical Results

This section provides prerequisite lemmas and other sufficient analysis to determine the equilibrium points and its stability.

Lemma 1. The solution set of the proposed model **(1)** along with the initial conditions remains non-negative for all $t > 0$.

Proof.

Consider

$$\begin{aligned} \frac{dS}{dt} &= (1-m)\Lambda - \beta S(E+I) - \mu S + oR \\ &\geq -(\beta(E+I)+\mu)S \\ \frac{dS}{S} &\geq -(\beta(E+I)+\mu)dt \end{aligned}$$

Integrating and simplifying, we get

$$logS \geq -(\beta(E+I)+\mu)t + logC$$

$$log\frac{S}{C} \geq -(\beta(E+I)+\mu)t$$

Taking exponentials, we get

$$S(t) \geq S(0)e^{-(\beta(E+I)+\mu)t}$$

As $t \to \infty, S(t) \geq 0$

Similarly, we can solve for $E(t) \geq 0,\ I(t) \geq 0,\ T_s(t) \geq 0,\ T_h(t) \geq 0$ and $R(t) \geq 0$.

Thus, the solution set of the dengue model remains positive for all $t > 0$.

As the study deals with dynamics of the dengue disease in human population, the above proof of positivity is necessary to show the non-negativity of the solutions of the proposed model.

Lemma 2. The region given by $\Phi = \left\{(S(t),\ E(t),\ I(t),\ T_s(t),\ T_h(t),\ R(t)) \in {}^{6}_{+} : N(t) \leq \frac{\Lambda}{\mu}\right\}$ contains the solution set of the dengue model (Equation (1)) with the initial conditions.

Proof.

Summing up each equation in the model (Equation (1)) gives

$$\begin{aligned}\frac{dN(t)}{dt} &= \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dT_s(t)}{dt} + \frac{dT_h(t)}{dt} + \frac{dR(t)}{dt} \\ &= \Lambda - \mu\left(S + E + I + T_s + T_h + R\right) - k_1 I \\ &\leq \Lambda - \mu N\end{aligned}$$

Thus, the solution of the above equation is given by$N(t) \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda}{\mu} - N_0\right) e^{-\mu t}$

where N_0 is the initial population. By the Bhirkoff-Rhota theorem [19], it is obtained as $N_0 \leq \frac{\Lambda}{\mu}$

As $t \to \infty, N(t) \to \frac{\Lambda}{\mu}$.

Thus, the feasible solution of the dengue model (Equation (1)) is well-posed epidemiologically in Φ and hence it is enough we analyse within Φ.

2.3 Existence of Equilibrium points

Initially, the disease-free equilibrium (DFE) is considered as below,

$E^0 = \left(S^0,\ E^0,\ I^0,\ T_s{}^0,\ T_h^0,\ R^0\right) = \left(\frac{(1-m)\Lambda}{\mu},\ 0,\ 0,\ 0,\ 0,\ 0\right)$.

Now to determine the endemic equilibrium (EE), all the equation of model (Equation (1)) is considered and equating each of it to zero, we get

$$(1-m)\Lambda - \beta S(E+I) - \mu S + \grave{o}R = 0 \tag{2}$$

$$\beta S(E+I) - (\gamma + \omega + \mu)E = 0 \tag{3}$$

$$\gamma E - (\eta_1 + \eta_2 + \delta_1 + k_1 + \mu)I = 0 \tag{4}$$

$$\eta_1 I - \delta_2 T_s - \mu T_s = 0 \tag{5}$$

$$\eta_2 I - (\delta_3 + \mu)T_h = 0 \tag{6}$$

$$m\Lambda + \omega E + \delta_1 I + \delta_2 T_s + \delta_3 T_h - (\mu + \grave{o})R = 0 \tag{7}$$

Now, solving (Equation (4)), we get

$$T_s^* = \frac{\eta_1}{\delta_2 + \mu} I^*$$

Similarly, solving for other values, we get

$$E^* = \frac{(\eta_1 + \eta_2 + \delta_1 + k_1 + \mu)}{\gamma} I^*$$

$$T_h^* = \frac{\eta_2}{\delta_3+\mu} I^*$$

$$R^* = \frac{m\Lambda + B_1 I^*}{(\grave{o}+\mu)}$$

$$S^* = \frac{(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)}{\beta(\eta_1+\eta_2+\delta_1+k_1+\mu+\gamma)} \tag{8}$$

$$I^* = \frac{\gamma\beta B_4(B_5+m\Lambda\grave{o}) - \mu\gamma\beta_2\beta_3(\grave{o}+\mu)}{\beta B_2 B_3 B_4(\grave{o}+\mu) - \gamma\grave{o}\beta B_1 B_4}$$

where $B_1 = \left(\frac{\omega(\eta_1+\eta_2+\delta_1+k_1+\mu)}{\gamma} + \frac{\delta_1(\delta_2+\mu)(\delta_3+\mu)+\delta_2\eta_1(\delta_3+\mu)+\delta_3\eta_2(\delta_2+\mu)}{(\delta_2+\mu)(\delta_3+\mu)}\right)$, $B_2 = (\gamma+\omega+\mu)$, $B_3 = \eta_1+\eta_2+\delta_1+k_1+\mu$, $B_4 = B_3+\gamma$ and $B_5 = \Lambda(1-m)(\grave{o}+\mu)$.

2.4 Determination of Reproduction number

Reproduction number (denoted as R_0) is a ratio which signifies the contagiousness of an infection. In other words, the R_0 shows the nature of spread of the disease and helps to confirm whether it is a pandemic or not [20]. Clearly, if $R_0 < 1$, then the dengue virus spread can be controlled and if $R_0 > 1$, then it becomes an epidemic.

The reproduction number of the proposed model is obtained by determining the Jacobian matrix of (Equation (1)).

$$J(E,I,T_s,T_h) = \begin{pmatrix} \beta S-(\gamma+\omega+\mu) & \beta S & 0 & 0 \\ \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 \\ 0 & \eta_1 & -(\delta_2+\mu) & 0 \\ 0 & \eta_2 & 0 & -(\delta_3+\mu) \end{pmatrix}$$

Clearly, the transmission matrix F and the transition matrix V are segregated as below.

$$F = \begin{pmatrix} \beta S & \beta S & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \; V = \begin{pmatrix} -(\gamma+\omega+\mu) & 0 & 0 & 0 \\ \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 \\ 0 & \eta_1 & -(\delta_2+\mu) & 0 \\ 0 & \eta_2 & 0 & -(\delta_3+\mu) \end{pmatrix}$$

In order to determine the expression for basic reproduction number it is necessary to find $\rho\left(FV^{-1}\right)$.

Thus, the inverse of the matrix V is obtained as follows,

$$V^{-1} = \begin{pmatrix} \frac{-1}{(\gamma+\omega+\mu)} & 0 & 0 & 0 \\ \frac{\gamma}{(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)} & \frac{-1}{(\eta_1+\eta_2+\delta_1+k_1+\mu)} & 0 & 0 \\ \frac{-\gamma\eta_1}{(\delta_3+\mu)(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)} & 0 & \frac{-1}{(\delta_2+\mu)} & 0 \\ \frac{-\gamma\eta_1}{(\delta_2+\mu)(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)} & 0 & 0 & \frac{-1}{(\delta_3+\mu)} \end{pmatrix}$$

Now,

$$FV^{-1} = \begin{pmatrix} \frac{\beta S\gamma}{(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)} - \frac{\beta S}{(\gamma+\omega+\mu)} & \frac{-\beta S}{(\eta_1+\eta_2+\delta_1+k_1+\mu)} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

The maximum absolute eigenvalue of the above matrix gives the basic reproduction number of the model.

Hence,

$$R_0 = \frac{\Lambda\beta(1-m)}{\mu(\gamma+\omega+\mu)}\left[\frac{\gamma}{(\eta_1+\eta_2+\delta_1+k_1+\mu)}+1\right] \tag{9}$$

2.5 Local Stability Analysis

This section reveals the stability of the proposed model at both the equilibrium point namely disease-free equilibrium and endemic equilibrium.

Theorem 3. The system (Equation (1)) is locally asymptotically stable at disease-free equilibrium provided only if $R_0 < 1$ and unstable otherwise.

Proof. The proof begins with determining the Jacobian matrix of the system (Equation (1)) as below,

$$J(M) = \begin{pmatrix} -[\beta(E+I)+\mu] & -\beta S & -\beta S & 0 & 0 & \grave{o} \\ \beta(E+I) & \beta S-(\gamma+\omega+\mu) & \beta S & 0 & 0 & 0 \\ 0 & \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\delta_2+\mu) & 0 & 0 \\ 0 & 0 & \eta_2 & 0 & -(\delta_3+\mu) & 0 \\ 0 & \omega & \delta_1 & \delta_2 & \delta_3 & -(\grave{o}+\mu) \end{pmatrix}$$

At $E^0 = \left(S^0,\, E^0,\, I^0,\, T_s^0,\, T_h^0,\, R^0\right) = \left(\frac{(1-m)\Lambda}{\mu},\, 0,\, 0,\, 0,\, 0,\, 0\right)$ the above matrix becomes

$$J(M_1) = \begin{pmatrix} -\mu & -\frac{\beta\Lambda(1-m)}{\mu} & -\frac{\beta\Lambda(1-m)}{\mu} & 0 & 0 & \grave{o} \\ 0 & \frac{\beta\Lambda(1-m)}{\mu}-(\gamma+\omega+\mu) & \frac{\beta\Lambda(1-m)}{\mu} & 0 & 0 & 0 \\ 0 & \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\delta_2+\mu) & 0 & 0 \\ 0 & 0 & \eta_2 & 0 & -(\delta_3+\mu) & 0 \\ 0 & \omega & \delta_1 & \delta_2 & \delta_3 & -(\grave{o}+\mu) \end{pmatrix}$$

Clearly, $\lambda_1 = -\mu, \lambda_2 = -(\delta_2+\mu), \lambda_3 = -(\delta_3+\mu)$ and $\lambda_4 = -(\grave{o}+\mu)$ are negative eigenvalues.

For the system to be stable it is enough the other two eigenvalues are negative. The other two eigenvalues are obtained by solving the below quadratic equations.

$a\lambda^2 + b\lambda + c = 0$where $a = 1, b = \left(\gamma+\omega+\mu+\eta_1+\eta_2+\delta_1+k_1+\mu-\frac{\Lambda\beta(1-m)}{\mu}\right)$ and

$$c = \left((\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu) - \left(\frac{\Lambda\beta(1-m)\gamma}{\mu} + \frac{\Lambda\beta(1-m)(\eta_1+\eta_2+\delta_1+k_1+\mu)}{\mu}\right)\right)$$

Solving for $\Delta = b^2 - 4ac$, we get

$$\begin{aligned} \Delta &= b^2 - 4a \times \left((\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu) - \left(\tfrac{\Lambda\beta(1-m)\gamma}{\mu} + \tfrac{\Lambda\beta(1-m)(\eta_1+\eta_2+\delta_1+k_1+\mu)}{\mu}\right)\right) \\ &= b^2 + 4a \times \left((\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)\left(\tfrac{\Lambda\beta(1-m)\gamma}{\mu(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)} + \tfrac{\Lambda\beta(1-m)}{\mu(\gamma+\omega+\mu)} - 1\right)\right) \\ &= b^2 + 4a \times ((\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)(R_0-1)) \end{aligned}$$

Thus, it is obvious that the two eigenvalues are negative only if $R_0 < 1$.

Hence, if $R_0 < 1$ the system (Equation (1)) is stable at the DFE.

Theorem 4. The system (Equation (1)) is locally asymptotically stable at the endemic equilibrium only if $R_0 > 1$.

Proof. The Jacobian matrix of system (Equation (1)) is given as

$$J(M) = \begin{pmatrix} -[\beta(E+I)+\mu] & -\beta S & -\beta S & 0 & 0 & \grave{o} \\ \beta(E+I) & \beta S-(\gamma+\omega+\mu) & \beta S & 0 & 0 & 0 \\ 0 & \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\delta_2+\mu) & 0 & 0 \\ 0 & 0 & \eta_2 & 0 & -(\delta_3+\mu) & 0 \\ 0 & \omega & \delta_1 & \delta_2 & \delta_3 & -(o+\mu) \end{pmatrix}$$

At the endemic equilibrium the matrix becomes

$$J(M_2)=\begin{pmatrix} -(\beta(E^*+I^*)+\mu] & -\beta S^* & -\beta S^* & 0 & 0 & \grave{o} \\ \beta(E^*+I^*) & \beta S^*-(\gamma+\omega+\mu) & \beta S^* & 0 & 0 & 0 \\ 0 & \gamma & -(\eta_1+\eta_2+\delta_1+k_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \eta_1 & -(\delta_2+\mu) & 0 & 0 \\ 0 & 0 & \eta_2 & 0 & -(\delta_3+\mu) & 0 \\ 0 & \omega & \delta_1 & \delta_2 & \delta_3 & -(\grave{o}+\mu) \end{pmatrix}$$

From the above matrix it is clear that $\lambda_1=-(\grave{o}+\mu), \lambda_2=-(\delta_3+\mu)$ and $\lambda_3=-(\delta_2+\mu)$ are negative eigenvalues.

To show the system is stable it is enough we prove the below cubic equation is stable.

$$\lambda^3+(D_1+D_3-D_2)\lambda^2+\left(\beta^2S^*(E^*+I^*)+D_1D_3-D_1D_2-D_2D_3-\gamma\beta S^*\right)\lambda \\ +\left(D_3\beta^2S^*(E^*+I^*)+\gamma\beta^2S^*(E^*+I^*)-D_1D_2D_3-D_1\gamma\beta S^*\right)=0$$

Where,$D_1=\beta(E^*+I^*)+\mu, D_2=\beta S^*-(\gamma+\omega+\mu)$ and $D_3=(\eta_1+\eta_2+\delta_1+k_1+\mu)$

Also, we have $a_0=\left(D_3\beta^2S^*(E^*+I^*)+\gamma\beta^2S^*(E^*+I^*)-D_1D_2D_3-D_1\gamma\beta S^*\right)$,

$a_1=\left(\beta^2S^*(E^*+I^*)+D_1D_3-D_1D_2-D_2D_3-\gamma\beta S^*\right)$ and $a_2=(D_1+D_3-D_2)$.

By Routh-Hurwitz stability criterion we need to show that $a_2a_1>a_0$ and a_0, a_1 and a_2 are all positive.

Clearly a_0, a_1 and a_2 are all positive.

Solving for $a_2a_1>a_0$, it is observed that a_0 is contained in $a_2\times a_1$ showing that $a_2\times a_1$ is greater than a_0 for $R_0>1$.

Thus, system (Equation (1)) is stable for $R_0>1$ at the EE.

2.6 Global Stability Analysis

In this section the global stability of system (Equation (1)) at disease-free equilibrium and endemic equilibrium is determined.

Theorem 5. When $R_0<1$, the disease-free equilibrium E^0 is globally stable whereas it is unstable when $R_0>1$.

Proof. To prove the global stability of E^0 a Lyapunov function is defined as below

$$L(t)=C_1E+C_2I$$

Now taking the derivative of L, we get

$$\begin{aligned} &\tfrac{dL(t)}{dt}=C_1\tfrac{dE}{dt}+C_2\tfrac{dI}{dt} \\ &=C_1[\beta S(E+I)-(\gamma+\omega+\mu)E]+C_2[\gamma E+(\eta_1+\eta_2+\delta_1k_1+\mu)I] \\ &=[C_1\beta S-C_1(\gamma+\omega+\mu)+C_2\gamma]E+[C_1\beta S+C_2(\eta_1+\eta_2+\delta_1+k_1+\mu)]I \end{aligned}$$

Considering $C_1=\gamma$ and $C_2=\beta S$ and solving the above equation becomes

$$\tfrac{dL(t)}{dt}=-\gamma(\gamma+\omega+\mu)E+(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)\left[\tfrac{\gamma\beta S}{(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)}+\tfrac{\beta S}{(\gamma+\omega+\mu)}-1+1\right]I$$

$$=-\gamma(\gamma+\omega+\mu)E+(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)[R_0-1]I+(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)I$$

Hence, $\tfrac{dL(t)}{dt}\le(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)[R_0-1]I$ if and only if $R_0<1$.

Clearly, $\tfrac{dL(t)}{dt}=0$ only if $I=0$.

The Lyapunov-LaSalle's principle states that the system (Equation (1)) is globally asymptotically stable at DFE if $R_0<1$.

Theorem 6. The endemic equilibrium E^* is globally stable if $R_0>1$.

Proof. Let us consider the following Lyapunov function

$$L=\frac{1}{2}l_1(S-S^*)^2+\frac{1}{2}l_2(E-E^*)E'+\frac{1}{2}l_3(I-I^*)I'+\frac{1}{2}l_4(T_s-T_s^*)T_s'+\frac{1}{2}l_5(T_h-T_h^*)T_h'+\frac{1}{2}l_6(R-R^*)R'$$

Differentiating with respect to ' t ', we get

$$\begin{aligned} L^{'} = \quad & l_1 (S-S^*) S^{'} + l_2 (E-E^*) E^{'} + l_3 (I-I^*) I^{'} + l_4 (T_s - T_s^*) T_s{}^{'} + l_5 \left(T_h - T_h^*\right) T_h{}^{'} + l_6 (R-R^*) R^{'} \\ = \quad & -l_1[\beta(E+I)+\mu](S-S^*)^2 + l_2[\beta S - (\gamma+\omega+\mu)](E-E^*)^2 - l_3(\eta_1+\eta_2+\delta_1+k_1+\mu)(I-I^*)^2 \\ & -l_4(\delta_2+\mu)(T_s-T_s^*)^2 - l_5(\delta_3+\mu)\left(T_h-T_h^*\right)^2 - l_6(o+\mu)(R-R^*)^2 - l_1\beta S(S-S^*)(E-E^*) \\ & -l_1\beta S(S-S^*)(I-I^*) - l_1 \grave{o}(S-S^*)(R-R^*) + l_2\beta(E+I)(S-S^*)(E-E^*) \\ & +l_2\beta S(E-E^*)(I-I^*) + l_3\gamma(E-E^*)(I-I^*) + l_4\eta_1(I-I^*)(T_s-T_s^*) + l_5\eta_2(I-I^*)\left(T_h-T_h^*\right) \\ & +l_6\omega(E-E^*)(R-R^*) + l_6\delta_1(I-I^*)(R-R^*) + l_6\delta_2(T_s-T_s^*)(R-R^*) + l_6\delta_3\left(T_h-T_h^*\right)(R-R^*) \end{aligned}$$

Thus, L' must be negative to conclude the hypothesis.

Hence, the condition for the global stability of the endemic equilibrium is determined as

$$\begin{gathered} a_{12}^2 < \tfrac{a_{11}a_{22}}{5},\ a_{13}^2 < \tfrac{a_{11}a_{33}}{6},\ a_{16}^2 < \tfrac{a_{11}a_{66}}{5},\ a_{21}^2 < \tfrac{a_{22}a_{11}}{5},\ a_{23}^2 < \tfrac{2a_{22}a_{33}}{15},\ a_{32}^2 < \tfrac{2a_{33}a_{22}}{15} \\ a_{43}^2 < \tfrac{a_{44}a_{33}}{3},\ a_{53}^2 < \tfrac{a_{55}a_{33}}{5},\ a_{62}^2 < \tfrac{4a_{66}a_{22}}{25},\ a_{63}^2 < \tfrac{2a_{66}a_{33}}{15},\ a_{64}^2 < \tfrac{2a_{66}a_{44}}{5},\ a_{65}^2 < \tfrac{2a_{66}a_{55}}{5} \end{gathered}$$

This can be written as follows

(i) $l_1(\beta S)^2 < \frac{l_2}{5}\mu(\gamma+\omega+\mu)$

(ii) $l_1(\beta S)^2 < \frac{l_3}{6}\mu(\eta_1+\eta_2+\delta_1+k_1+\mu)$

(iii) $l_1 \grave{o}^2 < \frac{l_6}{5}\mu(\grave{o}+\mu)$

(iv) $l_2[\beta(E+I)]^2 < \frac{l_1}{5}\mu(\gamma+\omega+\mu)$

(v) $l_2(\beta S)^2 < \frac{2l_3}{15}(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)$

(vi) $l_3\gamma^2 < \frac{-2l_2}{15}(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)$

(vii) $l_4\eta_1^2 < \frac{l_3}{3}(\eta_1+\eta_2+\delta_1+k_1+\mu)(\delta_2+\mu)$

(viii) $l_5\eta_2{}^2 < \frac{l_3}{3}(\eta_1+\eta_2+\delta_1+k_1+\mu)(\delta_3+\mu)$(ix) $l_6\omega^2 < \frac{4l_2}{25}(\gamma+\omega+\mu)(\grave{o}+\mu)$

(x) $l_6\delta_1^2 < \frac{2l_3}{15}(\grave{o}+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)$

(xi) $l_6\delta_2^2 < \frac{2l_4}{5}(o+\mu)(\delta_2+\mu)$

(xii) $l_6\delta_3^2 < \frac{2l_5}{5}(o+\mu)(\delta_3+\mu)$

Considering $l_2 = l_3 = 1$, $L^{'}$ will be negative. Clearly, the inequality (v) and (vi) holds only when $R_0 \geq 1$. Hence, the endemic equilibrium is globally asymptotically stable if $R_0 \geq 1$.

2.7 Sensitivity Analysis

The normalized sensitivity index, in the context of epidemiology is a measure that quantifies the sensitivity of the basic reproduction number (R_0) to make changes in specific parameters of an infectious disease model.

The sensitivity index is often used to assess the impact of changes in model parameters on the basic reproduction number which helps researchers and public health practitioners to understand which parameters have the most significant influence on the potential for disease spread. The normalized forward sensitivity index of R_0 with respect to the parameter say θ is given by $\Gamma_\theta^{R_0} = \frac{\partial R_0}{\partial \theta} \times \frac{\theta}{R_0}$.

The expression for the reproduction number is $R_0 = \frac{\Lambda\beta(1-m)[\gamma+(\eta_1+\eta_2+\delta_1+k_1+\mu)]}{\mu(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)}$ which is obtained from Equation (9).

By the definition of normalised forward sensitivity index of R_0, the sensitivity indices can be simulated as given below

$$\begin{aligned} \Gamma_\beta^{R_0} \quad &= \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} \\ &= +1 \end{aligned}$$

Similarly, the sensitivity index of other parameters is obtained as below

$$\Gamma_{\Lambda}^{R_0}=+1,\ \Gamma_{\eta_1}^{R_0}=\frac{-\gamma\eta_1}{(\eta_1+\eta_2+\delta_1+k_1+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)}$$
$$\Gamma_{\eta_2}^{R_0}=\frac{-\gamma\eta_2}{(\eta_1+\eta_2+\delta_1+k_1+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)}$$
$$\Gamma_{\delta_1}^{R_0}=\frac{-\gamma\delta_1}{(\eta_1+\eta_2+\delta_1+k_1+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)}$$
$$\Gamma_{k_1}^{R_0}=\frac{-\gamma k_1}{(\eta_1+\eta_2+\delta_1+k_1+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)},$$
$$\Gamma_{\gamma}^{R_0}=\frac{\gamma[\omega-(\eta_1+\eta_2+\delta_1+k_1)]}{(\gamma+\omega+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)},\ \Gamma_{\omega}^{R_0}=\frac{-\omega}{(\gamma+\omega+\mu)}$$
$$\Gamma_{\mu}^{R_0}=\frac{-\left\{\gamma\left[(\gamma+\omega)(\eta_1+\eta_2+\delta_1+k_1+2\mu)+2\mu(\eta_1+\eta_2\delta_1+k_1)+3\mu^2\right]+(\gamma+\omega+2\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)\right\}}{(\gamma+\omega+\mu)(\eta_1+\eta_2+\delta_1+k_1+\mu)(\gamma+\eta_1+\eta_2+\delta_1+k_1+\mu)}$$

The numerical value of each expression is discussed in the following section.

3 Results and Discussion

In this section, the justification of theoretical analysis of the proposed model is performed by numerical simulations.

Considering the parametric values given in Table 1, the simulation is carried out.

Table 1. Parametric values for the proposed model

Parameter	Value	Source	Parameter	Value	Source
Λ	2	Estimated	δ_1	1/7	[21]
m	0.8	[21]	δ_2	1/7	[21]
β	0.0046	Estimated	δ_3	1/7	[21]
γ	1/3	[21]	k_1	0.012	Estimated
ω	1/7	Assumed	μ	1/70.42	[22]
η_1	0.8	Assumed	o	1/14	[21]
η_2	0.4	Assumed			

Using, the values in Table 1, along with the initial conditions $S_0=1000,\ E_0=6,\ I_0=4,\ T_{S_0}=0,\ T_{h_0}=0$ and $R_0=0$, the dynamics of the proposed model is plotted and given below.

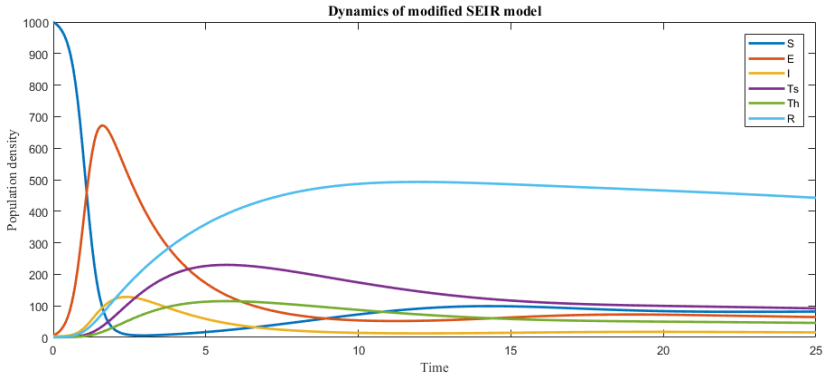

Fig 2. The Dynamic of the Proposed SEIR model

Also, the dynamics of the infected curve for different values of $\eta_1=0.1,\ 0.2,\ 0.4,\ 0.8$ is obtained as shown in Figure 3.

Figures 4, 5, 6 and 7 gives the dynamics of $I,\ T_s$ and T_h compartment by varying the values of $\eta_1,\ \eta_2$ and m correspondingly.

In addition, the infective class of the proposed model and the regular SEIR model with treatment[23] is compared and given in Figure 8.

From Figure 8, it is observed that the maximum possible infective individual of the proposed model is much controlled than the regular treatment model showing the proposed model is optimal.

For suppose considering the proposed model by neglecting the idea of segregated treatment approach and comparing it with the proposed model, the dynamics would be as given in Figure 9.

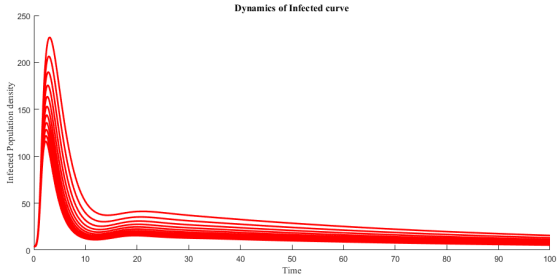

Fig 3. Dynamics of Infected population

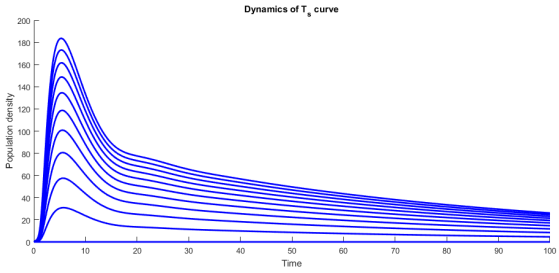

Fig 4. Treatment class for different values η_1

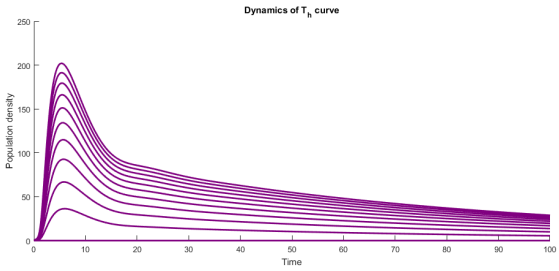

Fig 5. Treatment class for different values of η_2

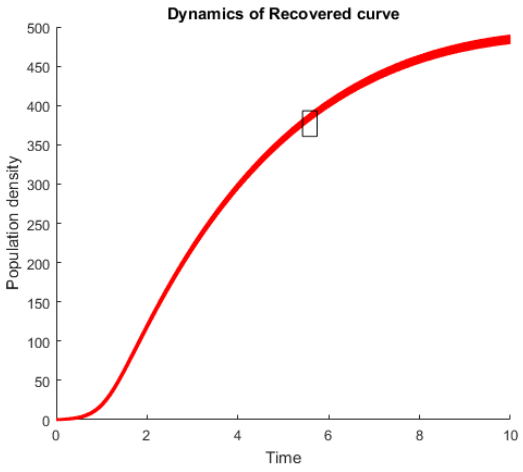

Fig 6. Dynamics of Recovered class for various m

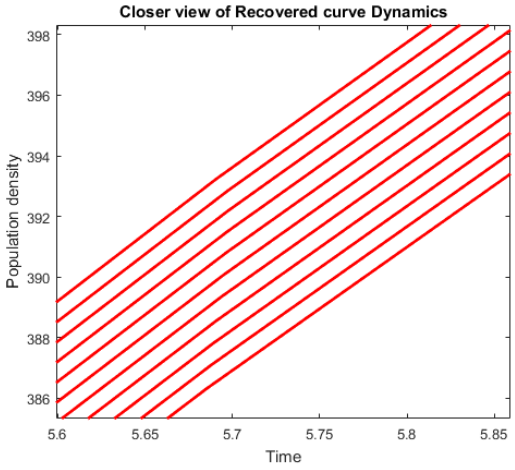

Fig 7. Selected portion of Figure 6

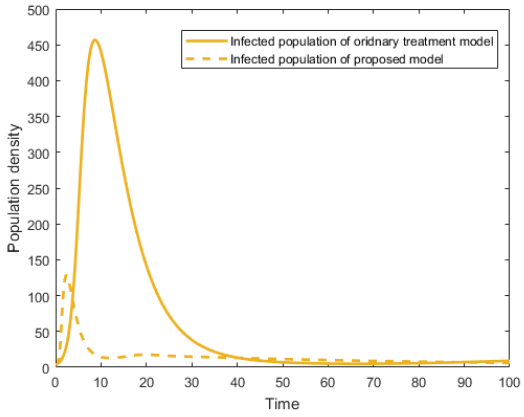

Fig 8. Comparative curve of the Infective classes

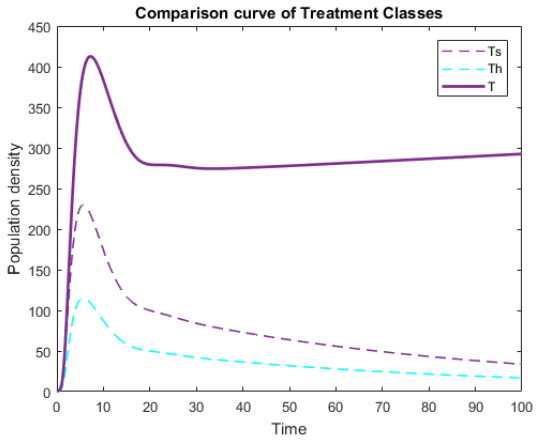

Fig 9. Comparison on Dynamics of Treatment classes

It is observed that the population density of a single treatment class would be greater than the proposed model.

Also, the numerical analysis of the Reproduction number is obtained as follows:

(i) At DFE: $R_0 = 0.3571 < 1$

(ii) At EE: $R_0 \geq 1$

It is observed that the R_0 value is good enough to control impact in spreading the outbreak. Thus, the proposed model is much enriched than the usual model.

The simulation of the proposed model concludes with the numerical analysis of the sensitivity index. The normalised forward sensitivity index of the basic reproduction number[24] of the model can be represented geometrically as shown in Figure 10.

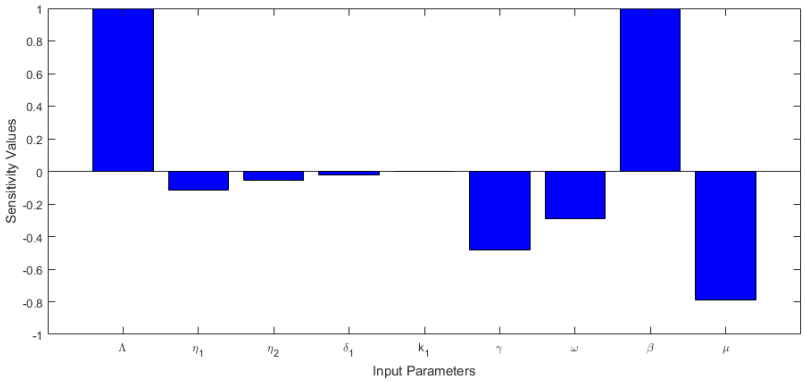

Fig 10. Normalised Forward Sensitivity Index of R_0

The analysis on sensitivity indices, provides information about the system dynamics. The value of Λ and β emphasises its substantial influence and shows that the model structure takes this into account carefully. Similarly, the negative values of η_1, η_2 and δ_1 highlights the need for mitigation of negative impacts on reproduction number. Also, the values of γ, ω and μ show their significant influence. Thus, by ranking interventions based on the sensitivity indices the system resilience and efficiency can be improved.

In summary, the numerical analysis begins with the representation of dynamics of the model which shows an inclined recovery curve. Further, the maximum infected population is also very less and declines within a short time, showing the disease can be controlled soon. Figure 3 shows that for increase of η_1 value the maximum number of infective is reduced. Also, Figures 4 and 5 gives how the effect of varying η_1 and η_2 values create impact in T_s and T_h compartment respectively. In order to highlight the novelty of the proposed model, Figures 6 and 7 reveals that the impact of immunology is not effectively increasing the recovered population. A very minute change in the recovery curve is observed. Also, Figure 8 proves the optimality of the proposed model revealing the fact that the maximum number of infected individuals is nearly beyond 450 individuals for the general treatment model, whereas the proposed model ensures to maintain less number of possible infected cases which is about 140 individuals. Further, the proposed model ensures an effective management of hospital resource for the infected individuals which is depicted in Figure 9. Overall, the numerical analysis enlightens the concept of segregating infected individuals can reduce the infected individuals and ensures an efficient treatment for the infective.

4 Conclusion

The mathematical analysis performed in this article provides a substantial proof that the proposed model is optimistic and efficient supported by numerical simulation. Thus, by categorizing the infective and segregating the treatment class into two, the frontline workers can give prior to the critical cases. From Figure 2, it is observed that the dynamical curve of the proposed model reveals that the recovery rate elevates and sustain with an adequate value. Also, it is observed that the infective curve is scant and hence the infected individuals is meagre in population. Further, Figures 3, 4 and 5 ensures the impact of η_1 and η_2 plays a major role in reducing the number of infective. Figures 6 and 7 reveals the impact of η_1 and η_2 is great, besides the role of immunity in the model. Further, Figures 8 and 9 justifies that segregation treatment methodology helps in controlled infective and efficient treatment allocation. The analysis on R_0 proves its stability, as $R_0 = 0.3571 < 1$ at DFE and at EE $R_0 \geq 1$ embracing the actual situation of the dengue infection. Finally, the sensitivity analysis identified the key parameters in preventing the spread of outbreak. Evidencing these observations, the proposed model strongly indicates it is optimal in reducing the infective and

helps in efficient hospital resource management.

Therefore, the proposed model aims in reducing the number of infective and the number of individuals taking treatment at hospital signifying the readers, how it helps in resource management and in organized treatment for infective during the outbreak. Beyond this, the model also helps to predict the expected number of infected cases in future which would be helpful to be prepared for it. Hence, the proposed model helps in case study of the outbreak in a region and be aware of the outbreak. However, there is a possibility of defect in proposed model in defining the parametric value for segregating the infective. So, the proposed model can be developed to rectify this issue as future research which would be more reliable. Also, incorporating various incidence rates, transforming the model into partial differential equations and even use of other methods in the numerical simulation can be performed in future works for testing the efficiency of model.

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