Mathematical Model of Dengue Transmission with Two Serotypes: Control Methods and Vaccination Strategies

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***Abstract*—This study presents a mathematical model to simulate the transmission dynamics of dengue virus (DENV) in human and mosquito populations, focusing on the urban area of Piedecuesta, Santander, Colombia. The model incorporates seasonal variations and evaluates the effectiveness of various control strategies, including mechanical controls, insecticides, larvicides, and vaccination. Through numerical simulations, we demonstrate that the combination of **vaccination and insecticide use** is the most effective strategy for reducing dengue transmission. This combination results in the lowest peak of infected individuals (occurring at day 0) and rapidly increases the number of recovered individuals with immunity. The findings provide actionable recommendations for public health interventions in regions with similar epidemiological profiles.**

I. Introduction and Theoretical Framework

The Dengue virus (DENV) is the fastest-spreading arboviral infection in the world, causing an estimated 390 million infections and 60 million symptomatic cases annually worldwide. It has also been estimated that approximately 40% of the global population lives in tropical and subtropical areas at risk of DENV transmission, corresponding to 2.5 billion people in over 100 countries. Additionally, a large proportion of infections are asymptomatic or cause mild febrile syndromes, making it impossible to quantify transmission using the incidence of clinically suspected cases, as often reported by surveillance systems, since it only represents a fraction of infections [1]. The trend toward global expansion and the increasing burden of the disease, especially in resource-limited countries, has made dengue a public health problem requiring high priority [2].

The disease is transmitted through the bite of an infected female mosquito of the species *Aedes aegypti* or *Aedes albopictus*. For transmission to occur, the mosquito must have bitten an infected person during the viremia period, which occurs after an incubation period of approximately 7-10 days. Additionally, it has been estimated that the incubation period of the disease in the mosquito's body is 4 to 6 days [3].

The mosquito has an aquatic phase (egg-larva-pupa) during which it is not infectious. Furthermore, transmission can occur from host to host through blood transfusion, organ transplantation, and vertical transmission [4].

The virus has four serotypes (DEN 1-4). Individuals infected with one serotype maintain lifelong protective immunity against homologous virus infection, but protective immunity against heterologous serotypes is temporary. When short-term cross-protection wanes, patients experiencing a secondary infection with another serotype are at higher risk of severe disease through the process of antibody-dependent enhancement (ADE). Additionally, due to the short lifespan of mosquitoes, they never recover from the virus. Finally, it has been reported that seasons and temperature influence the infection dynamics [5].

Various studies have identified several potentially effective control methods against dengue spread. Among these, the availability of a tetravalent vaccine against this virus stands out [6], [7]. However, the few vaccines that have achieved this level of immunization (such as TAK-003) are not widely distributed in Colombia [8]. For this reason, other control methods—such as insecticides, mechanical barriers (nets), and awareness campaigns—have gained importance in dengue control; however, further research is needed to evaluate their effectiveness in different regions of the country [6], [7], [9].

This project focuses on the population of Piedecuesta, Santander, as epidemiological studies in this area facilitate the selection of parameter values for the equations. Additionally, according to the Ministry of Health and SIVIGILA, this region is at moderate risk and is part of the municipalities with persistent endemic transmission, accounting for 50% to 70% of cases in Colombia [1]. According to the National Institute of Health, the serotypes present in this region are DENV1 and DENV2, with a higher incidence of DENV2 (10:7) [10], [11].

Considering the above, the following research question is posed: What method or combination of control methods reduces the spread of DENV1 and DENV2 to the greatest extent in the urban area of Piedecuesta, Santander?

The objective of this project is to answer this question to provide recommendations to the Piedecuesta mayor's office that can be applied to reduce dengue cases in the region. For this purpose, the development of a mathematical model is a good alternative, as it allows simultaneous visualization of the population dynamics of humans, mosquitoes, and larvae, with and without controls, and studies their stability and equilibria. Therefore, an SEIR model for human dynamics and an SEI model for mosquitoes are proposed to evaluate the behavior of the coupled systems when including mechanical, chemical, and vaccination control methods. Additionally, simulations were conducted where the model parameters change according to the favorability of climatic conditions for mosquito popula-

tion growth. This design was inspired by the models proposed in [12], [13], and [14].

To answer the research question, the infected human population of the null model was compared with the controls to determine which method or combination of methods reduces the number of infected humans the fastest and to the greatest extent over one year, as this period allows observation of when the disease tends to disappear. For this purpose, the measurement method used is the dynamics of infected humans, their peak, and their value at the end of the December-September period. Additionally, the dynamics of infected mosquitoes are studied as a complement but not as a decision-making method, as even when infected mosquitoes are eliminated from the system, susceptible mosquitoes are still present, and infected humans cause the reappearance of infected mosquitoes [12].

II. Data

Epidemiological data from Piedecuesta were obtained from the National Institute of Health and SIVIGILA [10], [1], primarily to define initial population values and the incidence of each serotype. Parameter values were chosen based on a literature review of various articles and are presented in Figures 8, 9, 10, 11.

III. Calculations and Mathematical Model

A. Null Model

In Figures 1, 2, the block diagram of the null system is presented. The first three stages of the mosquito's life correspond to aquatic states in which it does not acquire the disease (egg, larva, pupa). Therefore, for the purposes of the model, these three states are grouped into a single aquatic phase (A), which will henceforth be called larvae. It is assumed that all larvae become susceptible adults at a rate γ, and mosquito growth will be limited by the carrying capacity of logistic growth of larvae. Logistic growth will be determined by the oviposition rate r, and the number of larvae born will be limited by the carrying capacity k, which reflects the available water resource. A susceptible mosquito (M_S) will become an exposed mosquito (M_E) when it bites an infected human at an effective contact rate between infected humans and susceptible mosquitoes given by β_M. The subscripts 1 and 2 of each variable represent the serotypes DENV1 and DENV2, respectively. Finally, an exposed mosquito will become an infectious mosquito after the incubation period σ_m. Adult mosquitoes will die at a rate μ_1, and larvae at a rate μ_2.

In Figure 2, the dynamics of the human population are shown. A susceptible human (H_S) will become an exposed human (H_E) to the virus when bitten by an infectious mosquito, given by an effective contact rate between infectious mosquitoes and susceptible humans represented by β_H. After the incubation period σ_H, exposed humans will become infected humans (H_I). Finally, infected humans will recover at a rate α and gain permanent immunity to the first serotype they are infected with and cross-immunity to the other serotype, modeled by a percentage reduction $0 < \lambda < 1$ of the contact rate β_H. After this period, humans can be exposed to a secondary infection (H_E12, H_E21: H_I12, H_I21) after which they will recover with permanent immunity to both serotypes. The natural mortality rate for all humans will be μ_H and will be equal to their birth rate to maintain a constant population.

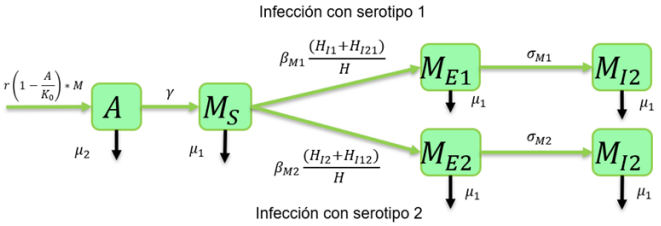

Figure 1. SEI Model for Mosquitoes

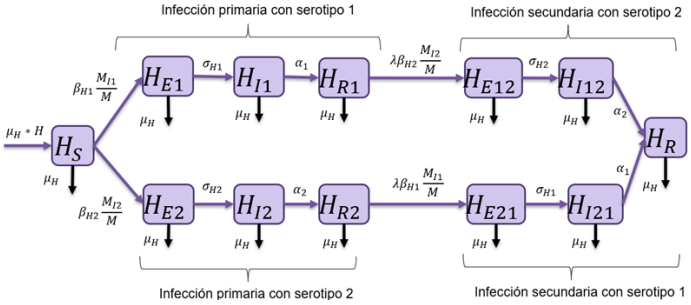

Figure 2. SEIR Model for Humans

Additionally, it is noted that the above parameters are defined within the null system following the assumptions shown below:

- Infection occurs from vector to host and from host to vector.
- Infectious mosquitoes do not become susceptible again due to their short lifespan.
- Two serotypes are considered. There is cross-immunity but no ADE (antibody-dependent enhancement).
- The incubation periods of DENV1 and DENV2 in mosquitoes are 6 days and 4 days (σ_M) and 10 and 7 days in humans (σ_H).
- Vital dynamics: Growth and death rates for humans:
  - There is only one death rate for humans that accounts for natural death.
  - No mortality associated with the virus is considered.
- It is assumed that all humans are born susceptible to the disease.
- Constant human population $N = S + E + I + R$. That is, the death rate (natural death and emigration) is equal to the growth rate (birth, immigration).
- Disease transmission dynamics are modeled considering the law of mass action.

The equations of the null model are found in the appendix.

Additionally, the year was divided into three periods considering the favorability of rainfall. The favorable period corresponds to the months of December-March, intermediate to April-May, and unfavorable to June-September. During each

period, the parameters of the rate of conversion of larvae to adult mosquitoes γ, the mortality rate of adult mosquitoes μ_1, and the carrying capacity K_0 change. The initial conditions and parameter values of the null model are found in the appendices 8, 9, 10, 11.

B. Model with Controls

1) Mechanical Controls: Mechanical controls correspond to strategies implemented to reduce larval breeding sites. When implementing this control in the model, the carrying capacity is modified by $K = K_i * K_0$. Where K_0 is the carrying capacity in the absence of controls, which varies according to climate, and K_i is a dimensionless number between 0 and 1 that indicates the proportion of K_0 that decreases with mechanical controls.

2) Chemical Controls: For chemical controls, two controls were implemented simultaneously: insecticides, which add a value μ_i to the mortality rate of adult mosquitoes μ_1, and larvicides, which add a value μ_l to the mortality rate of larvae μ_2. These rates are modeled as functions that decay exponentially over time from the release of chemicals. It is assumed that chemicals are released during the first week of each month and persist in the environment for 15 days. On the first day, the release is maximum, and at most 10% of the total amount of chemicals released remains in the environment. These functions also vary with the rainy season.

3) Vaccination: When implementing this control, permanent immunity is provided to susceptible humans. When adapting this situation to the proposed model, susceptible humans pass at a rate v to the recovered human population. In this case, it is assumed that the vaccine is 100% effective.

In the appendix 13, 14, the box diagram obtained when implementing the controls is shown. The effect of mechanical controls is underlined in blue, chemical controls in green, and vaccination in yellow. Additionally, in the appendix section in Table 12, the parameter values and variations associated with each control are found.

IV. Analysis and Discussion of Results

In a closed population where the birth rate equals the death rate, there is a proliferation of dengue within the first 30 to 40 days of disease transmission. This is evidenced in many studies, including one conducted by Side and colleagues [15], who evaluated dengue fever transmission in Medan, a province in Indonesia with climatic conditions similar to Piedecuesta, Santander. They found that the population dynamics consist of a decrease in the number of susceptibles, which is proportional to the increase in exposed cases. Around the first third of the simulation period, it is observed that the number of humans bitten by mosquitoes is close to a third of the total population, followed by a peak in infected individuals, which occurs four days after the peak of exposed individuals, associated with the virus incubation period in the body; then, there is a decrease in infected individuals, reaching its minimum around 50 days from the start of the epidemic. This is consistent with the results of the obtained model that does not consider any type of control in disease transmission. A proportional decrease in the number of recovered individuals is observed during the favorable period of the year, then stabilizes for the other two periods of the year. Since there is a high proliferation of mosquitoes during the favorable period, the number of vectors is sufficient to infect most of the population in the first four months of the epidemic, with a peak in exposed individuals around day 51, decreasing to zero at the start of the intermediate period. Regarding infected individuals, a critical peak is observed around day 63, corresponding to 40,631 people, consistent with the 10 days designated as the virus incubation period in the body after being bitten by mosquitoes 3 and appendix. Due to this high number of infected individuals, it is necessary to implement controls for disease transmission to achieve the best time-disease peak relationship.

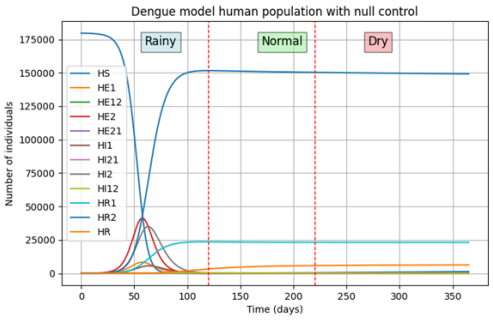

Figure 3. Null SEIR Model: Dengue transmission in humans for the December-September period, sum of each variable

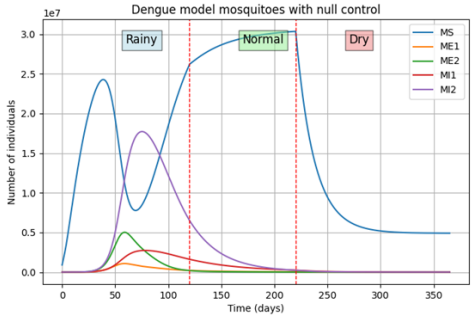

Figure 4. Null SEI Model: Dengue transmission in mosquitoes for the December-September period

Simulations were also conducted for each control and each pair of controls. That is, the model was run for mechanical control, mechanical control + insecticide, mechanical control + larvicide, and so on until all control pairs were completed. This was done to determine which control or pair of controls offered the best method for controlling disease spread.

After experimentation, it was determined that the most effective control is Vaccination + Insecticide fumigation 5. To study the variable of interest (infected humans), the variables of humans infected by each serotype were summed. The effect of vaccination + insecticide fumigation on mosquitoes results in an exponential decrease in the susceptible population. This can be seen in Figure 7, where the mosquito population

decreases very rapidly and does not allow contagion between mosquitoes. In Figure 6, it can be observed that human population shows no visible variations in the number of susceptibles, but due to the vaccination the recovered individuals grows fast, indicating that it could even eradicate the population and reach a point where human contagions are zero.

Control	Peak (HI)	Day
Null	40631	63
Mechanical	40633	61
Mechanical + Insecticide	17801	365
Mechanical + Larvicide	40309	61
Mechanical + Vaccination	17456	66
Insecticide	18226	365
Insecticide + Larvicide	11037	130
Insecticide + Vaccination	156	0
Larvicide	40211	63
Larvicide + Vaccination	16602	68
Vaccination	16843	68

Figure 5. Peaks of infected humans by control.

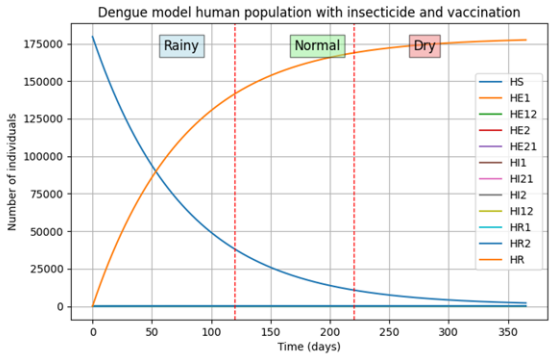

Figure 6. Computational model for the behavior of dengue disease in the human population with insecticide use and vaccination

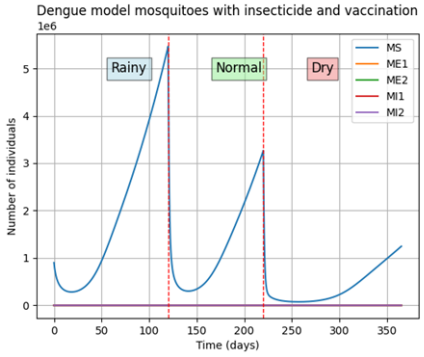

Figure 7. Computational model for the behavior of dengue disease in the mosquito population with insecticide use and vaccination

This mathematical model was compared with the rest of the models for the combination of dengue spread prevention methods and has similar behaviors to some combinations; however, since this is a combined method, it means more expensive costs than a single method. Therefore, it can be affirmed that this would be the recommended model for the mayor's office, as it has better effectiveness than the other methods and their combinations.

V. Conclusion

Regarding future work, efforts should be made to reduce the system so that equilibria can converge, and stability can be studied, and the basic reproductive number can be calculated. Additionally, the model could be generalized for n serotypes and their interactions. These interactions have been modeled based on the genetic similarity of serotypes to define their reactivity, cross-immunity, and antibody-dependent enhancement [16]. Another factor to consider in the future is the lack of implementation of a dengue vaccination plan in Colombia. The only vaccine authorized for distribution by the WHO is Dengvaxia, which can only be administered to people over 9 years of age who have been previously infected with the virus [17]. Therefore, future research should focus on identifying combinations of control mechanisms other than preventive vaccination that allow achieving results similar to those obtained with vaccination campaigns in terms of reducing dengue cases in humans. However, thanks to this model, an approximation to the reality of disease behavior without any control and the behavior of populations when each control is applied can be obtained. It can be concluded from the models that the method to recommend and that best prevents the appearance of the disease with the lowest investment cost is frequent insecticide use. Therefore, to answer the research question, the method that reduces the spread to the greatest extent for the population of the urban area of Piedecuesta, Santander, is chemical control through insecticides combined with vaccination.

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VI. Appendices

A. Null Model Equations:

Vectors:

$$M = M_S + M_{E1} + M_{E2} + M_{I1} + MI_2 \tag{1}$$

$$\frac{dA}{dt} = r\left(1 - \frac{A}{K_0}\right)M - \mu_2 A - \gamma A \tag{2}$$

$$\frac{dM_S}{dt} = \gamma A - \mu_1 M_S - \beta_{M1} M_S \frac{(H_{I1} + H_{I21})}{H} - \beta_{M2} M_S \frac{(H_{I2} + H_{I12})}{H} \tag{3}$$

$$\frac{dM_{E1}}{dt} = \beta_{M1} M_S \frac{(H_{I1} + H_{I21})}{H} - \mu_1 M_{E1} - \sigma_{M1} M_{E1} \tag{4}$$

$$\frac{dM_{E2}}{dt} = \beta_{M2} M_S \frac{(H_{I2} + H_{I12})}{H} - \mu_2 M_{E2} - \sigma_{M2} M_{E2} \tag{5}$$

$$\frac{dM_{I1}}{dt} = \sigma_{M1} M_{E1} - \mu_1 M_{I1} \tag{6}$$

$$\frac{dM_{I2}}{dt} = \sigma_{M2} M_{E2} - \mu_2 M_{I2} \tag{7}$$

Humans:

$$H = H_s + H_{E1} + H_{E2} + H_{I1} + H_{I2} + H_{E12} + H_{I12} + H_{E21} + H_{I21} + H_{R1} + H_{R2} + H_R \tag{8}$$

$$\frac{dH_S}{dt} = \mu_H H - \mu_H H_S - \beta_{H1} H_S \frac{M_{I1}}{M} - \beta_{H2} H_S \frac{M_{I2}}{M} \tag{9}$$

$$\frac{dH_{E1}}{dt} = \beta_{H1} H_S \frac{M_{I1}}{M} - \sigma_{H1} H_{E1} - \mu_H H_{E1} \tag{10}$$

$$\frac{dH_{E2}}{dt} = \beta_{H2} H_S \frac{M_{I2}}{M} - \sigma_{H2} H_{E2} - \mu_H H_{E2} \tag{11}$$

$$\frac{dH_{I1}}{dt} = \sigma_{H1} H_{E1} - \mu_H H_{I1} - \alpha_1 H_{I1} \tag{12}$$

$$\frac{dH_{I2}}{dt} = \sigma_{H2} H_{E2} - \mu_H H_{I2} - \alpha_2 H_{I2} \tag{13}$$

$$\frac{dH_{R1}}{dt} = \alpha_1 H_{I1} \ - \mu_H H_{R1} - \lambda \beta_{H2} H_{R1} \frac{M_{I2}}{M} \tag{14}$$

$$\frac{dH_{R2}}{dt} = \alpha_2 H_{I2} \ - \mu_H H_{R2} - \lambda \beta_{H1} H_{R2} \frac{M_{I1}}{M} \tag{15}$$

$$\frac{dH_{E12}}{dt} = \lambda \beta_{H2} H_{R1} \frac{M_{I2}}{M} - \mu_H H_{E12} - \sigma_{H2} H_{E12} \tag{16}$$

$$\frac{dH_{E21}}{dt} = \lambda \beta_{H1} H_{R2} \frac{M_{I1}}{M} - \mu_H H_{E21} - \sigma_{H1} H_{E21} \tag{17}$$

$$\frac{dH_{I12}}{dt} = \sigma_{H2} H_{E12} - \mu_H H_{I12} - \alpha_2 H_{I12} \tag{18}$$

$$\frac{dH_{I21}}{dt} = \sigma_{H1} H_{E21} - \mu_H H_{I21} - \alpha_1 H_{I21} \tag{19}$$

$$\frac{dH_R}{dt} = \alpha_2 H_{I12} + \alpha_1 H_{I21} - \mu_H H_R \tag{20}$$

B. Control Model Equations:

Vectors:

$$\frac{dA}{dt} = r\left(1 - \frac{A}{K_i * K_0}\right) M - (\mu_2 + \mu_l)A - \gamma A \tag{21}$$

$$\frac{dM_S}{dt} = \gamma A - (\mu_1 + \mu_i)M_S - \beta_{M1} M_S \frac{(H_{I1} + H_{I21})}{H} - \beta_{M2} M_S \frac{(H_{I2} + H_{I12})}{H} \tag{22}$$

$$\frac{dM_{E1}}{dt} = \beta_{M1} M_S \frac{(H_{I1} + H_{I21})}{H} - (\mu_1 + \mu_i)M_{E1} - \sigma_{M1} M_{E1} \tag{23}$$

$$\frac{dM_{E2}}{dt} = \beta_{M2} M_S \frac{(H_{I2} + H_{I12})}{H} - (\mu_2 + \mu_i)M_{E2} - \sigma_{M2} M_{E2} \tag{24}$$

$$\frac{dM_{I1}}{dt} = \sigma_{M1} M_{E1} - (\mu_1 + \mu_i)M_{I1} \tag{25}$$

$$\frac{dM_{I2}}{dt} = \sigma_{M2} M_{E2} - (\mu_1 + \mu_i)M_{I2} \tag{26}$$

Humans:

$$\frac{dH_S}{dt} = \mu_H H - \mu_H H_S - \beta_{H1} H_S \frac{M_{I1}}{M} - \beta_{H2} H_S \frac{M_{I2}}{M} - v H_S \tag{27}$$

$$\frac{dH_{E1}}{dt} = \beta_{H1} H_S \frac{M_{I1}}{M} - \sigma_{H1} H_{E1} - \mu_H H_{E1} \tag{28}$$

$$\frac{dH_{E2}}{dt} = \beta_{H2} H_S \frac{M_{I2}}{M} - \sigma_{H2} H_{E2} - \mu_H H_{E2} \tag{29}$$

$$\frac{dH_{I1}}{dt} = \sigma_{H1} H_{E1} - \mu_H H_{I1} - \alpha_1 H_{I1} \tag{30}$$

$$\frac{dH_{I2}}{dt} = \sigma_{H2} H_{E2} - \mu_H H_{I2} - \alpha_2 H_{I2} \tag{31}$$

$$\frac{dH_{R1}}{dt} = \alpha_1 H_{I1} \ - \mu_H H_{R1} - \lambda \beta_{H2} H_{R1} \frac{M_{I2}}{M} \tag{32}$$

$$\frac{dH_{R2}}{dt} = \alpha_2 H_{I2} \ - \mu_H H_{R2} - \lambda \beta_{H1} H_{R2} \frac{M_{I1}}{M} \tag{33}$$

$$\frac{dH_{E12}}{dt} = \lambda \beta_{H2} H_{R1} \frac{M_{I2}}{M} - \mu_H H_{E12} - \sigma_{H2} H_{E12} \tag{34}$$

$$\frac{dH_{E21}}{dt} = \lambda \beta_{H1} H_{R2} \frac{M_{I1}}{M} - \mu_H H_{E21} - \sigma_{H1} H_{E21} \tag{35}$$

$$\frac{dH_{I12}}{dt} = \sigma_{H2} H_{E12} - \mu_H H_{I12} - \alpha_2 H_{I12} \tag{36}$$

$$\frac{dH_{I21}}{dt} = \sigma_{H1} H_{E21} - \mu_H H_{I21} - \alpha_1 H_{I21} \tag{37}$$

$$\frac{dH_R}{dt} = \alpha_2 H_{I12} + \alpha_1 H_{I21} - \mu_H H_R + v H_S \tag{38}$$

Variables de estado	Descripción	Unidades	Valor	Referencia
H_{S0}	Población de humanos susceptibles	[Humanos]	179.668	*Estupiñan et al, 2020.*
H_{E1_0}	Población de humanos expuestos al serotipo 1	[Humanos]	0	
H_{E2_0}	Población de humanos expuestos al serotipo 2	[Humanos]	0	
H_{I1_0}	Población de humanos infectados por el serotipo 1	[Humanos]	23	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
H_{I2_0}	Población de humanos infectados por el serotipo 2	[Humanos]	55	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
H_{R1_0}	Población de humanos recuperados del serotipo 1	[Humanos]	0	
H_{R2_0}	Población de humanos recuperados del serotipo 2	[Humanos]	0	
H_{E12_0}	Población de humanos expuestos al serotipo 2 después de ser infectados por el serotipo 1	[Humanos]	0	
H_{E21_0}	Población de humanos expuestos al serotipo 1 después de ser infectados por el serotipo 2	[Humanos]	0	
H_{I12_0}	Población de humanos infectados por el serotipo 2 después de ser infectados por el serotipo 1	[Humanos]	23	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
H_{I21_0}	Población de humanos infectados por el serotipo 1 después de ser infectados por el serotipo 2	[Humanos]	55	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
H_{R_0}	Población de humanos recuperados de ambos serotipos	[Humanos]	0	
A_0	Población de mosquitos en estado acuático	[Larvas]	2.000.000	Por cada humano se aproximan 11 larvas
M_{S0}	Población de mosquitos en estado adulto susceptibles	[Mosquitos]	900.000	
M_{E1_0}	Población de mosquitos en estado adulto expuestos al serotipo 1	[Mosquitos]	0	
M_{E2_0}	Población de mosquitos en estado adulto expuestos al serotipo 2	[Mosquitos]	0	
M_{I1_0}	Población de mosquitos en estado adulto infectados por el serotipo 1	[Mosquitos]	0	
M_{I2_0}	Población de mosquitos en estado adulto infectados por el serotipo 2	[Mosquitos]	0	
Parámetros relativos a la	Descripción	Unidades	Valor	Referencia

Figure 8. Initial conditions and parameter values of the null model.

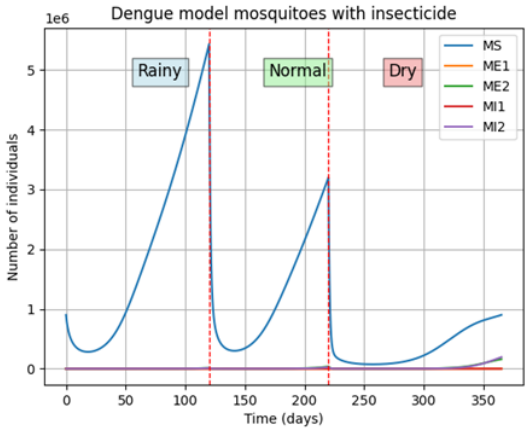

Figure 23. SEI Model: Dengue transmission among mosquitoes with frequent insecticide use.

Parámetros relativos a la población de humanos	Descripción	Unidades	Valor	Referencia
μ_H	Tasa de muerte (muerte natural y emigración) es igual a la tasa de crecimiento (natalidad e inmigración).	[1/t] t → [días]	0.000042 (Aprox 70 años)	*Carvalho et al, 2019.*
σ_{H1}	Periodo de incubación del serotipo 1 en humanos	[1/t]	0.1 (10 días)	*WHO, 2018. Aguiar et al, 2022*
σ_{H2}	Periodo de incubación del serotipo 2 en humanos	[1/t]	0.142 (7 días)	*WHO, 2018. Aguiar et al, 2022*
α_1	Tasa de recuperación de los humanos al serotipo 1	[1/t]	0.126 (7.9 días)	*Lourenco et al, 2016*
α_2	Tasa de recuperación de los humanos al serotipo 2	[1/t]	0.142 (7 días)	*Lourenco et al, 2016*

Figure 9. Initial conditions and parameter values of the null model.

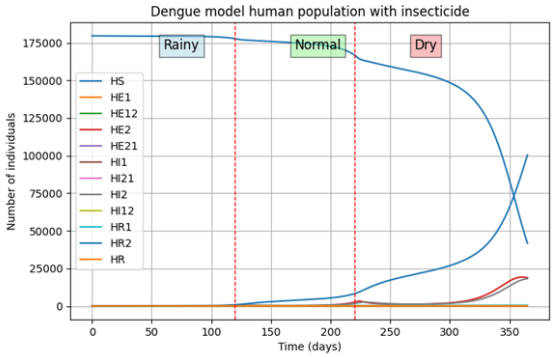

Figure 24. SEIR Model: Dengue transmission among humans with frequent insecticide use.

Parámetros relativos a la población de mosquitos	Descripción	Unidades	Valor	Referencia
r	Crecimiento intrínseco (tasa de oviposición de A. Aegyti)	[1/t]	1	*Carvalho et al, 2019.*
γ	Tasa de conversión de larvas a mosquitos adultos	[1/t]	0.346, favorable 0.323, intermedio 0.091, no favorable	*Carvalho et al, 2019.*
μ_1	Tasa de mortalidad de mosquitos adultos	[1/t]	0.042, favorable 0.040, intermedio 0.059, no favorable	*Carvalho et al, 2019.*
μ_2	Tasa de mortalidad de mosquitos en estado acuático	[1/t]	0.05	*Carvalho et al, 2019.*
K_0	Capacidad de carga para los mosquitos en estado acuático en ausencia de controles	[Mosquitos]	4.500.000, Favorable 4.000.000, Intermedio 3.500.000, Desfavorable	*Carvalho et al, 2019.*
σ_{M1}	Periodo de incubación del serotipo 1 en mosquitos	[1/t]	0.167 (6 días)	*Carvalho et al, 2019*
σ_{M2}	Periodo de incubación del serotipo 2 en mosquitos	[1/t]	0.25 (4 días)	*Carvalho et al, 2019.*

Figure 10. Initial conditions and parameter values of the null model.

Parámetros relativos a la transmisión del virus	Descripción	Unidades	Valor	Referencia
β_{M1}	Tasa de contacto efectivo entre humanos infectados con el serotipo 1 y mosquitos susceptibles	[1/t]	0.7	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
β_{M2}	Tasa de contacto efectivo entre humanos infectados con el serotipo 2 y mosquitos susceptibles	[1/t]	0.75	*Carvalho et al, 2019.*
β_{H1}	Tasa de contacto efectivo entre mosquitos infectados por el serotipo 1 y humanos susceptibles	[1/t]	0.325	*Pérez-Castro et al, 2016. Aguiar et al, 2022*
β_{H2}	Tasa de contacto efectivo entre mosquitos infectados por el serotipo 2 y humanos susceptibles	[1/t]	0.375	*Carvalho et al, 2019.*
λ	Porcentaje de reducción de la infección secundaria (inmunidad cruzada). $0 < \lambda < 1$	[Adimensional]	0.011 (3 meses)	*Lourenco et al, 2016. Centre virchow Villerme, 2018*

Figure 11. Initial conditions and parameter values of the null model.

Parámetros control mecánico	Descripción	Unidades	Valor según el control mecánico
K_i	Número adimensional entre 0 y 1 que indica la proporción de K_0 que disminuye con relación a los controles mecánicos	[Adimensional]	1 sin control mecánico 0.5 con control mecánico
Parámetros control químico	**Descripción**	**Unidades**	**Valor según el control químico**
μ_i	Tasa de mortalidad asociadas al efecto del insecticida.	$[\frac{1}{t}]$	$0.958e^{-0.00151t}$ favorable $0.960e^{-0.00151t}$ intermedio $0.941e^{-0.00149t}$ desfavorable
μ_l	Tasa de mortalidad asociadas al efecto del larvicida.	$[\frac{1}{t}]$	$0.810e^{-0.00139t}$ favorable $0.825e^{-0.00141t}$ intermedio $0.884e^{-0.00145t}$ desfavorable
Parámetros vacunación	**Descripcion**	**Unidades**	**Valor según la vacunación**
v	Tasa de vacunación	$[\frac{1}{t}]$	0 sin vacunación 0.13 con vacunación intensiva 0.013 con vacunación poco intensiva

Figure 12. Control parameters.

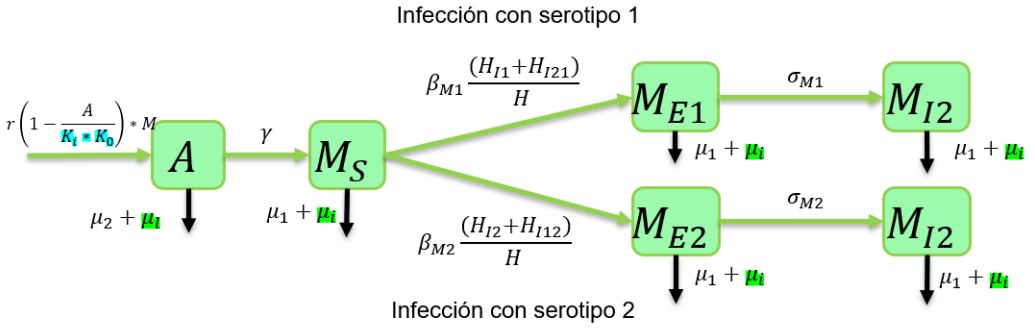

Figure 13. Block diagram of the system with controls for the mosquito population

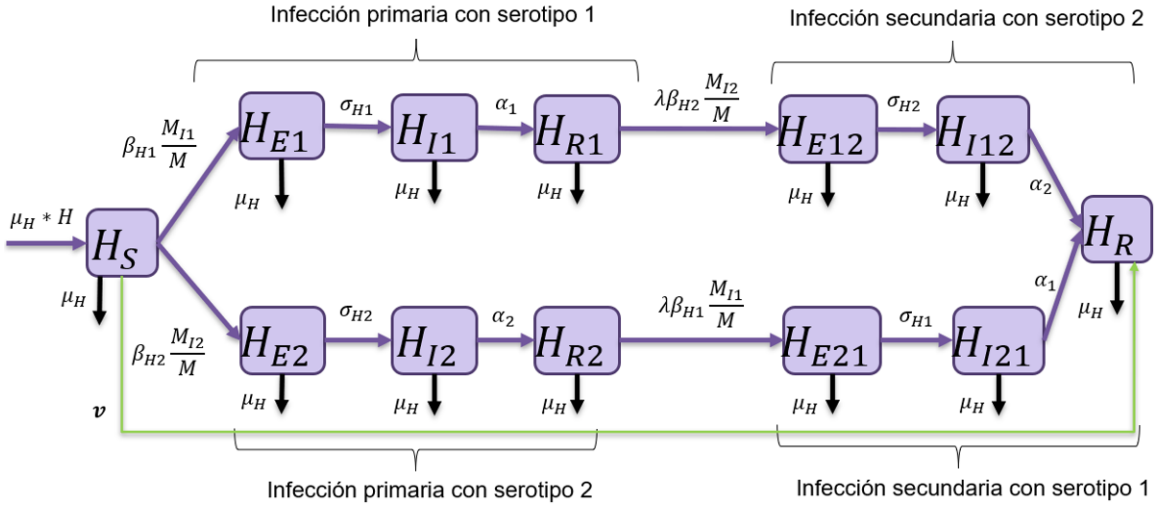

Figure 14. Block diagram of the system with controls for the human population

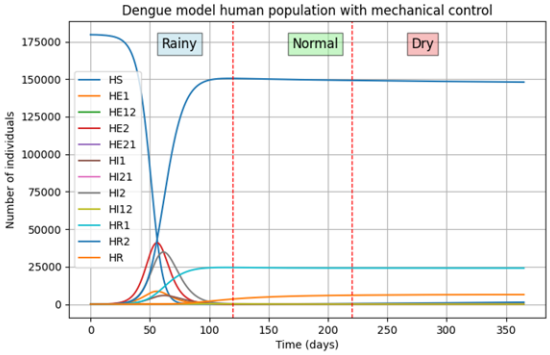

Figure 15. Computational model for the behavior of dengue disease in the human population with mechanical control

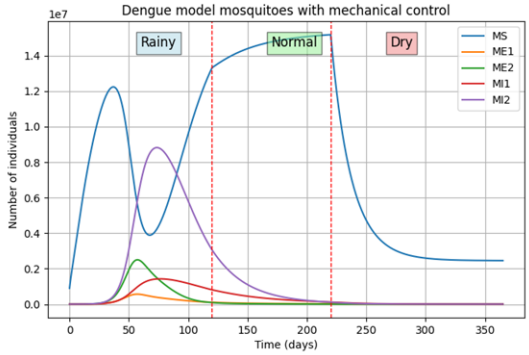

Figure 16. Computational model for the behavior of dengue disease in the mosquito population with mechanical control

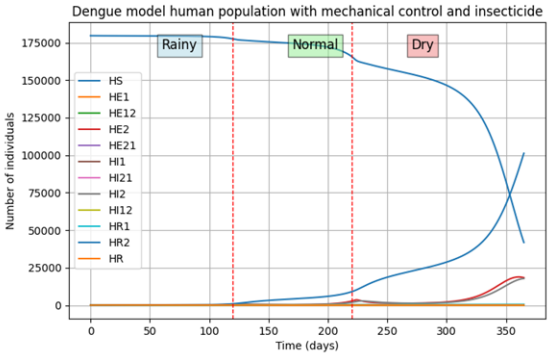

Figure 17. Computational model for the behavior of dengue disease in the human population with mechanical control and insecticide use

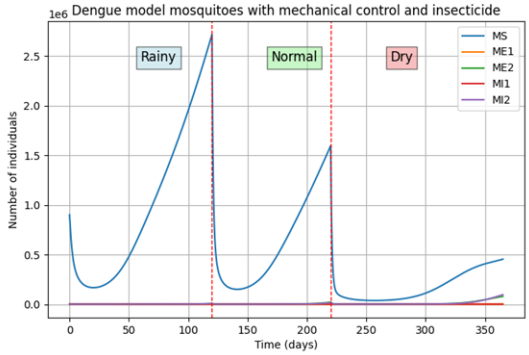

Figure 18. Computational model for the behavior of dengue disease in the mosquito population with mechanical control and insecticide use

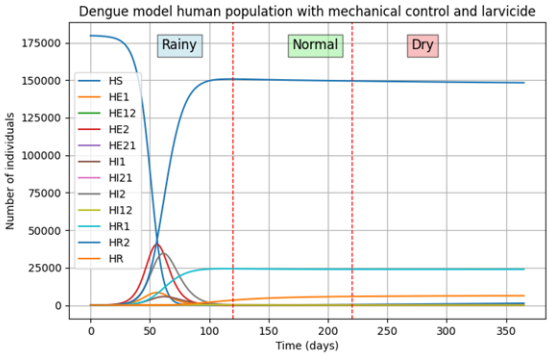

Figure 19. Computational model for the behavior of dengue disease in the human population with mechanical control and larvicide use

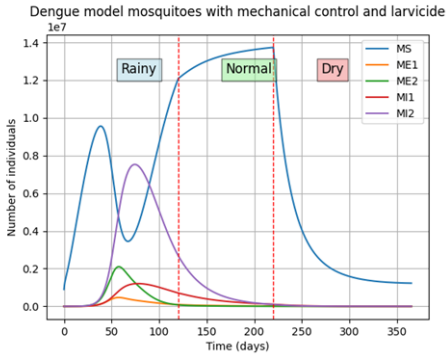

Figure 20. Computational model for the behavior of dengue disease in the mosquito population with mechanical control and larvicide use

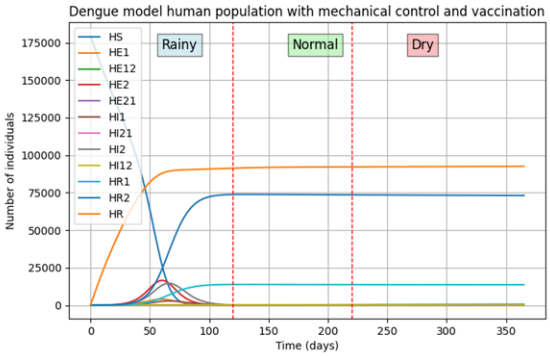

Figure 21. Computational model for the behavior of dengue disease in the human population with mechanical control and vaccination

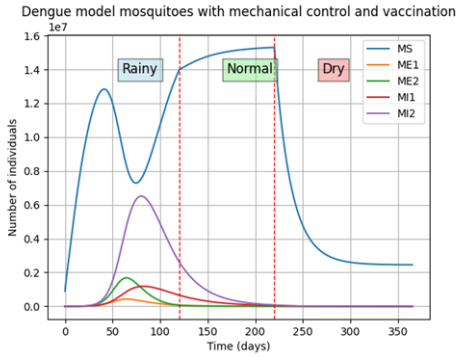

Figure 22. Computational model for the behavior of dengue disease in the mosquito population with mechanical control and vaccination.

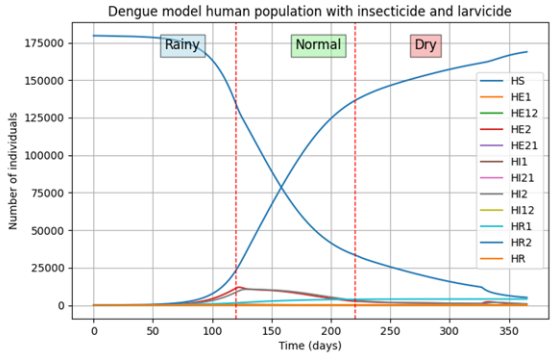

Figure 25. Computational model for the behavior of dengue disease in the human population with insecticide and larvicide use

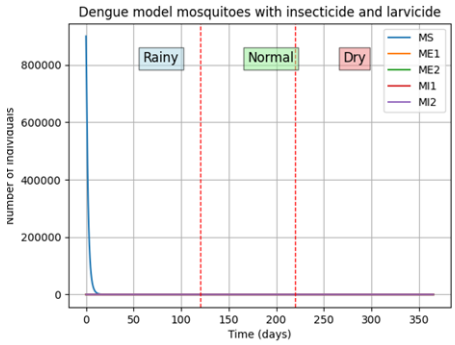

Figure 26. Computational model for the behavior of dengue disease in the mosquito population with insecticide and larvicide use

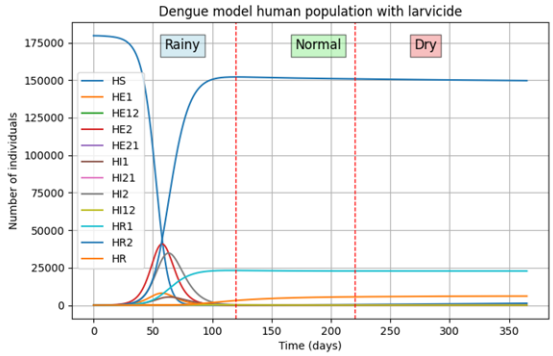

Figure 27. Computational model for the behavior of dengue disease in the human population with larvicide use

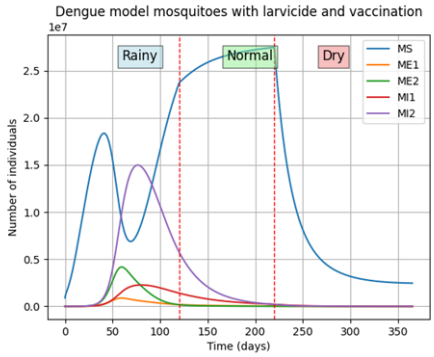

Figure 28. Computational model for the behavior of dengue disease in the mosquito population with larvicide use

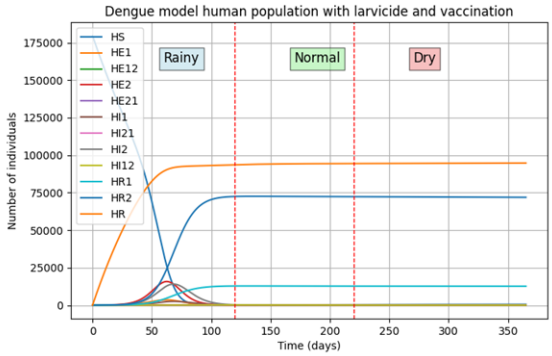

Figure 29. Computational model for the behavior of dengue disease in the human population with larvicide use and vaccination

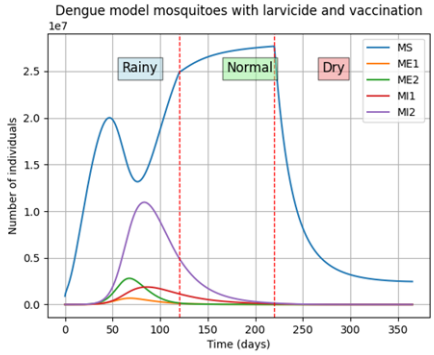

Figure 30. Computational model for the behavior of dengue disease in the mosquito population with larvicide use and vaccination

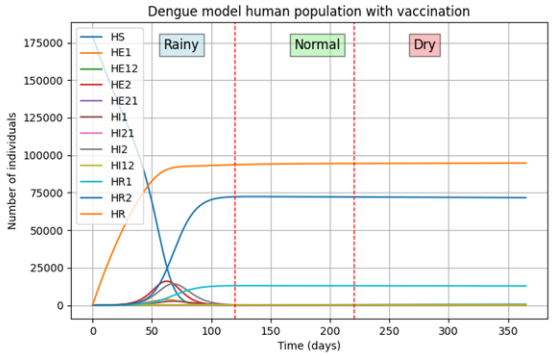

Figure 31. Computational model for the behavior of dengue disease in the human population with vaccination

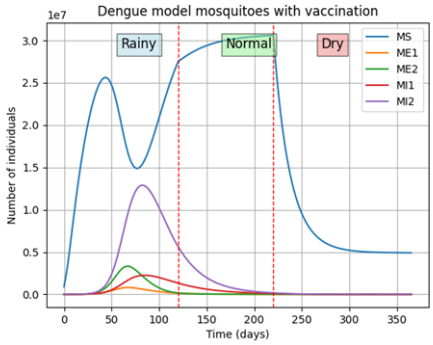

Figure 32. Computational model for the behavior of dengue disease in the mosquito population with vaccination

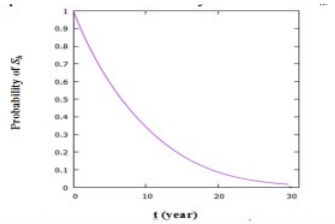

Figure 4. Probability of S_h through simulation

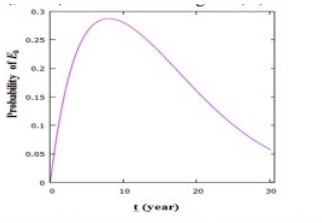

Figure 5 Probability of E_h through simulation

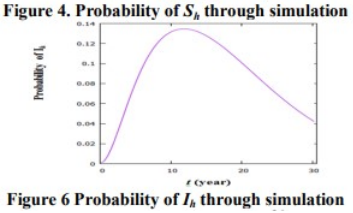

Figure 6 Probability of I_h through simulation

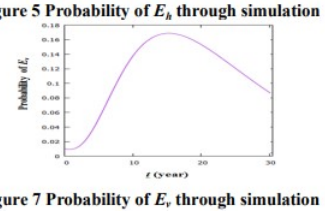

Figure 7 Probability of E_v through simulation

Figure 33. Simulation results obtained in the study available in [15], illustrating the population dynamics of dengue in Medan