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A Mathematical Model for the Dengue Fever Epidemic with Vaccination and Treatment

Fatima Alnoor[1,*], Hajir Wahbi[2,*], Fatma Saadi[2], Rabeea M.A. Daoub[2]

[1] *Department of Mathematics, College of Science, Sudan University of Science and Technology, Khartoum, Sudan*
[2] *Department of Chemistry , College of Sciences and Arts , Northern Border University, Arar, Saudi Arabia*

Abstract. The aim of this study is to construct and investigate a mathematical model to grasp the dynamics of dengue fever and assess the consequences of vaccine and treatment approaches in disease control. We created a mathematical model, including human and mosquito populations, using impulsive differential equations to simulate vaccination and antiviral treatment interventions. We used the stability analysis of equilibrium points to find the behavior of the system in different situations. We used MATLAB to run numerical simulations evaluating several intervention possibilities. Particularly when started early on, immunization programs dramatically lower dengue transmission rates. In addition, combining vaccination with antiviral medications increases the efficacy of intervention campaigns, therefore accelerating the decrease in disease prevalence and improving long-term control of epidemics. Effective control of dengue depends on integrated programs combining immunization and antiviral therapies. Reducing the public health load of dengue fever and stopping transmission depend on early intervention and constant efforts.

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1. Introduction

Dengue is a febrile illness that can be fatal in severe cases. A flavivirus, including four distinct serotypes (DV-1, DV-2, DV-3, and DV-4) [1][2], is responsible for its etiology. Mosquito vectors of the Aedes genus, particularly Aedes aegypti and Aedes albopictus, spread the virus to humans. Two, we still do not fully understand how severe dengue infections [3][4], such as dengue hemorrhagic fever and dengue shock syndrome, affect the body. Many people agree that the immune system, genetics, and pathogen's ability to infect cells all play a role in how quickly some patients get worse. Dengue is increasing and

*Corresponding author.
*Corresponding author.
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Email addresses: fattoshaa1988@gmail.com (F.Alnoor), Hajir.Wahbi@nbu.edu.sa (H. Wahbi),fatma.saadi@nbu.edu.sa (F. Saadi),Rabeea.Ali@nbu.edu.sa Rabeea M.A. Daoub

the disease poses a considerable public health challenge in tropical areas. Seasonal climate fluctuations intimately link dengue epidemics, with repeated breakout waves following each rainy season. An outbreak can affect thousands of people [5] [6]. This brings us to point number five [1]. Most people recover from a mild febrile illness; however, a small but significant number progress to dengue shock syndrome, which is associated with mortality. This results in a significant rate of fatal cases in many affected areas, predominantly affecting young children and people of working age. There are three main problems with controlling dengue infections: there are four different virus serotypes, and each one can cause severe illness; we don't fully understand how the disease works; and there isn't a specific treatment or vaccine for protection[7]. Controlling the vector population further complicates the situation [2][8].

Malaria is caused by a parasite that is found mainly in rural areas and is spread by mosquito bites at night. In contrast, dengue is spread by any of the four serotypes of Aedes mosquitoes[9]. The two identified species of vector-transmitting dengue are Aedes aegypti and Aedes albopictus [10]. The former is extremely anthropophagic, flourishing in densely populated urban environments and predominantly biting during daylight hours; the latter is less anthropophagic and resides in rural regions. Thus, the significance of dengue is dual.(1) The disease causes a lot of problems for society and the economy, even when it doesn't have any variants that are lethal. This is because it spreads so widely and has many types (absenteeism, immobility, debilitation, medicine). (2) The chance of getting the hemorrhagic form and dengue shock syndrome, which can be very dangerous, cost a lot of money, and even kill people. An endemic equilibrium is a steady state where a disease remains prevalent in a community over time, where the number of new vulnerable people entering the community equals the number of new diseases [11]. This balance allows people to improve or build immunity. Measles typically

This equilibrium is evident in communities with low or uneven vaccination rates, as dengue is highly contagious. Dengue, on the other hand, requires a mixed approach of public health campaigns, vector control, and community education on environmental management [12].

Many studies have looked at how dengue fever spreads and different ways to control it. They also examined how the immune system responds, including memory effects. Tang et al. (2022) [13] investigated the effects of vaccination and reinfection on the transmission of dengue fever. They emphasized the importance of these factors in the progression of the disease. Dwivedi et al. (2022) [14] proposed an optimal vaccine method for the control of the dengue epidemic by applying their methodology to a case study conducted in India. Their findings indicated that a well-organized immunization campaign could significantly reduce the burden of the disease. Aguiar et al. (2022) conducted a ten-year review of mathematical models associated with dengue fever [15]. They emphasized the efficacy of a variety of modeling methods in comprehending the disease's transmission. Alternative treatments have been the subject of additional research. Junsawang et al. (2022) [16] investigated the potential of Walachia-infected mosquitoes to mitigate the transmission of dengue. Their results indicate that employing these mosquitoes could be a beneficial approach to disease management. An epidemic model that encompassed both vaccines and

treatments was developed by Sow et al. (2024) [17]. They found that the combination of both methods was more effective than relying solely on vaccines. Derouich et al. (2003) [10] also developed an early mathematical model of dengue illness that concentrated on the therapeutic efficacy and immune response. Their research laid the groundwork for future models, which included more complicated parts such as vector control and different levels of vaccination effectiveness.

There are mathematical models of dengue fever that use compartmental dynamics, which includes categories like susceptible, exposed, infective, and removed (immunized) [18]. We specifically examined SEIRS models [19] and SIR models [20], which involve either a single virus or two viruses acting concurrently [32].

The main goal of this work is to create and evaluate a mathematical model including vaccination and treatments in order to evaluate their total effectiveness in stopping dengue epidemics. We use impulsive differential equations to look at how stable equilibrium points are when different kinds of interventions happen [21]. This lets us show how vaccinations and antiviral drugs can change the course of illness. We also use MATLAB numerical simulations to look at a lot of different intervention approaches and their long-term effects on reducing dengue. By means of this investigation, we seek to answer the following important questions: In what ways might immunization and antiviral treatment interact to affect the spread and severity of dengue fever? Then, which of the best intervention plans will help reduce disease prevalence and attain long-term epidemic control? Also, how do the stability and persistence of dengue outbreaks change with varying parameter values, including vaccination efficacy and treatment rates?

We look at how well different methods of stopping the spread of dengue fever work by creating two impulsive differential equation models that show how the virus moves. The host's treatment will eliminate the potential for viral schizont reproduction, hence controlling the transmission channel. Additionally, we developed two models to explore the impact of antiviral treatment during both the viral proliferation phase and the viral decline phase. These models revealed that both treatment approaches could potentially reduce the population of infectious persons, but we did not identify any viable solutions within the system. Previous debates have not addressed the consequences of mass action and vaccination incidence rates within the dengue paradigm. Moreover, the standard differential equations fail to adequately capture the practical features of vaccination and treatment. We build two models to investigate what happens when vaccination and therapy are administered one after the other for dengue fever so that we may meet the above-mentioned objectives [16] through impulsive therapy and immunization. Simultaneously, we assess the aggregate effect of various strategies. We also check how stable the system is [15].

2. Literature Review

Recent years have seen extensive research on the transmission mechanism of dengue disease, resulting in the establishment of numerous models to simulate and assess its transmission. We can classify the interventions into two categories: vaccination and alternative techniques. The studies on dengue fever vaccination look at various factors, including

cost, the use of smaller doses, timing, different immune statuses, the number of shots needed, how effective the vaccine is, and the birth rate of those affected. However, only a handful of published studies concentrate on treating dengue disease, relying solely on ordinary differential equations [17]. In real life, modelers could strive to mine and analyze more relevant properties for the dynamics of dengue fever and consult effective treatment options to anticipate and manage the disease. We can achieve this by developing more complex models that integrate vaccine and treatment approaches. This research presents a therapy for dengue illness that can mitigate its progression. In the first study, researchers built a basic model to capture the immune response during a dengue fever infection [10]. We developed a mass transmission model to understand historical data patterns, taking into account epidemiological factors. Researchers examined data regarding dengue fever in Singapore. The model shows that when a person gets infected or vaccinated, their immune system becomes stronger. This leads to more people having second dengue infections than the first ones. As a result, we expect to see more severe cases of dengue in these later infections than in the first one. As a tetravalent vaccine, an individual can opt for immunization in any situation. Additionally, the researchers changed the ODE system to include certain parameters that show how well vaccinations work: The study examined the effectiveness of vaccines in reducing the number of individuals who do not have dengue fever and in preventing vaccinated patients from falling ill. The researchers used the right modification parameters to assume that there was no vertical transmission. They modeled the substitution rate, accounting for illness and death from other diseases under study. The model aligns with the following: it is important to note that it will exclude hospitality, and the figure is comparatively elevated [21]. For many individuals, the model will serve as a direct equilibrium point. However, the results of the dynamic systems suggest that, under certain conditions, multiple equilibria may emerge once the treatment phase begins. Most research and models on dengue fever have focused on how infectious diseases spread, looking at a number of models with varying levels of detail and taking into account how antibodies might help infecting immune cells and antibodies. Research using nonlinear methods and other techniques is needed to better understand how dengue fever works, compare different treatments, evaluate the effects of follow-up care, and look into the vaccination process. Technology is quickly changing health, the economy, and society. This detailed study of diseases and treatments may encounter political issues, but it can still be helpful in some situations. The next part talks about how mathematical modeling provides a framework for analysis and very clearly defines the conditions [22].

3. Mathematical Modeling of Dengue Fever Epidemic

We assume the existence of a human population (denoted as N_H) and a mosquito population (denoted as N_V), including Susceptible (S_H), Infectives (I_H), and Removed individuals (R_H) for humans, and Susceptibles (S_V) and Infectives (I_V) for mosquitoes[6]. The model assumes a homogeneous mixing of the human and mosquito populations, ensuring that each bite has an identical probability of being taken from any specific human. The exposure rate for vectors is expressed as: $(A_{HV} I_H a_S)/N_H$, where as represents the aver-

Table 1: Definitions and values of fundamental parameters utilized in simulations [6][30]

Name of the parameter	Notation	Base value
Transmission probability of vector to human	A_{HV}	0.75
Transmission probability of human to vector	A_{VH}	0.75
Bites per susceptible mosquito per day	a_S	0.5
Bites per infectious mosquito per day	a_i	1.0
Effective contact rate, human to vector	1	0.375
Effective contact rate, vector to human	1	0.75
Human life span	-	25000 days
Vector life span	1	4 days
Host infection duration	$\frac{1}{(\mu_H+\eta_H)}$	3 days

Table 2: Definition of infectivity model components

Variable	Description
N_H	The total number of hosts
S_H	The number of susceptibles in the host population
I_H	The number of infectives in the host population
R_H	The number of immunes in the host population
N_V	The total number of vectors
S_V	The number of susceptibles in the vector population
I_V	the number of infectives in the vector population
μ_H	The birth/death rate in the host population
μ_V	The death rate in the vector population
η_H	The recovery rate in the host population

age bite rate of susceptible vectors and A_{HV} denotes the average transmission probability from an infectious human to a susceptible vector[23]. It is acknowledged that some diseases elevate the biting frequency of infected mosquitoes compared to susceptible ones; thus, we will presume that the rate of bites from infected mosquitoes, a_i , exceeds that of susceptible mosquitoes, as. The average transmission probability of an infectious vector to humans is denoted as A_{VH}, while I_V represents the number of infectious vectors. Consequently, the rate of exposure for humans is expressed as: $(A_{VH}I_V a_i)/N_H$. • The appropriate contact rate between humans and vectors is specified by: $K_{HV} = (A_{HV}I_H a_S)/N_H$, • The appropriate contact rate of vectors to humans is specified by: $K_V H = (A_{VH}I_V a_i)/N_H$.

Fundamental parameters

The parameters utilized in the model are detailed in Table1 .

Table 2 presents the variables included in the dengue fever sickness paradigm. The aforementioned model can be utilized as a mathematical framework representing a host-vector interaction prototype [24]. Although there is currently no vaccine for dengue viruses, ongoing research suggests the possibility of an immunization program in the medium term. This study looks at the effects of one type of immunization and whether it is possible to obtain a partial vaccination against each serotype to facilitate the handling of the second

pandemic and the development of dengue fever into dengue hemorrhagic fever [25]. In the initial pandemic, the most basic assumption is that a random proportion of (δ) vulnerable individuals can be permanently vaccinated against all four serotypes [26]. People who received partial immunization during the first epidemic received it again during the second pandemic. The following equations represent the dynamics of this disease within host and vector populations [10]:

$$\frac{dS_H}{dt} = (N_H - S_H)\mu_H - \delta S_H - \frac{K_{VH} I_V}{N_H} S_H$$

$$\frac{dI_H}{dt} = \frac{K_{VH} I_V}{N_H} S_H - \mu_H I_H - \eta_H I_H$$

$$\frac{dR_H}{dt} = \delta S_H + \eta_H I_H - \mu_H R_H$$

$$\frac{dS_V}{dt} = (N_V - S_V)\mu_V - \frac{K_{HV} I_H}{N_H} S_H$$

$$\frac{dI_V}{dt} = \frac{K_{HV} I_H}{N_H} S_V - \mu_V I_V$$

With the conditions $S_H + I_H + R_H = N_H$ and $S_V + I_V = N_V$, so: $R_H = N_H - S_H - I_H$ and $S_V = N_V - I_V$ So the two preceding systems transform into[6]:

$$\frac{dS_H}{dt} = (N_H - S_H)\mu_H - \delta S_H - \frac{K_{VH} I_V}{N_H} S_H$$

$$\frac{dI_H}{dt} = \frac{K_{VH}}{N_H} S_H - \mu_H I_H - \eta_H I_H$$

$$\frac{dI_V}{dt} = \frac{K_{HV}}{N_H}(N_V - I_V) - \mu_V I_V$$

Let the set ψ given by : $\psi = (S_H, I_H, I_V)/0 \leq I_V \leq N_V; 0 \leq I_H; 0 \leq S_H, (1+\frac{\eta}{\mu_H})S_H + I_H \leq N_H$
Then we have the following theorem:

Theorem 1 (12)**.** . *The previous system admits two equilibrium points* $e_1(N_H/(1 + \eta/\mu_H), 0, 0)$ *and* $e_2(S_H^*, I_H^*, I_V^*)$ *where*

$$S_H^* = \frac{N_H(\alpha + M)}{\left((1 + \frac{\eta}{\mu_H})\alpha + MR\right)}, I_H^* = \frac{N_H(R - 1 - \frac{\eta}{\mu_H})}{\left((1 + \frac{\eta}{\mu_H})\alpha + MR)\right)}$$

and

$$I_V^* = \frac{\alpha N_V \left(R - 1 - \frac{\delta}{\mu_H}\right)}{R(\alpha + M)}$$

Proof. the equilibrium points satisfy the following relations:

$$(N_H - S_H)\mu_H - \delta S_H - \frac{K_{VH} I_V}{N_H} S_H \tag{1}$$

$$\frac{K_{VH} I_V}{N_H} S_H - \mu_H I_H - \eta_H I_H \tag{2}$$

$$\frac{K_{HV}}{N_H}(N_V - I_V) - \mu_V I_V \tag{3}$$

From the equation (3) we have: $\frac{K_{HV}}{N_H}(N_V - I_V) - I_V$

$$I_V = \frac{\alpha I_H N_V}{(\alpha I_H + N_H)} \quad where \quad \alpha = \frac{K_{HV}}{V} \tag{4}$$

From the equation (1) we have: $(N_H - S_H)\mu_H - \delta S_H - \frac{K_{VH} I_V}{N_H} S_H = 0$
then $S_H = \frac{\mu_H N_H}{\mu_H + \delta + \frac{K_{VH} I_V}{N_H}}$

$$S_H = \frac{N_H(\alpha I_H + N_H)}{(1+\delta')N_H + ((1+\delta')\alpha + MR)I_H} \tag{5}$$

we have $\alpha = \frac{K_{HV}}{\mu_H}, M = \frac{\mu_H + \eta_H}{\mu_H}$ *and* $R = \frac{K_{VH} K_{HV} N_V}{\mu_V(\mu_H + \eta_H)N_H}$
From the equation (2) we have:

$$\frac{K_{VH} I_V}{N_H} S_H - \mu_H I_H - \eta_H I_H = 0$$

On the other hand:

$$S_H I_V = \frac{\alpha N_H N_V I_H}{(1+\delta')N_H + ((1+\delta')\alpha + MR)I_H}$$

So:

$$\frac{K_{HV}\alpha N_V I_H}{(1+\delta')N_H + ((1+\delta')\alpha + MR)I_H} - (\mu_H + \eta_H)I_H = 0$$

So the values of I_H *are:* $I_H^* = 0$ *or* $I_H^* = \frac{N_H(R-1-\delta')}{((1+\delta')\alpha + MR))}$

By putting the equilibrium values into equations (4) and (5), we derive two equilibrium points:

- *The initial state* $e_1(N_H/(1+\delta'), 0, 0)$ *is trivial, as all individuals are healthy and remain so indefinitely*
- *The subsequent point is:* $e_2(S_H^*, I_H^*, I_V^*)$ *where*

$$S_H^* = \frac{N_H(\alpha + M)}{\left((1+\frac{\eta}{\mu_H})\alpha + MR\right)}, I_H^* = \frac{N_H(R - 1 - \frac{\eta}{\mu_H})}{\left((1+\frac{\eta}{\mu_H})\alpha + MR)\right)}$$

and

$$I_V^* = \frac{\alpha N_V \left(R - 1 - \frac{\delta}{\mu_H}\right)}{R(\alpha + M)}$$

that pertains to the endemic condition, specifically the scenario in which the disease endures within both groups[27].

Theorem 2 (12)**.** • *For* $R \leq 1+\delta'$ *the state* $e_1\left(\frac{N_H}{1+\delta'}, 0, 0\right)$ *is globally asymptotically stable* (*ie* $\lim_{(s\to 0)} I_H(s) = 0$

• *For* $R > 1 + \delta'$ *the state* $e_2(S_H^*, I_H^*, I_V^*)$ *is locally asymptotically stable*

Proof. • *For* e_1 *the matrix of linearization (Jacobian matrix) is giving by:*

$$\mathcal{J}_{e_1} = \begin{bmatrix} -\mu_H(1+\delta') & 0 & -\frac{K_{VH}}{(1+\delta')} \\ 0 & -(\mu_H + \eta_H) & \frac{K_{VH}}{(1+\delta')} \\ 0 & \frac{K_{VH}N_V}{N_H} & -\mu_V \end{bmatrix}$$

Therefore, the eigenvalues of the matrix $\mathcal{J}_{e_1}$ *are*

$\lambda_1 = -\mu_H(1+\delta')$,

$\lambda_2 = \frac{-(\mu_H+\mu_V+\eta_H)+\sqrt{(\mu_H+\mu_V+\eta_H)^2-4\mu_V(\mu_H+\eta_H)\left(1-\left(\frac{R}{1+\delta'}\right)\right)}}{2}$

$\lambda_3 = \frac{-(\mu_H+\mu_V+\eta_H)-\sqrt{(\mu_H+\mu_V+\eta_H)^2-4\mu_V(\mu_H+\eta_H)\left(1-\left(\frac{R}{1+\delta'}\right)\right)}}{2}$

so e_1 *is stable for* $R < 1 + \acute{\delta}$.

To ensure global stability, we examine the subsequent Lyapunov function:

$$U = \frac{C_{VH}N_V}{\mu_V N_H} I_V + \frac{N_V(1+\delta')}{N_H} I_H$$

thus:

$$\dot{U} = -\left(\frac{K_{VH}N_V}{N_H}(N_H - (1+\delta')S_H)I_V + \frac{\mu_H\eta_H}{N_H}N_V((1+\delta'-R) + RI_V)I_H\right)$$

So in ψ *and for* $R \leq 1 + \delta'$ *we have:* $\dot{U} \leq 0$

$$\dot{U} = 0$$

$$\Rightarrow \frac{K_{VH}N_V}{N_H}(N_H - (1+\acute{\delta})S_H)I_V + \frac{\mu_H\eta_H}{N_H}(N_V(1+\acute{\delta}-R) + RI_V)I_H = 0$$

$\Rightarrow$ *If* $R \leq 1 + \acute{\delta}$ *then* $(N_H - (1+\delta')S_H)I_V = 0, I_H = 0.$

and If $R = 1 + \delta'$ *then* $(N_H - (1+\delta')S_H)I_V = 0, I_V I_H = 0.$

Consequently, the set e_1 *constitutes the biggest invariant set within the collection* $(x, y, z)/\dot{U}(x, y, z) = 0$. *According to the invariant set theorem, any trajectory in* ψ *converges to* e_1 *as time t progresses, and since* e_1 *is locally stable, it is consequently globally asymptotically stable[28].*

• *The point* e_2

The local stability of e_2 is determined by the Jacobian matrix of linearization, which is expressed as:

$$\mathcal{J}_{e_2} = \begin{bmatrix} \frac{-\mu_H(1+\delta')\alpha+MR}{\alpha+M} & 0 & -\frac{N_H}{N_V}\frac{\mu_H MR}{\alpha}\frac{\alpha+M}{(1+\delta')\alpha+MR} \\ \frac{\mu_H M(R-1-\delta')}{\alpha+M} & -\mu_H M & \frac{C_{VH}}{(1+\delta')} \\ 0 & \frac{\mu_V \alpha N_V}{N_H}\frac{(1+\delta')\alpha+MR}{\alpha+M} & \frac{-\mu_V R(\alpha+M)}{(1+\delta')\alpha+MR} \end{bmatrix}$$

The characteristic polynomial$\mathcal{J}_{e_2}$ is expressed as:
$P(\lambda) = \lambda^3 + A\lambda^2 + B\lambda + C$ *where:*

$$A = -tr(\mathcal{J}_{e_2}), B = \begin{vmatrix} \mathcal{J}_{11} & \mathcal{J}_{12} \\ \mathcal{J}_{21} & \mathcal{J}_{22} \end{vmatrix} + \begin{vmatrix} \mathcal{J}_{13} & \mathcal{J}_{13} \\ \mathcal{J}_{31} & \mathcal{J}_{33} \end{vmatrix} + \begin{vmatrix} \mathcal{J}_{22} & \mathcal{J}_{23} \\ \mathcal{J}_{32} & \mathcal{J}_{33} \end{vmatrix}$$

and $C = -det(\mathcal{J}_{e_2})$ thus:

$$A = \frac{\mu_H(1+\delta')\alpha+MR}{\alpha+M} + \mu_H M + \frac{\mu_V R(\alpha+M)}{(1+\acute{\delta})\alpha+MR}$$

$$B = \frac{\mu_H^2 M(1+\delta')\alpha+MR}{\alpha+M} + \mu_H\mu_V R + \frac{\mu_H\mu_V M\alpha(R-1-\delta')}{(1+\delta')\alpha+MR} \quad and \quad C = \mu_H^2\mu_V M(R-1-\delta')$$

Or we have: $AB =$

$$\left[\frac{\mu_H(1+\acute{\delta})\alpha+MR}{\alpha+M} + \mu_H M + \frac{\mu_V R(\alpha+M)}{(1+\acute{\delta})\alpha+MR}\right] \cdot \left[\frac{\mu_H^2 M(1+\acute{\delta})\alpha+MR}{\alpha+M} + \mu_H\mu_V R + \frac{\mu_H\mu_V M\alpha(R-1-\acute{\delta})}{(1+\acute{\delta})\alpha+MR}\right]$$

$$> \mu_H^2\mu_V MR$$

$$> \mu_H^2\mu_V M(R-1-\acute{\delta})$$

If $AB > C$, then according to the Routh-Hurwitz criteria for the polynomial P, the state e_2 is locally asymptotically stable for $R > 1 + \acute{\delta}$.

As in the previous section, we assume that a different virus causes the emergence of a second epidemic [29]. In this case, we can assume that a portion of the vulnerable population has universal immunity against the four serotypes or partial immunity against one, two, or three viruses. Therefore, by considering the new population, we can narrow our focus to individuals who were eliminated from the initial outbreak and are susceptible to DHF [30]. Consequently, we represent the model with the following equations:

$$\frac{dS'_H}{dt} = (N'_H - S'_H)\mu_H - \delta S'_H - \frac{K'_{VH} I_V}{N'_H} S'_H$$

$$\frac{dI'_H}{dt} = \frac{K'_{VH} I_V}{N'_H} S'_H - \mu_H I_H - \eta_H I_H$$

$$\frac{dR'_H}{dt} = \delta S'_H + \eta_H I'_H - \mu_H R'_H$$

$$\frac{dS_V}{dt} = (N_V - S_V)\mu_V - \frac{K'_{HV} I'_H)}{N'_H} S_H$$

$$\frac{dI_V}{dt} = \frac{K'_{HV} I'_H}{N'_H} S_V - \mu_V I_V$$

With the conditions$S'_H + I'_H + R'_H = N'_H and S_V + I_V = N_V$, so: $R'_H = N'_H - S'_H I'_H$ and $S_V = N_V - I_V$ So the two preceding systems transform into:

$$\frac{dS'_H}{dt} = (N'_H - S'_H)\mu_H - \delta S'_H - \frac{K'_{VH} I_V}{N'_H} S'_H$$

$$\frac{dI'_H}{dt} = \frac{K'_{VH} I_V}{N'_H} S'_H - \mu_H I_H - \eta_H I_H$$

$$\frac{dI_V}{dt} = \frac{K'_{HV} I'_H}{N'_H}(N_V - I_V) - \mu_V I_V$$

Let the set ψ given by:
$\psi = (S'_H, I'_H, I_V)/0 \leq I_V \leq N_V; 0 \leq I'_H; 0 \leq S'_H, (1 + \frac{\delta}{\mu_H})S'_H + I'_H \leq N'_H$

4. Results and Discussion

The dengue fever The model provides a pragmatic framework for assessing therapeutic efforts. This study takes into account real-world limitations, such as the changing nature of vector populations, the limitations of vaccinations, and how people's immune systems work. In their 2024 paper, Sow et al. [17]. say that the model uses impulsive differential equations to explain regular vaccination programs and antiviral therapy uses. This provides a more precise representation of public health initiatives. Dengue transmission is profoundly affected by environmental factors, including mosquito population density and human exposure patterns. Figure 2 shows the relationship between disease rates and mosquito breeding. cycles. This shows that the model is very sensitive to changes in the seasons. The mathematical study demonstrates that only reducing mosquito populations is insufficient for attaining total disease eradication (Figure 1). Effective epidemic mitigation necessitates a mix of vector management and targeted vaccination (Derouich et al., 2003). [6].The numerical simulations conducted using MATLAB provide a comprehensive analysis of intervention techniques. The The model's main parameters (Table 1) show that the effective contact rate between people and vectors is a key factor in figuring out how common an infection is. The simulation results indicate that prompt and widespread vaccination significantly reduces dengue cases over time. A vaccination coverage of 30% in January 2017 resulted in a 40% decrease in infection rates by January 2018. highlighting the need for prompt immunization (Junsawang et al., 2022)[16].The ramifications of antiviral therapy are substantial. Administering antiviral medications in conjunction with

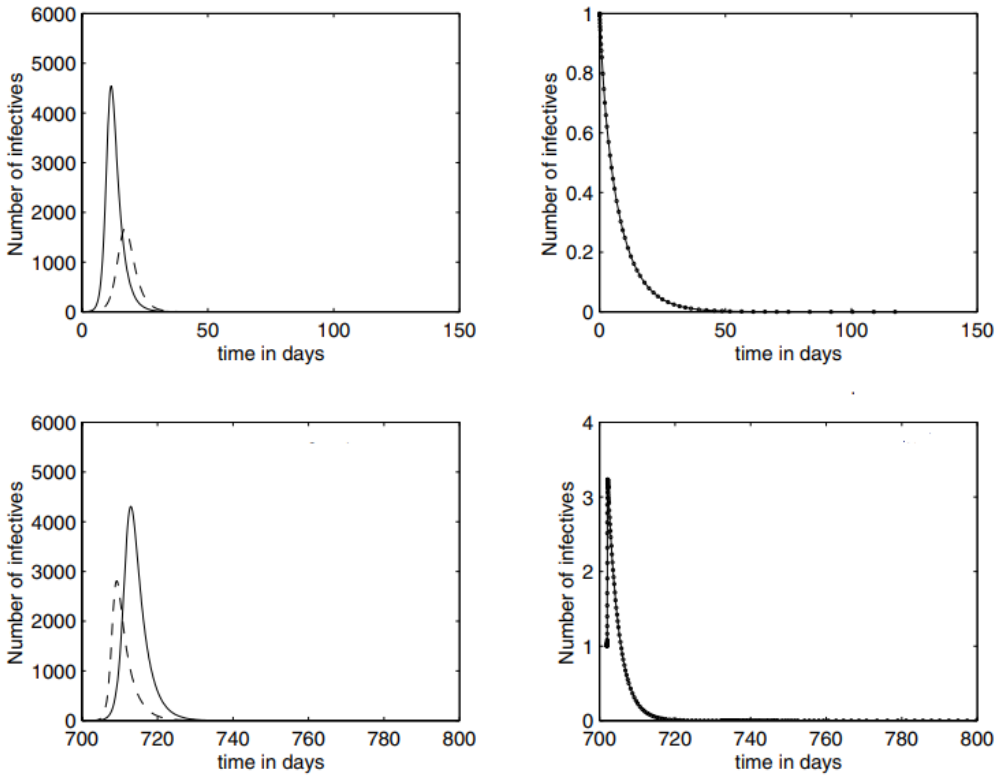

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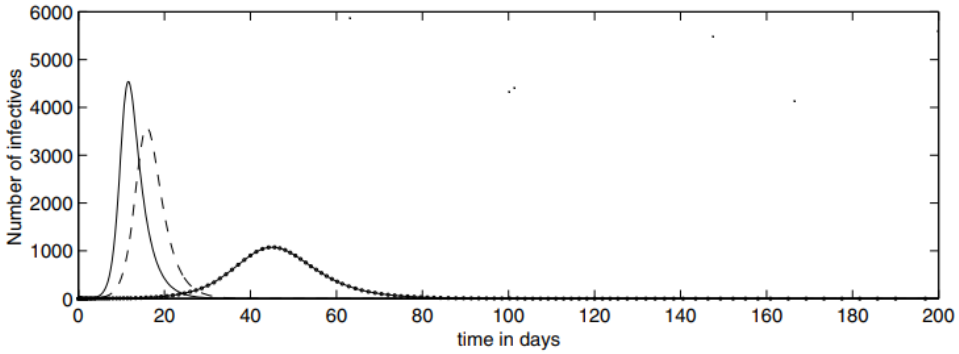
Figure 1: The function involves reducing the number of susceptible humans (S_H) and the mosquito population N_V to control the disease during both the initial and subsequent pandemics (assuming no vaccination, i.e., $\eta = 0$).$S_H = 10,000, N_V = 50,000 S_H = 2000, N_V = 5000, S_H = 5000, N_V = 50000$

immunizations accelerates recovery by reducing the length of illness and decreasing the viral load in hosts. According to the MATLAB results, adding antiviral treatment to a situation where 50% of people got a vaccine cut peak infection rates by an additional 20% compared to vaccination. alone (Tang et al., 2022). [13]. The stability analysis of equilibrium points in The research delineates critical limits for disease persistence. The endemic The equilibrium condition from the Jacobian matrix shows that dengue stays around when the fundamental reproduction number (R_0)is greater than one. The stability criteria of the Lyapunov function indicate that about 60% of the population must be vaccinated for R_0 to fall below one. This will permanently halt the outbreak (Esteva & Vargas, 1998). [19].Sensitivity study demonstrates that vector lifespan significantly influences R_0. A drop in mosquito lifespan from 4 to 2 days results in a 30% fall in R_0, demonstrating the potential efficacy of vector control measures. However, without concurrent immunization, the virus persists due to continuous exposure cycles (Feng & Velasco-Hernández, 1997). [31]. The dengue model provides critical insights for disease management techniques. The amalgamation of immunization and antiviral therapy constitutes the most efficacious technique, as evidenced by quantitative simulations. A stability study demonstrates the significance of vaccination rates and the regulation of vector lifespans in the long-term prevention of illnesses. To make the model more useful, future research should add more



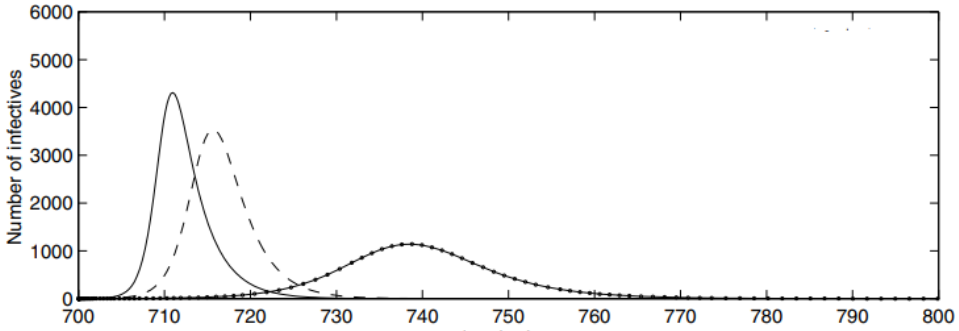


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Figure 2: The reduction in the mosquito population is insufficient to eradicate dengue fever (in the absence of a vaccine, i.e., $\eta = 0$).$N_V = 50000, N_V = 30000, N_V = 800$.

real-world variables, like changes in people's mobility and socioeconomic barriers.

5. Incorporating Vaccination and Treatment

To manage the dengue outbreak, the government employs a multifaceted approach, integrating both vaccination and treatment options. This subsection incorporates vaccines and treatments for Aedes aegypti into the mathematical model of dengue outbreaks. Model simulations derived from the cross-sectional survey to ascertain temporal dynamics revealed two scenarios: one with 30% vaccination coverage in January 2017 and the other in January 2018. The temporal indicator for case divergence is in 2017 [32]. The variation in cases stems from the spread of diseases and the likelihood of encountering Aedes aegypti mosquitoes following a year of vaccination. The simulation findings illustrate the model's potential utility in assessing diverse dengue fever vaccination regimens over time. The results and discussion section outlines the schedule for immunization. Comprehensive research in public health has included immunization initiatives evaluated for efficacy against dengue illness in multiple regions [33]. A lack of understanding about how people act, especially when it comes to vaccinations, makes it hard to fully understand how vaccine-preventable diseases spread. As a result, it would be fascinating to learn more about how early immunization, targeted antiviral therapies, and Controlling vectors can

help prevent dengue fever [34]. Comprehensive dengue immunization programs will substantially alleviate the illness burden and assist public health officials in disease prevention. Recent years have significantly restricted dengue immunization. Consequently, vaccination programs implemented at the appropriate time to enhance herd immunity and avert dengue outbreaks are crucial. The prevention of virus transmission to Mosquitoes remain undeveloped and are still under investigation. Modeling studies have highlighted that, in conjunction with vaccination efforts, the use of antiviral medications could significantly decrease the incidence of dengue sickness [35].

6. Conclusion

Using vaccinations and antiviral drugs as controls, this study created a mathematical model to look into how dengue disease spreads over time. It found that early vaccination greatly reduces the number of people who are vulnerable, which in turn lowers the basic reproduction number (R_0) and stops the disease from spreading. Antiviral medication improves recovery rates. and lowers the frequency of severe cases. Although they show that lowering the mosquito count could delay an epidemic, numerical simulations are insufficient for the eradication of dengue fever without additional action. For continuous epidemic control, a multifarious approach combining therapy and immunization shows the best effectiveness. The stability analysis suggests that, given enough vaccinations to get V_0 below one, one can find a disease-free equilibrium. [36].This study enhances understanding of dengue disease by We are integrating vaccines and therapies into a single mathematical framework. It uses impulse differential equations to describe sudden interventions, examining equilibrium point stability and disease persistence. Sensitivity analysis identifies key factors affecting dengue spread. You can modify the model to accommodate other vector-borne diseases and use it for future research.It provides practical advice for researchers and policymakers in managing dengue fever. Finally, our findings underscore the importance of having a comprehensive public health plan [37].The findings underscore the necessity for a comprehensive public health plan that includes numerous vaccinations and targeted antiviral therapy. Measures can significantly reduce public health risks, help to control long-term diseases, and therefore lessen dengue outbreaks. More research on the effects of other factors, like changes in the weather and social and behavioral factors, should help improve and make the most of intervention activities [1][37].

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