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SCHOOL OF APPLIED MATHEMATICS**

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**UTILIZATION OF ENVIRONMENTAL AND EPIDEMIOLOGICAL
INDICATORS IN THE STUDY OF MALARIA DYNAMICS**

Rio de Janeiro
2023

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Bachelor dissertation presented to the School
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Area of Study: Biological modeling.

Advisor: Flávio Codeço Coelho

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And approved in 12/12/2023

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Abstract

Malaria is an infectious disease transmitted by mosquitoes infected by protozoa of the genus *Plasmodium*, with the Amazon region being considered an endemic area for the disease. This work aims to analyze the behavior of this transmission based on climatic and environmental changes, such as temperature, precipitation and deforestation, through proposed modifications to the SIR and SEI models, in order to contribute to the study of applications of external effects on the evolution of the disease. The Trajetórias Project, developed by the Synthesis Center on Biodiversity and Ecosystem Services (SinBiose/CNPq) will be used as a reference base for the analyses.

Keywords: Biological modelling. Malaria. Amazon. SIR. SEI.

Resumo

A malária é uma doença infecciosa transmitida por mosquitos infectados por protozoários do gênero *Plasmodium*, sendo a região amazônica considerada área endêmica para a doença. Esse trabalho tem como intuito analisar o comportamento dessa transmissão baseado em modificações climáticas e ambientais, como temperatura, precipitação e desmatamento, através de modificações propostas aos modelos SIR e SEI, de forma a contribuir no estudo de aplicações de efeitos externos na evolução da doença. O Projeto Trajetórias, desenvolvido pelo Centro de Biodiversidade e Serviços Ecosistêmicos (SinBiose/CNPq), será usado como base de referência para as análises.

Palavras-chave: Modelagem biológica. Malária. Amazônia. SIR. SEI.

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1 Introduction

The Amazon is one of the largest and most biodiverse tropical forests in the world, harboring numerous species of plants, animals, and microorganisms, including vectors and pathogens responsible for the transmission of various diseases. Among them, one of the most common is malaria, caused by protozoa of the genus *Plasmodium*, transmitted by the bite of the infected female mosquito of the genus *Anopheles*. It is present in 22 American countries, but the areas with the highest risk of infection are located in the Amazon region, encompassing nine countries, which accounted for 68% of infection cases in 2011 (PIMENTA et al., 2015). Although malaria is prevalent in the Americas, it is not limited to this continent and is found in countries in Africa and Asia, resulting in more than two million cases of infection and 445,000 deaths worldwide in 2016 (JOSLING; WILLIAMSON; LLINÁS, 2018).

Notably, vector-borne disease transmission is closely related to environmental changes that interfere with the ecosystem of both transmitting organisms and affected organisms. In the case of the Amazon, agricultural and livestock settlements are among the factors that most favor disease transmission, both due to the deforestation they cause for establishment and the clustering of people in environments close to the vector's habitat (SILVA-NUNES, M. da et al., 2008), especially by clustering non-immune migrants near these natural and artificial breeding sites (SILVA-NUNES, Mônica da et al., 2012).

Additionally, other factors such as rainfall, wildfires, and mining also significantly influence disease transmission in the region. These events result in habitat loss, ecosystem fragmentation, and climate changes, affecting the distribution and abundance of vectors and hosts, as well as their interaction with pathogens. Furthermore, population growth and urbanization also play a crucial role in disease spread, increasing human exposure to vectors and infection risks.

In this context, this work aims to investigate vector-borne disease transmission in the Amazon and analyze how environmental impacts influence the dynamics of malaria transmission, the ecological and socioeconomic factors affecting this spread, and possible prevention and control strategies. The main reference for this research is the Trajetórias Project, developed by the Center for Biodiversity and Ecosystem Services (SinBiose/CNPq), which is a dataset including environmental, epidemiological, economic, and socioeconomic indicators for all municipalities in the Legal Amazon, analyzing the spatial and temporal relationship between economic trajectories linked to the dynamics of agrarian systems, whether they are family-based rural or large-scale agricultural and livestock production, the availability

of natural resources, and the risk of diseases ([RORATO et al., 2023](#)).

2 Metodology

For the elaboration of the work, population data from the Trajetórias Project dataset and climatic data from the Climate Data will be used. Methods of disease transmission based on ordinary differential equations, such as the SIR model, will be addressed. Starting with a simple modeling approach, environmental phenomena such as deforestation and burning will be included to assess how modifications in the ecosystem will interfere with the previously developed model. Computational calculations were performed using the SageMath 9.2 environment, utilizing Scipy's numerical integration functions to solve the method.

First I will be describing SIR ([COELHO, n.d.](#); [PRASAD et al., 2022](#)), which can be considered the foundation of the models that will be used throughout the project. Developed by W. O. Kermack and A. G. McKendrick in 1927, SIR is one of the most widely used models for epidemic modeling, considering three compartments:

S : number of susceptible individuals

I : number of infected individuals

R : number of recovered individuals

In this model, healthy individuals in the S class are susceptible to contact with individuals in the I class and are transferred to this compartment if they contract the disease. Infected individuals can spread the disease through direct contact with susceptible individuals, but they can also become immune over time and are transferred to the R compartment. In general, R includes the total of recovered (immune) individuals and those who died from the disease, but we can assume that the number of deaths is very low compared to the total population size and can be ignored. We also assume that individuals in this category will not revert to being susceptible or infectious.

Considering an epidemic over a short period and that the disease is not fatal, we can ignore vital dynamics of birth and death. With this, we can describe the SIR model through the following system of ordinary differential equations (ODEs):

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta SI}{N} \\ \frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I \\ \frac{dR}{dt} = \gamma I \end{cases}$$

In the model, $N(t) = S(t) + I(t) + R(t)$, i.e., the total population at time t , while β is the infection rate, and γ is the recovery rate. Given that $S + I + R$ is always constant if we ignore birth and death, we have $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$.

For the disease to spread, it is easy to see that $\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I > 0$. Thus, $\frac{\beta SI}{N} > \gamma I \Rightarrow \frac{\beta S}{N} > \gamma$. Assuming we are at the beginning of the infection, given that we want to observe its spread, I will be very small, and $S \approx N$. We then conclude that $\frac{\beta N}{N} > \gamma \Rightarrow \frac{\beta}{\gamma} > 1$.

This dimensionless value can be derived by nondimensionalizing the model: let $y^* = \frac{S}{N}$, $x^* = \frac{I}{N}$, $z^* = \frac{R}{N}$, and $t^* = \frac{t}{1/\gamma} = \gamma t$, so that $y^* + x^* + z^* = 1$. Substituting the system of ODEs above using these values:

$$\begin{cases} \frac{dS}{dt} = \frac{d(y^*N)}{d(t^*/\gamma)} = -\frac{\beta SI}{N} = -\frac{\beta(y^*N)(x^*N)}{N} = -\beta y^*N x^* \\ \frac{dI}{dt} = \frac{d(x^*N)}{d(t^*/\gamma)} = \frac{\beta SI}{N} - \gamma I = \frac{\beta(y^*N)(x^*N)}{N} - \gamma(x^*N) = \beta y^*N x^* - \gamma x^*N \\ \frac{dR}{dt} = \frac{d(z^*N)}{d(t^*/\gamma)} = \gamma I = \gamma(x^*N) \end{cases}$$

Now, canceling the factors N and γ on both sides of the equations:

$$\begin{cases} \frac{d(y^*)}{d(t^*)} = -\frac{\beta y^* x^*}{\gamma} \\ \frac{d(x^*)}{d(t^*)} = \frac{\beta y^* x^*}{\gamma} - x^* \\ \frac{d(z^*)}{d(t^*)} = x^* \end{cases}$$

Thus, we have a system given only by y^* and x^* and the parameter $\frac{\beta}{\gamma}$, which we can call R_0 .

As this work will be primarily focused on malaria modeling, I will now present one of the first models developed specifically for this disease, by Sir Ronald Ross in 1911 (BACAËR, 2011), which uses two distinct ordinary differential equations (ODEs) different from those presented above:

$$\begin{cases} \frac{dI}{dt} = bp'i \frac{N-I}{N} - aI \\ \frac{di}{dt} = bp(n-i) \frac{I}{N} - mI \end{cases}$$

In this case, N is the total human population, $I(t)$ is the number of infected humans at time t , n is the total mosquito population, $i(t)$ is the number of infected mosquitoes at time t , b is the biting rate, p is the probability of transmission from human to mosquito per bite, p' is the probability of transmission from mosquito to human per bite, a is the recovery rate of human infection, and m is the mosquito mortality rate. $bp'ii \frac{N-I}{N} dt - aI dt$ represent, respectively, the number of new infected humans and the number of recovered humans in the interval dt , while $bp(n-i) \frac{I}{N} dt - mI dt$ represent, respectively, the number of new infected mosquitoes and the number of mosquitoes that die in that time interval, assuming that infection does not affect the mosquito mortality rate.

For this model, Ross discussed two equilibrium points, where $\frac{dI}{dt} = \frac{di}{dt} = 0$. They occur when $I = i = 0$, which is the case where there is no malaria, and, for $I, i > 0$, $I = N \frac{1 - amN/(b^2 pp'n)}{1 + aN/(bp'n)}$ and $i = n \frac{1 - amN/(b^2 pp'n)}{1 + m/(bp)}$. Furthermore, for the disease to establish itself, n must be greater than a threshold value $n^* = \frac{amN}{b^2 pp'}$. In this case, the disease becomes endemic. If $n < n^*$, the equilibrium will be at $I = i = 0$, and the disease will disappear.

Dividing the equations of the equilibrium points by $I \times i$, we have:

$$\begin{cases} \frac{bp}{N} = \frac{bpn}{Ni} - \frac{m}{I} \\ \frac{bp'}{N} = \frac{bp'}{I} - \frac{a}{i} \end{cases}$$

Which transforms the problem into a linear system with two unknowns, I and i .

Now, I will present the model that will be used for the development of the work, based on the one developed by Paul E. Parham and Edwin Michael in 2010, which takes into account factors such as rainfall and temperature (R and T , respectively) (PARHAM; MICHAEL, 2010).

Defining the equations that will be used:

$$\left\{ \begin{array}{l} \frac{dS_H}{dt} = -ab_2 \left(\frac{I_M}{N} \right) S_H \\ \frac{dI_H}{dt} = ab_2 \left(\frac{I_M}{N} \right) S_H - \gamma I_H \\ \frac{dR_H}{dt} = \gamma I_H \\ \frac{dS_M}{dt} = b - ab_1 \left(\frac{I_H}{N} \right) S_M - \mu S_M \\ \frac{dE_M}{dt} = ab_1 \left(\frac{I_H}{N} \right) S_M - \mu E_M - ab_1 \left(\frac{I_H}{N} \right) S_M l(\tau_M) \\ \frac{dI_M}{dt} = ab_1 \left(\frac{I_H}{N} \right) S_M l(\tau_M) - \mu I_M \end{array} \right.$$

It is necessary to mention that the original model used $I_M(t - \tau)$ in $\frac{dI_H}{dt}$ and $I_H(t - \tau)$ in $\frac{dE_M}{dt}$ (in the transition from E to I) and $\frac{dI_M}{dt}$, respectively. However, as this would make the model based on delay differential equations, it was recommended by the advisor to disregard this difference and use only the current time t .

Having the model equations for the human and mosquito populations, I will first define the parameters used in the modeling and other necessary functions, and then the variables used:

Parameter	Definition	Formula
$T(t)$	Temperature	$T_1(1 + T_2 \cos(\omega_1 t - \phi_1))$
$R(t)$	Precipitation	$R_1(1 + R_2 \cos(\omega_2 t - \phi_2))$
$b(R, T)$	Mosquito birth rate (/ day)	$\frac{B_{EP_E}(R)p_L(R, T)p_P(R)}{(\tau_E + \tau_L(T) + \tau_P)}$
$a(T)$	Biting rate (/day)	$\frac{(T - T_1)}{D_1}$
$\mu(T)$	Mosquito mortality rate per capita (/ day)	$-\log(p(T))$
$\tau_M(T)$	Duration of the sporozoite cycle (days)	$\frac{DD}{(T - T_{min})}$
$\tau_L(T)$	Duration of larval development phase (days)	$\frac{1}{c_1 T + c_2}$
$p(T)$	Daily mosquito survival rate	$e^{(-1/(AT^2+BT+C))}$
$p_L(R)$	Probability of larval survival dependent on rainfall	$(\frac{4p_{ML}}{R_L^2})R(R_L - R)$
$p_L(T)$	Probability of larval survival dependent on temperature	$e^{-(c_1 T + c_2)}$
$p_L(R, T)$	Probability of larval survival dependent on temperature and rainfall	$p_L(R)p_L(T)$
$l(\tau_M)(T)$	Probability of mosquito survival during the sporozoite cycle (/ day)	$p(T)^{\tau_M(T)}$
$M(t)$	Total number of mosquitoes	$S_M(t) + E_M(t) + I_M(t)$
$N(t)$	Total number of humans	$S_H(t) + I_H(t) + R_H(t)$

Table 1 – Parameters used in the modeling

Parameter	Definition
b_1	Proportion of bites from susceptible mosquitoes on infected humans that result in infection
b_2	Proportion of bites from infected mosquitoes on susceptible humans that result in infection
γ	1/Average duration of infectiousness in humans (days^{-1})
T_1	Mean temperature in the absence of seasonality ($^{\circ}\text{C}$)
T_2	Amplitude of seasonal variability in temperature
R_1	Average monthly precipitation in the absence of seasonality (mm)
R_2	Amplitude of seasonal variability in precipitation
ω_1	Angular frequency of seasonal oscillations in temperature (months^{-1})
ω_2	Angular frequency of seasonal oscillations in precipitation (months^{-1})
ϕ_1	Phase lag of temperature variability (phase shift)
ϕ_2	Phase lag of precipitation variability (phase shift)
B_E	Number of eggs laid per adult per oviposition
p_{ME}	Maximum probability of egg survival
p_{ML}	Maximum probability of larval survival
p_{MP}	Maximum probability of pupal survival
τ_E	Duration of the egg development phase (days)
b_3^*	Infection rate in exposed mosquitoes ($1/\tau_M(T)$)

Table 2 – Parameters used in the modeling

Parameter	Definition
τ_P	Duration of the pupal development phase (days)
R_L	Rainfall threshold until breeding sites are eliminated, removing immature individuals (mm)
T_{min}	Minimum temperature, below which there is no development of the parasite: 14.5 ($^{\circ}C$)
DD	Degree-days for parasite development. Number of degrees by which the daily average temperature exceeds the minimum development temperature. "Sum of heat" for maturation: 105 ($^{\circ}C$ days)
A	Empirical sensitivity parameter ($^{\circ}C^2$ days) $^{-1}$
B	Empirical sensitivity parameter ($^{\circ}C$ days) $^{-1}$
C	Empirical sensitivity parameter (days $^{-1}$)
D_1	Constant: 36.5 ($^{\circ}C$ days)
c_1	Empirical sensitivity parameter ($^{\circ}C$ days) $^{-1}$
c_2	Empirical sensitivity parameter (days $^{-1}$)
T'^*	Empirical temperature parameter ($^{\circ}C$)

Table 3 – Parameters used in the modeling

Parameters marked with * were added during the development of the modeling to correct inaccuracies derived from the original equations in the reference article. The definition of DD was taken from (MCCORD, 2016) and (DETINOVA; BERTRAM; ORGANIZATION, 1962).

Having the equations and parameters, the modeling was initially done using data from the rural area of Manaus, in the period from 2004 to 2008, which were selected due to the higher incidence of malaria cases caused by *P. vivax*, the species responsible for the highest number of cases in Brazil (OLIVEIRA-FERREIRA et al., 2010; CODEÇO et al., 2021). Using the incidence function used in the Trajetorias project (RORATO et al., 2023), we have:

$$\text{Inc}(d, m, z, t_1, t_2) = \frac{\text{Cases}(d, m, z, t_1, t_2)}{\text{Pop}(m, z, (t_1 + t_2)/2) \times 5 \text{ years}} \times 10^5,$$

where $\text{Cases}(d, m, z, t_1, t_2)$ is the number of cases of disease d in zone z of municipality m ,

and t_1 and t_2 are the initial and final years of the interval, while $\text{Pop}(m, z, (t_1 + t_2)/2) \times 5$ years is the population in zone z of municipality m in the middle of the period multiplied by the total number of observation years. In this case, we could indicate as:

$$\text{Inc}(\text{Vivax}, \text{Manaus}, \text{Rural}, 2004, 2008) = \frac{\text{Cases}(\text{Vivax}, \text{Manaus}, \text{Rural}, 2004, 2008)}{\text{Pop}(\text{Manaus}, \text{Rural}, 2006) \times 5 \text{ years}} \times 10^5$$

$$184030.8 = \frac{78745}{5\text{Pop}} \times 10^5 \Rightarrow \text{Pop} \approx 8558$$

Using data on the total population of Manaus in this period, with an incidence of 3106.4 and a number of cases of 262264, the total population of the municipality was estimated to be 1688540 inhabitants. Thus, the rural population could be considered as approximately 0.5% of the municipality's population.

Having estimated the percentage size of the rural population in the city, it was possible to calculate this population for each of the years of the analysis through linear interpolation using historical series data from IBGE ([IBGE, n.d.](#)):

Year	Estimated rural population
2004	7717
2005	7889
2006	8061
2007	8233
2008	8492
2009	8751

Table 4 – Manaus' rural population from 2004 to 2009

As there were population data for the years 2000, 2007, and 2010, interpolations were performed with different initial and final points, using data from 2000 to 2007 for 2004-2007 and from 2007 to 2010 for 2008-2009, ensuring the correct use of the 2007 population.

Now, describing a bit of the theory behind environmental factors, according to ([NORRIS, 2004](#)), the removal of tree canopies allowed the resurgence of malaria in South America. In deforested areas, without tree canopies covering the ground, water puddles under sunlight

attract mosquitoes of the species *Anopheles darlingi*, the main vector related to human malaria in the Amazon (RONDÔNIA, n.d.). They are usually less commonly found in still intact forests. This is because light and heat favor the development of larvae and pupae, in addition to a greater availability of algae for larval feeding (SILVA-NUNES, 2010). The increase in ambient temperature also favors the vectorial capacity of mosquitoes. Deforestation also attracts and brings humans closer to take part in logging, agriculture, and road construction activities, bringing individuals infected with *Plasmodium* to an area where both the vector and the environment have already been modified to favor transmission. Furthermore, agriculture also promotes river sedimentation, providing suitable environments for breeding sites. Therefore, it can be considered a relevant change for the model to take into account deforestation, the increase in survival probabilities of eggs, larvae, and pupae, as well as increasing the proportion of bites that lead to infection, due to the increased human population density in areas near mosquito breeding sites.

3 Results

Analyzing the results obtained with the original parameters from the article by Parham and Michael ($T_1 = 23.2$, $T_2 = 0.07$, $\omega_1 = 0.67$, $\phi_1 = 1.53$, $R_1 = 85.9$, $R_2 = 0.98$, $\omega_2 = 0.65$, $\phi_2 = 1.99$, $A = -0.03$, $B = 1.31$, $C = -4.4$, $b_1 = 0.04$, $b_2 = 0.09$, $T_{min} = 14.5$, $\gamma = 1/120$, $R_L = 50$, $c_1 = 0.00554$, $c_2 = -0.06737$ (PARHAM; MICHAEL, 2010), (OKUNEYE; GUMEL, 2017)), and using the previously estimated average population and an arbitrary value for the mosquito population, of 10000, assuming 1000 infected humans and 5000 exposed mosquitoes at $t = 0$, the modeling is as follows ¹:

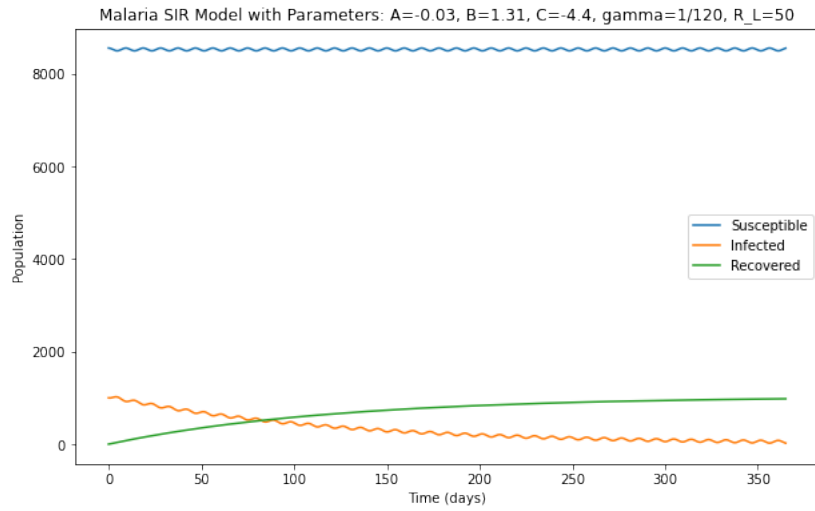


Figure 1 – SIR with original parameters

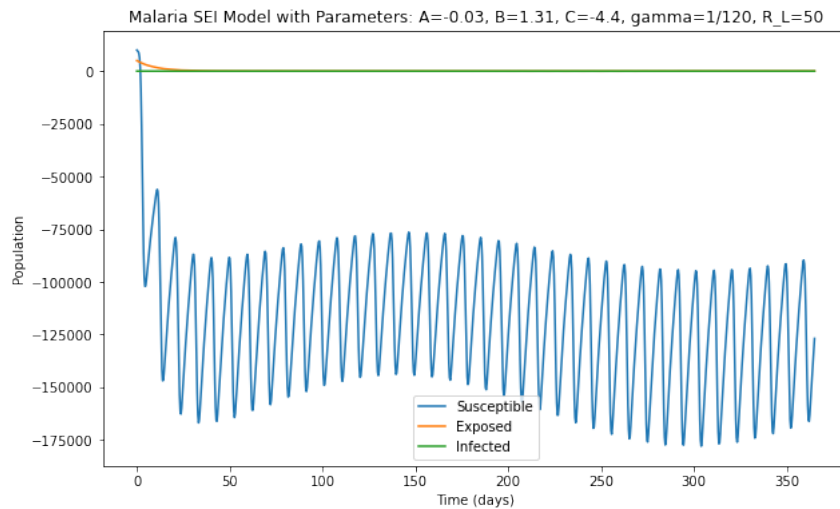


Figure 2 – SEI with original parameters

¹ The development of the model with the original data can be found at https://github.com/RaphaLevy/Undergraduate_Dissertation/blob/main/modeling_files/Original_Parameters.ipynb.

With this initial modeling, a strong oscillation in the number of susceptible humans and mosquitoes, as well as infected humans, is noticeable. Furthermore, it is evident that with these parameters, the epidemic will not stabilize, as the number of infected humans tends to 0 throughout the year, while the population of susceptible mosquitoes becomes negative, and the population of exposed and infected individuals also tends to 0. These effects were characterized by temperature and precipitation oscillating in very short periods of time, attributed to a high value of ω for both functions.

Starting with only a single infected human and exposed mosquito, the oscillating population isn't noticeable in the SIR plot, however the mosquito population still becomes negative.

Now, the first necessary modification is to correct the temperature and precipitation to consider data from Manaus, as the original paper uses data from Tanzania. So, collecting climatological data from Manaus from ([CLIMATEDATA.ORG](https://climatedata.org), n.d.), the average temperature and precipitation were estimated as 26.4°C and 250.083 mm, respectively. With this data, the amplitude of seasonal variability, angular frequency, and phase lag of variability for both were defined to approximate the real values:

Parameter	Value
T_1	26.4°C
T_2	0.025
ω_1	$0.017 \text{ (meses)}^{-1}$
ϕ_1	-1.45
R_1	250.083 mm
R_2	0.565
ω_2	$0.02 \text{ (meses)}^{-1}$
ϕ_2	1.6

Table 5 – Values for climatic parameters

The amplitude parameters (T_2 and R_2) and phase lag parameters (ϕ_1 and ϕ_2) are dimensionless. The temperature and precipitation throughout the year then evolve as

follows ²:

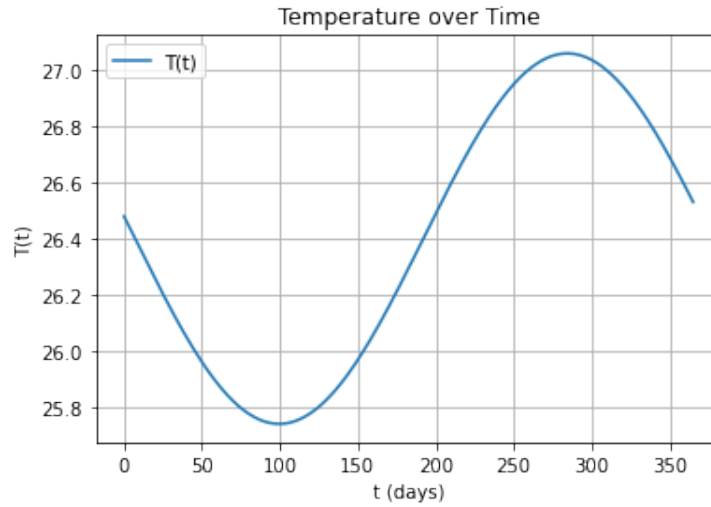


Figure 3 – Temperature graph

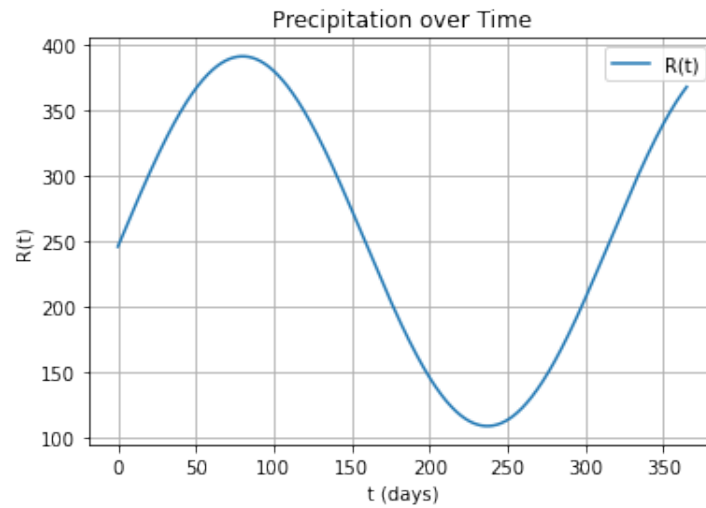


Figure 4 – Precipitation graph

In order to ensure the correctness of the function with the parameters used, I calculated the temperature values for the months of October and May, which are the hottest and coldest months of the year with average temperatures of 27.6°C and 25.8°C , respectively. Additionally, I calculated the precipitation values in March and August, which are the months with the highest and lowest precipitation, with 395 mm and 114 mm, respectively. The obtained average values were 27.06°C , 25.86°C , 390.67 mm, and 112.89 mm.

With the temperature and precipitation parameters ready for an initial analysis, the

² The development of the model with the original data can be found at https://github.com/RaphaLevy/Undergraduate_Dissertation/blob/main/modeling_files/Adapting_T_and_R.ipynb.

evolution of human and mosquito populations was verified. The results can be found in Appendices 1 and 2 ³.

Notably, this result is still incorrect, as the mosquito population still becomes negative over time, and the human population also turns negative near the middle portion of the year. However, the daily oscillations were indeed eliminated, due to the modifications made to ω_1 and ω_2 . Another parameter that should also be adapted in consideration for our new climatic values was R_L , the rainfall limit beyond which breeding sites get flushed out and no immature stages survive. In the original paper, R_L was defined as 50 mm, which is too low for the high precipitation in the Amazon, which has a maximum of almost 400 mm in March. Because of this, I increased R_L from 50 mm to 450 mm. The results can be found in Appendices 3 and 4 ⁴.

Com essas modificações, a evolução da população humana parece mais viável do que estava anteriormente, com o número de infectados inicialmente baixo, aumentando ao longo do ano e posteriormente decaindo. Por outro lado, é possível notar que próximo do tempo final de análise, o número de humanos infectados se torna negativo. A modelagem de mosquitos, por sua vez, se manteve relativamente estável.

Analizando o comportamento da modelagem de mosquitos de forma que a população ficasse aproximadamente constante ao longo do período, dadas as equações diferenciais do modelo SEI, e os parâmetros passados para atingir esse objetivo, o valor de μ passado fica muito próximo de 0, enquanto que $l(\tau_M)$, uma probabilidade, fica muito próxima de 1. Por isso, $\frac{dE_M}{dt}$ também fica bem próximo de 0, fazendo com que a função de expostos seja linear, aproximadamente constante no número inicial de infectados, enquanto que a população de mosquitos que sai do compartimento de suscetíveis quase que simultaneamente entra no compartimento de infectados, causando as ondulações espelhadas de S e I . Para contornar esse efeito, foi necessário modificar o uso de b_1 , para passar apenas mosquitos do compartimento S para E , necessitando da inclusão de um novo parâmetro, b_3 , para passar mosquitos do compartimento E para I . Essa taxa é inversa ao período de incubação, então definimos

$$b_3 = \frac{T - T_{min}}{DD}$$

Ademais, foi removido o parâmetro $a(T)$ na passagem de mosquitos expostos para infectados, visto que nessa mudança do compartimento E para I não ocorrem novas picadas, assim como o parâmetro T_1 utilizado na fórmula de $a(T)$ também teve de ser modificado,

³ The development of the graphs above can be found at https://github.com/RaphaLevy/Undergraduate_Dissertation/blob/master/modeling_files/Adapting_T_and_R.ipynb.

⁴ The development of the graphs above can be found at https://github.com/RaphaLevy/Undergraduate_Dissertation/blob/master/modeling_files/Adapting_T_and_R.ipynb.

visto que o artigo original aplica T_1 com dois valores diferentes, para a taxa de picadas e para a equação da temperatura por tempo. Com o intuito de manter a formatação de $T(t)$ e $R(t)$ a mesma, T_1 de a foi modificado para T' .

Considerando então o que foi dito acima, também se tornou necessário modificar as equações diferenciais do SEI, que ficaram dessa forma:

$$\begin{cases} \frac{dS_M}{dt} = b - ab_1 \left(\frac{I_H}{N} \right) S_M - \mu S_M \\ \frac{dE_M}{dt} = ab_1 \left(\frac{I_H}{N} \right) S_M - \mu E_M - b_3 E_M l \\ \frac{dI_M}{dt} = b_3 E_M l - \mu I_M \end{cases}$$

Com as adaptações feitas à transmissão da doença entre os mosquitos (Apêndices 5 e 6), é possível ver que não só o equilíbrio mudou, de forma que agora o número de expostos tende a 0, e não o de infectados, podemos ver também que a população de humanos é fortemente impactada, novamente oscilando constantemente, e com o número de infectados ficando negativo repetidas vezes ⁵. Analisando modificações nos parâmetros empíricos citados acima, foi notado que esse comportamento se dá devido em especial ao alto valor em módulo de A , visto que com $A = -217.925$, o comportamento do modelo foi similar. Até mesmo usando $A = 17.925$, o comportamento observado foi similar. Contudo, para $A = 0$, a população humana teve um comportamento bem mais viável, enquanto que a população de mosquitos tendeu à extinção (Apêndices 7 e 8). ⁶

Analisando o efeito de A , notou-se que, para A muito grande, μ se torna extremamente pequeno:

Para $A = 317.925$, $\mu = 4.505961269611858e - 06$

Para $A = 17.925$, $\mu = 7.78802370175753e - 05$

Para $A = -217.925$, $\mu = -6.599014101979344e - 06$

Para $A = 0$, $\mu = 0.0028800184321179124$

Com isso, a mortalidade de mosquitos será extremamente baixa para valores grandes de A , positivos ou não. Contudo, como uma forma de possibilitar o uso de valores altos para

⁵ Observação: as modelagens do SIR mostradas ao longo de 5 anos de análise foram feitas usando o valor médio estimado para a população no período entre 2004 e 2008, com $N=8558$

⁶ A elaboração dos gráficos indicados acima pode ser encontrada em https://github.com/RaphaLevy/TCC/blob/main/Discuss%C3%A3o/Correcao_de_Modelagens_2.ipynb

esse parâmetro, e ainda garantir que as populações tenham valores sempre não-negativos, foi aplicado um máximo nas taxas com subtração, segundo recomendação do orientador, de forma que o valor resultante entre $T(t) - T_{min}$, $R_L - R(t)$ e $T' - T(t)$ seja o máximo entre essas diferenças e uma tolerância pequena, no caso foi utilizado um $\epsilon = 10^{-5}$. Isso foi suficiente para garantir que, mesmo com valores grandes de A , o modelo não tomasse valores negativos (Apêndices 9 a 12).

Com essas modificações, é possível notar como, independente do valor de A , a evolução das populações humanas será bem similar, com uma estabilização da população de suscetíveis logo antes de se tornar 0. A de mosquitos, por sua vez, é menos oscilante para valores grandes de A , e não se estabiliza mesmo em mais de 10000 dias. Por outro lado, se $A = 0$, a população de mosquitos se aproxima da extinção, com pequenos picos de mosquitos unicamente suscetíveis, sem estabilização da doença. Contudo, ainda que usando o máximo entre um pequeno ϵ e as diferenças notadas acima, $T' = 26.4$ é um valor menor que a temperatura máxima calculada para o ano, que é próxima de 27.1. Assim, foi recomendado pelo orientador que fosse testado um valor maior que esse máximo, no caso $T' = 27.4$ (Apêndice 13 e 14).

Tendo então o modelo SIR/SEI devidamente corrigido, foi possível partir para as análises de aplicação do desmatamento. Para isso, iniciei com o cálculo do \mathcal{R}_0 para ambos SIR e SEI, e também para o modelo acoplado, usando como referência a formulação de P. van den Driessche ([VAN DEN DRIESSCHE; WATMOUGH, 2002](#)):

Definimos X_s como o conjunto de todos os estados livres de doença,

$$X_s = \{x \geq 0 | x_i = 0, i = 1, \dots, m\},$$

onde $X = (x_1, \dots, x_n)^T$, tal que $x_i \geq 0$ seja o número de indivíduos em cada compartimento, e supomos cada função continuamente diferenciável pelo menos duas vezes em cada variável (C^2).

Agora, reordenamos as equações para que as m primeiras sejam as que contém infectados. Seja $\mathcal{F}_i(x)$ a taxa de aparecimento de novas infecções no compartimento i , $\mathcal{V}_i^+(x)$ a taxa de entrada de indivíduos no compartimento i por outros meios e $\mathcal{V}_i^-(x)$ a taxa de saída de indivíduos do compartimento i . O modelo de transmissão da doença consiste em condições iniciais não negativas juntamente com o seguinte sistema de equações:

$$\dot{x} = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x), i = 1, \dots, n,$$

em que $\mathcal{V}_i(x) = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)$. Definimos também $F = \left[\frac{\partial \mathcal{F}_i(x_0)}{\partial x_j} \right]$ e $V = \left[\frac{\partial \mathcal{V}_i(x_0)}{\partial x_j} \right]$, onde x_0 é um DFE (Equilíbrio livre de doença) e $1 \leq i, j \leq m$.

Isto equivale à jacobiana destas duas matrizes, após substituir x_0 ou seja, $S = 1$. R_0 será dado por $\rho(FV^{-1})$, ou seja, será o raio espectral da matriz FV^{-1} . Com as definições necessárias, podemos calcular o R_0 de ambos os modelos como a seguir:

- **SIR:** Nesse caso, $m = 1$, e nossos compartimentos serão colocados da forma $[I_H, S_H, R_H]$. Como o R_0 é calculado com valores normalizados, multiplicaremos as equações necessárias por N para remover o denominador, e especificamente no caso do SIR, como R_H não é utilizado em nenhuma das equações, podemos apenas fazer em função de S e I . Portanto:

$\mathcal{F}_i(x)$: taxa de surgimento de novos infectados no compartimento i

$$\mathcal{F} = [ab_2 I_H S_H]$$

Além disso, temos

$\mathcal{V}_i(x)^-$: taxa de saída do compartimento i

$\mathcal{V}_i(x)^+$: taxa de entrada do compartimento i

Logo:

$$\mathcal{V}^- = [\gamma I_H]$$

$$\mathcal{V}^+ = [0]$$

$$\mathcal{V}_i(x) = \mathcal{V}_i(x)^- - \mathcal{V}_i(x)^+$$

Então,

$$\mathcal{V} = [\gamma I_H]$$

Portanto

$$F = \frac{\partial \mathcal{F}}{\partial I_H} = [ab_2 S_H]$$

$$V = \frac{\partial \mathcal{V}}{\partial I_H} = [\gamma]$$

No equilíbrio, $[S_H^*, I_H^*] = [1, 0]$, então $F = [ab_2]$, $V = [\gamma]$ e $R_0 = \left| \frac{ab_2}{\gamma} \right|$.

- **SEI:** Nesse caso, $m = 2$, e nossos compartimentos serão colocados da forma $[E_M, I_M, S_M]$. Novamente multiplicaremos as equações necessárias por N para remover o denominador. Portanto:

$$\mathcal{F} = \begin{bmatrix} ab_1 I_H S_M \\ 0 \end{bmatrix}$$

$$\mathcal{V}^- = \begin{bmatrix} E_M(\mu + b_3 l) \\ \mu I_M \end{bmatrix}$$

$$\mathcal{V}^+ = \begin{bmatrix} 0 \\ b_3 E_M l \end{bmatrix}$$

$$\mathcal{V}_i(x) = \mathcal{V}_i(x)^- - \mathcal{V}_i(x)^+$$

Então,

$$\mathcal{V} = \begin{bmatrix} E_M(\mu + b_3 l) \\ \mu I_M - b_3 E_M l \end{bmatrix}$$

Portanto

$$F = \frac{\partial \mathcal{F}}{\partial E_M, I_H} = \begin{bmatrix} \frac{\partial ab_1 I_H S_M}{\frac{\partial E_M}{\partial 0}} & \frac{\partial ab_1 I_H S_M}{\frac{\partial I_H}{\partial 0}} \\ \frac{\partial E_M}{\partial E_M} & \frac{\partial I_H}{\partial I_H} \end{bmatrix} = \begin{bmatrix} 0 & ab_1 S_M \\ 0 & 0 \end{bmatrix}$$

$$V = \frac{\partial \mathcal{V}}{\partial E_M, I_M} = \begin{bmatrix} \frac{\partial E_M(\mu + b_3 l)}{\frac{\partial \mu I_M - b_3 E_M l}{\partial E_M}} & \frac{\partial E_M(\mu + b_3 l)}{\frac{\partial \mu I_M - b_3 E_M l}{\partial I_M}} \\ \frac{\partial E_M}{\partial E_M} & \frac{\partial I_M}{\partial I_M} \end{bmatrix} = \begin{bmatrix} \mu + b_3 l & 0 \\ -b_3 l & \mu \end{bmatrix}$$

No equilíbrio, $[S_M^*, E_M^*, I_M^*] = [1, 0, 0]$, então

$$F = \begin{bmatrix} 0 & ab_1 \\ 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \mu + b_3 l & 0 \\ -b_3 l & \mu \end{bmatrix}$$

$$\text{e } R_0 = \left| \frac{ab_1 b_3 l}{(b_3 l + \mu)\mu} \right|.$$

- **SIR/SEI:** Nesse caso, $m = 3$, e nossos compartimentos serão colocados da forma $[I_H, E_M, I_M, S_H, S_M]$. Novamente multiplicaremos as equações necessárias por N para remover o denominador. Portanto:

$$\mathcal{F} = \begin{bmatrix} ab_2 I_M S_H \\ ab_1 I_H S_M \\ 0 \end{bmatrix}$$

$$\mathcal{V}^- = \begin{bmatrix} \gamma I_H \\ E_M(\mu + b_3 l) \\ \mu I_M \end{bmatrix}$$

$$\mathcal{V}^+ = \begin{bmatrix} 0 \\ 0 \\ b_3 E_M l \end{bmatrix}$$

$$\mathcal{V}_i(x) = \mathcal{V}_i(x)^- - \mathcal{V}_i(x)^+$$

Então,

$$\mathcal{V} = \begin{bmatrix} I_H \gamma \\ E_M(\mu + b_3 l) \\ \mu I_M - b_3 E_M l \end{bmatrix}$$

Portanto

$$F = \frac{\partial \mathcal{F}}{\partial I_H, E_M, I_M} = \begin{bmatrix} \frac{\partial ab_2 I_M S_H}{\partial ab_1 I_H S_M} & \frac{\partial ab_2 I_M S_H}{\partial ab_1 I_H S_M} & \frac{\partial ab_2 I_M S_H}{\partial ab_1 I_H S_M} \\ \frac{\partial I_H}{\partial 0} & \frac{\partial E_M}{\partial 0} & \frac{\partial I_M}{\partial 0} \\ \frac{\partial I_H}{\partial I_H} & \frac{\partial E_M}{\partial E_M} & \frac{\partial I_M}{\partial I_M} \end{bmatrix} = \begin{bmatrix} 0 & 0 & ab_2 S_H \\ ab_1 S_M & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V = \frac{\partial \mathcal{V}}{\partial I_H, E_M, I_M} = \begin{bmatrix} \frac{\partial \gamma I_H}{\partial E_M(\mu + b_3 l)} & \frac{\partial \gamma I_H}{\partial E_M(\mu + b_3 l)} & \frac{\partial \gamma I_H}{\partial E_M(\mu + b_3 l)} \\ \frac{\partial I_H}{\partial \mu I_M - b_3 E_M l} & \frac{\partial E_M}{\partial \mu I_M - b_3 E_M l} & \frac{\partial I_M}{\partial \mu I_M - b_3 E_M l} \\ \frac{\partial I_H}{\partial I_H} & \frac{\partial E_M}{\partial E_M} & \frac{\partial I_M}{\partial I_M} \end{bmatrix} = \begin{bmatrix} \gamma & 0 & 0 \\ 0 & b_3 l + \mu & 0 \\ 0 & -b_3 l & \mu \end{bmatrix}$$

No equilíbrio, $[S_H^*, S_M^*, I_H^*, E_M^*, I_M^*] = [1, 1, 0, 0, 0]$, então

$$F = \begin{bmatrix} 0 & 0 & ab_2 \\ ab_1 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \gamma & 0 & 0 \\ 0 & b_3 l + \mu & 0 \\ 0 & -b_3 l & \mu \end{bmatrix}$$

$$\text{e } R_0 = \left| \sqrt{\frac{a^2 b_1 b_2 b_3 l}{(b_3 l + \mu) \gamma \mu}} \right| = \sqrt{\mathcal{R}_{0SIR} \times \mathcal{R}_{0SEI}}.$$

Tendo obtido a fórmula fechada de \mathcal{R}_0 , foi possível calcular o seu valor para o modelo atual, apresentado nos apêndices 13 e 14. No caso, $\mathcal{R}_0 = 88.16804666190774$ e a taxa de picadas no tempo $t = 0$ foi de 0.025218306088151666, ou seja, aproximadamente uma picada por mosquito a cada 40 dias. Notavelmente, esses são valores muito altos para o número de reprodução de uma doença, nesse caso indicando que um indivíduo poderia infectar outros 88. Sendo assim, seria necessário modificar os parâmetros para que o valor de \mathcal{R}_0 ficasse próximo de 1, para que seja mais fácil analisar como pequenas modificações nos parâmetros causariam a extinção ou a continuação da doença. Antes disso, porém, foi necessário mais uma vez corrigir as equações do SEI, já que a probabilidade diária de sobrevivência de mosquitos durante o ciclo de esporozoítos (l), não é diretamente associada à taxa de infecção dos expostos, e também não é parte da taxa de entrada de novos indivíduos no compartimento I . Outra coisa que teve de ser corrigida foi a fórmula da taxa

de picadas (a), que voltou a ser $\frac{(T(t) - T')}{D_1}$ como originalmente. As equações corrigidas do SEI ficaram como a seguir:

$$\begin{cases} \frac{dS_M}{dt} = b - ab_1\left(\frac{I_H}{N}\right)S_M - \mu S_M \\ \frac{dE_M}{dt} = ab_1\left(\frac{I_H}{N}\right)S_M - \mu E_M - b_3 E_M - l E_M \\ \frac{dI_M}{dt} = b_3 E_M - \mu I_M \end{cases}$$

Tendo corrigido as equações, o \mathcal{R}_0 do SEI e do modelo acoplado foram $\left| \frac{ab_1 b_3}{(b_3 + l + \mu)\mu} \right|$ e $\left| \sqrt{\frac{a^2 b_1 b_2 b_3}{(b_3 + l + \mu)\gamma\mu}} \right|$, respectivamente. Podemos verificar que \mathcal{R}_0 é de fato adimensional: a , b_3 , l , μ e γ são funções de unidade 1/dia, enquanto b_1 e b_2 são adimensionais. Sendo assim, \mathcal{R}_0 do SIR tem dimensão (1/dia)/(1/dia), \mathcal{R}_0 do SEI tem dimensão (1/dia²)/(1/dia²) e o acoplado tem dimensão (1/dia³)/(1/dia³).

Novamente calculando o \mathcal{R}_0 do modelo atual, seu valor para o modelo acoplado foi de 63.745319442750855, o que já é menor que o encontrado previamente, mas ainda muito alto. Foi necessário analisar que parâmetros poderiam ser modificados de forma que \mathcal{R}_0 se aproximasse de 1 tanto para o SIR quanto para o SEI, já que pelas equações é possível aproximar o valor acoplado de 1 enquanto que o valor de um dos outros dois modelos fosse menor que 1, fazendo com que a doença não se estabilizasse ou a população tendesse à extinção, no caso do SEI.

Dessa maneira, as modificações ideais para aproximar \mathcal{R}_0 de 1 foram as seguintes:

$$T' = 27.4 \Rightarrow 25.6$$

$$D_1 = 36.5 \Rightarrow 55$$

$$A = 317.925 \Rightarrow 15$$

$$b_2 = 0.3 \Rightarrow 0.2$$

$$\gamma = 1/1825 \Rightarrow 1/365$$

Com essas adaptações,

$$\mathcal{R}_0 \text{ SIR} = 1.167378607783994$$

$$\mathcal{R}_0 \text{ SEI} = 1.7805860145371295$$

$$\mathcal{R}_0 \text{ SIR/SEI} = 1.4417413161486372$$

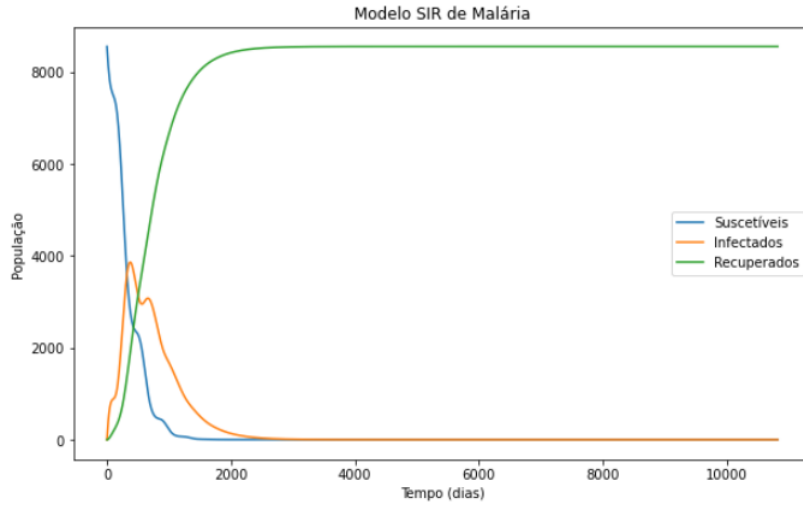


Figure 5 – SIR com $T' = 25.6^{\circ}C$, $A = 15 (^{\circ}C^2 \text{ dias})^{-1}$, $D_1 = 55 (^{\circ}C \text{ dias})$, $b_2 = 0.2$, $\gamma = 1/365$

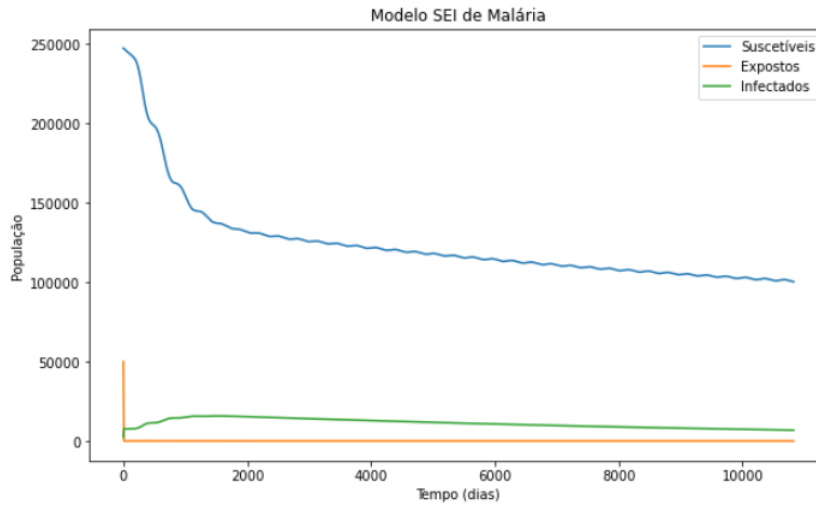


Figure 6 – SEI com $T' = 25.6^{\circ}C$, $A = 15 (^{\circ}C^2 \text{ dias})^{-1}$, $D_1 = 55 (^{\circ}C \text{ dias})$, $b_2 = 0.2$, $\gamma = 1/365$

Discutindo essa aproximação de \mathcal{R}_0 a 1 com o orientador do Trabalho, foi decidido que ao invés de aumentar o valor D_1 , um parâmetro empírico usado para estabilizar a taxa de picadas, seria ideal modificar b_1 e b_2 , que são as proporções de picadas gerando infecção em mosquitos e humanos suscetíveis, respectivamente, tendo em vista que, conforme temos mais ocorrências de áreas desmatadas, haverá um maior contato entre humanos e mosquitos, aumentando a proporção de picadas gerando infecção.

Junto com essa correção, também foram modelados os gráficos de evolução das taxas utilizadas, em função da temperatura e precipitação, ao invés do tempo, para que fosse possível analisar o comportamento dessas taxas conforme a temperatura e precipitação variam. Os gráficos estão indicados no apêndice, partindo do 15. Gerando esses gráficos, também foi percebido que seria ideal aumentar R_L do valor atual de 312 mm para 450

mm, para evitar que a probabilidade de sobrevivência de mosquitos durante as diferentes fases se tornasse muito próximo de 0, o que estava afetando a taxa de nascimentos $b(R, T)$, e diminuir γ de 1/365 dias para 1/120, valor original do artigo de referência (PARHAM; MICHAEL, 2010), de forma que a curva epidêmica fosse mais próxima do analisado na realidade, visto na Figura 5 que a curva de infectados começa a crescer logo no início da análise, e a infecção só deixa de ocorrer após mais de 5 anos.

Após o desenvolvimento dos gráficos das taxas, foi iniciada a modelagem usando os dados “reais” da população obtidos através da interpolação dos dados de população de Manaus indicada na Tabela 4. Com os valores previamente obtidos, a taxa anual de nascimentos foi estimada como sendo de 206.8 nascimentos por ano. Sendo assim, são 0.56657 nascimentos por dia, aproximadamente, e 0.00007 nascimentos diários por pessoa, já que a população rural média em Manaus é de aproximadamente 8078.5 pessoas entre 2004 e 2008.

Assumindo que a taxa de natalidade e mortalidade de humanos é a mesma, foi então incluído na modelagem o parâmetro μ_H , com valor 0.00007, representando a taxa diária de nascimentos e mortes. O modelo atualizado ficou como a seguir:

$$\begin{cases} \frac{dS_H}{dt} = \mu_H N - ab_2 \left(\frac{I_M}{N} \right) S_H - \mu_H S_H \\ \frac{dI_H}{dt} = ab_2 \left(\frac{I_M}{N} \right) S_H - \gamma I_H - \mu_H I_H \\ \frac{dR_H}{dt} = \gamma I_H - \mu_H R_H \end{cases}$$

A elaboração dos gráficos, a partir do Apêndice 27, foi feita em ⁷.

Tendo corrigido as equações, o \mathcal{R}_0 do SIR e do modelo acoplado foram $\left| \frac{ab_2}{\gamma + \mu_H} \right|$ e $\left| \sqrt{\frac{a^2 b_1 b_2 b_3}{(b_3 + l + \mu)(\gamma + \mu_H)\mu}} \right|$, respectivamente. Podemos verificar que \mathcal{R}_0 é de fato adimensional: a , b_3 , l , μ e γ são funções de unidade 1/dia, enquanto b_1 e b_2 são adimensionais. Sendo assim, \mathcal{R}_0 do SIR tem dimensão (1/dia)/(1/dia), \mathcal{R}_0 do SEI tem dimensão (1/dia²)/(1/dia²) e o acoplado tem dimensão (1/dia³)/(1/dia³).

Como esperado, com \mathcal{R}_0 menor que 1, e apenas 1 mosquito exposto e 1 humano infectado, a doença não consegue se estabelecer, como pode ser visto abaixo:

Dos apêndices 27 a 30, aumentando o número de infectados e expostos, inicialmente com um “pequeno” aumento, com 1/30 da população de mosquitos exposta à doença, e 6.5% de

⁷ https://github.com/RaphaLevy/TCC/blob/main/Modelagem_com_Dinamica_Pop/Modelagem_com_Entrada_Populacional.ipynb

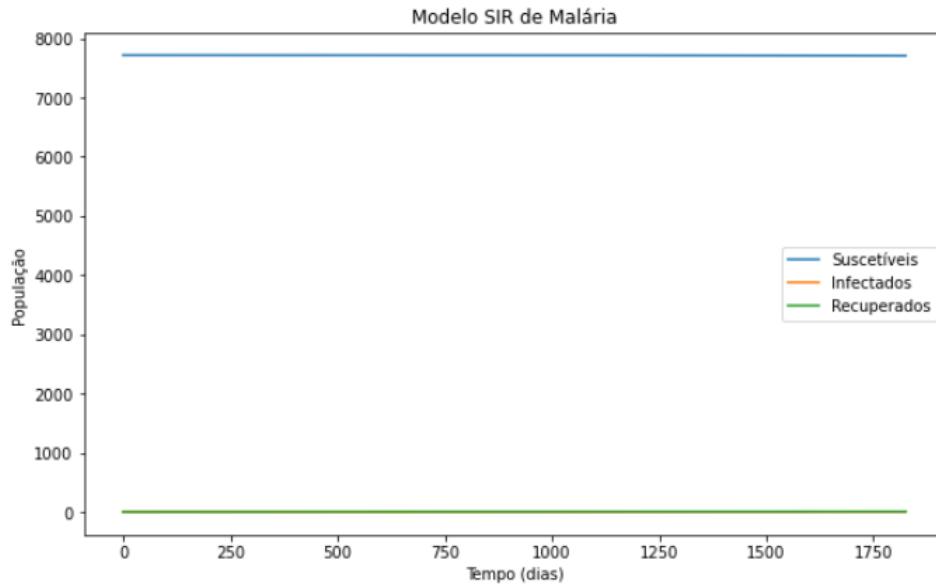


Figure 7 – SIR com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 1$, $I_{H0} = 1$

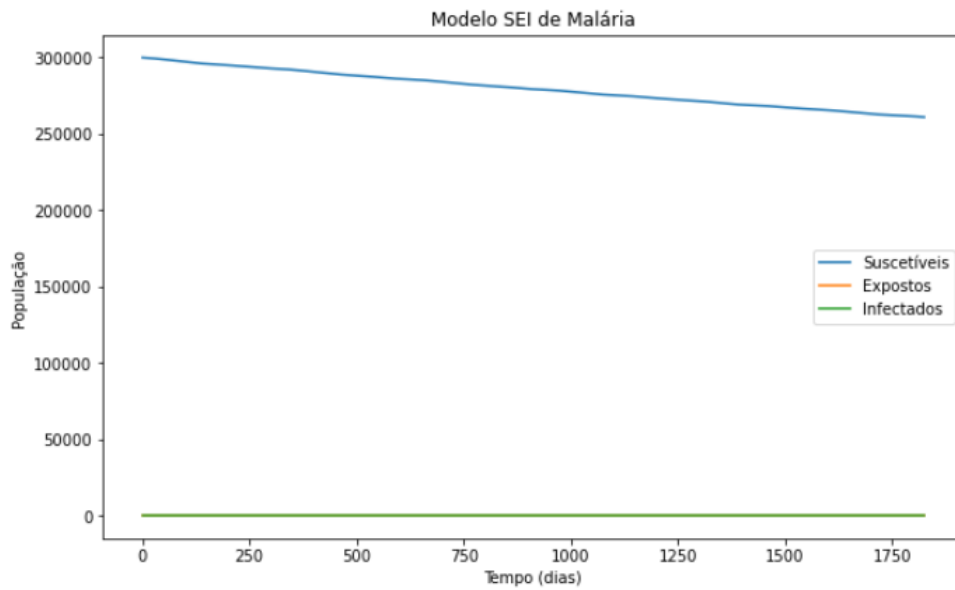


Figure 8 – SEI com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 1$, $I_{H0} = 1$

humanos infectados, a população de infectados humanos começa a aumentar, mas não consegue se estabelecer. Prolongando o tempo de análise, será possível ver que a população se extinguirá nos anos seguintes. Idealmente, seria possível verificar a população de infectados tendendo a 0 ainda no tempo da análise. Por outro lado, iniciando a população humana com cerca de 13% de infectados, é possível ver que essa população tem um aumento ao longo do primeiro ano, chegando a quase 41% da população, mas posteriormente decaindo.

No caso dos mosquitos, a população inicial de expostos quase que imediatamente se torna de infectados, se estabilizando em cerca de 5000 indivíduos, mas não é possível

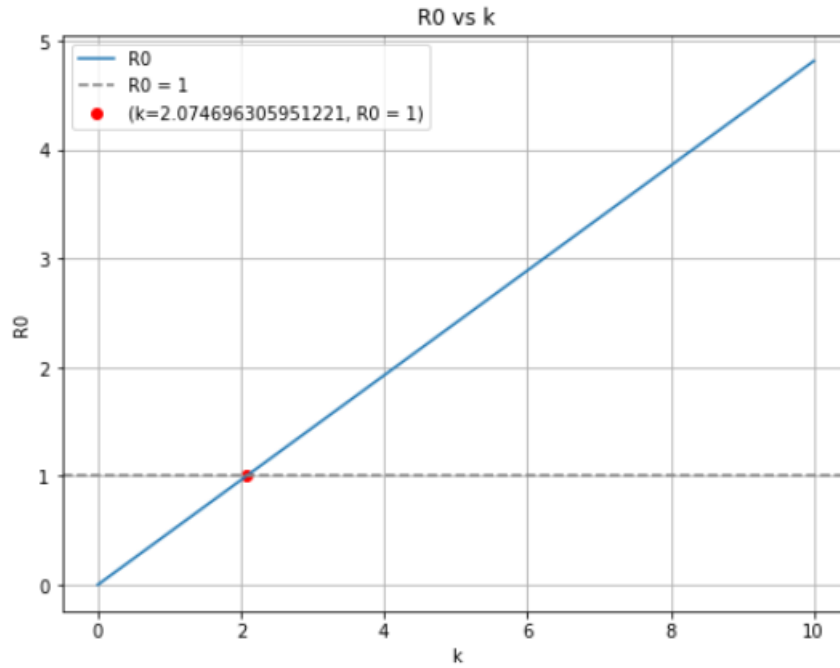
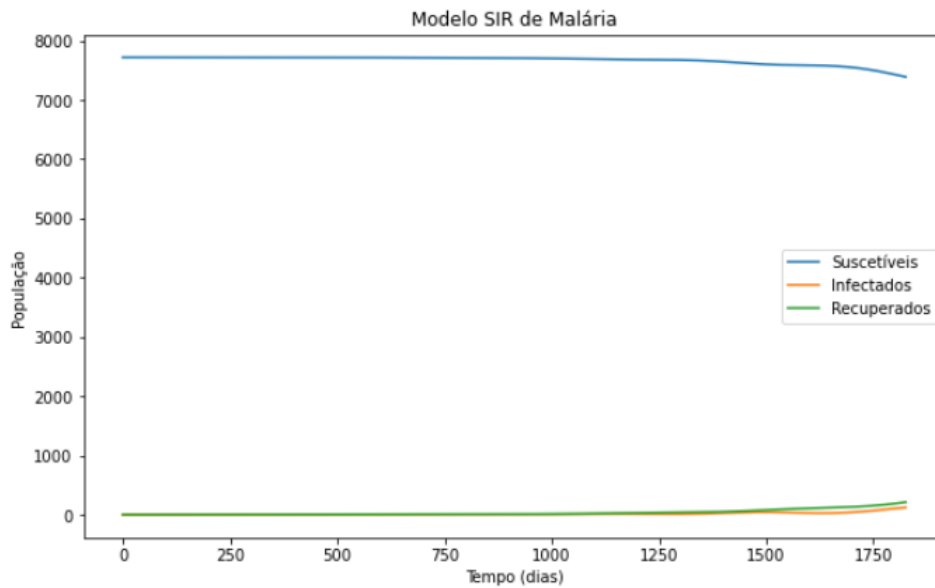
perceber a população de infectados se extinguindo, mesmo prolongando o tempo de análise. Esse comportamento ainda pode ser notado quando 1/3 da população começa exposto, rapidamente se tornando infectada, mas se estabelecendo em cerca de 10000 indivíduos.

Agora, voltando a um único exposto e infectado inicialmente, e incluindo um fator multiplicativo k nas proporções b_1 e b_2 , representando o aumento de contato entre humanos e mosquitos devido ao desmatamento, foi possível analisar o efeito desse impacto na evolução da doença. A formulação final do modelo ficou como a seguir:

$$\left\{ \begin{array}{l} \frac{dS_H}{dt} = \mu_H N - akb_2 \left(\frac{I_M}{N} \right) S_H - \mu_H S_H \\ \frac{dI_H}{dt} = akb_2 \left(\frac{I_M}{N} \right) S_H - \gamma I_H - \mu_H I_H \\ \frac{dR_H}{dt} = \gamma I_H - \mu_H R_H \\ \frac{dS_M}{dt} = b - akb_1 \left(\frac{I_H}{N} \right) S_M - \mu S_M \\ \frac{dE_M}{dt} = akb_1 \left(\frac{I_H}{N} \right) S_M - \mu E_M - b_3 E_M - l E_M \\ \frac{dI_M}{dt} = b_3 E_M - \mu I_M \end{array} \right.$$

Aumentando as proporções em 20 e 50% ($k = 1.2$ e $k = 1.5$), não foi possível perceber nenhuma diferença visível na evolução da doença, isso porque $\mathcal{R}_0 < 1$ para $k < 2.0746963059512207$, como pode ser visto abaixo:

Sendo assim, mesmo um aumento de 100% nas proporções de picadas causando infecção não seria suficiente para que a doença se torne endêmica. Abaixo estão testes com $k = 2.5$, 5 e 10:

Figure 9 – \mathcal{R}_0 em função de k Figure 10 – SIR com $T' = 25.6^\circ C$, $A = 12.5 (\text{°C}^2 \text{ dias})^{-1}$, $B = 15 (\text{°C dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $k = 2.5$

Aumentando em 150% a proporção de picadas causando infecção, já é possível notar que o número de infectados começa a aumentar, ainda que quase no final do período de análise. Isso porque com o aumento em k , \mathcal{R}_0 passou de 0.48, com $k = 1$, para 1.2, permitindo que a doença se estabeleça a longo prazo. Como k está relacionado tanto com b_1 quanto b_2 , esse fator aparecerá como k^2 no numerador de \mathcal{R}_0 , portanto afetando seu valor de forma linear. Aumentando k para 5, será bem mais perceptível a evolução da doença:

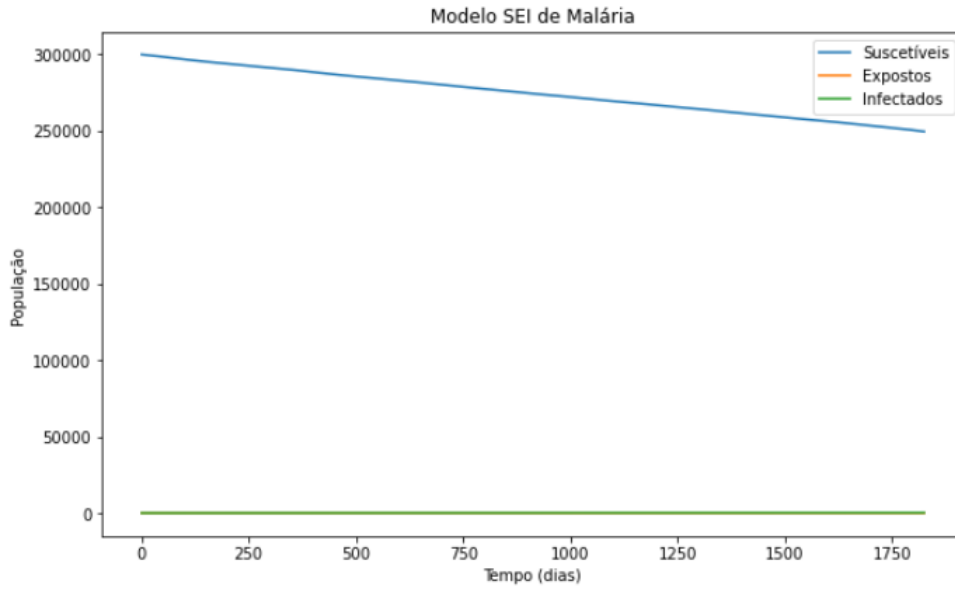


Figure 11 – SEI com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $k = 2.5$

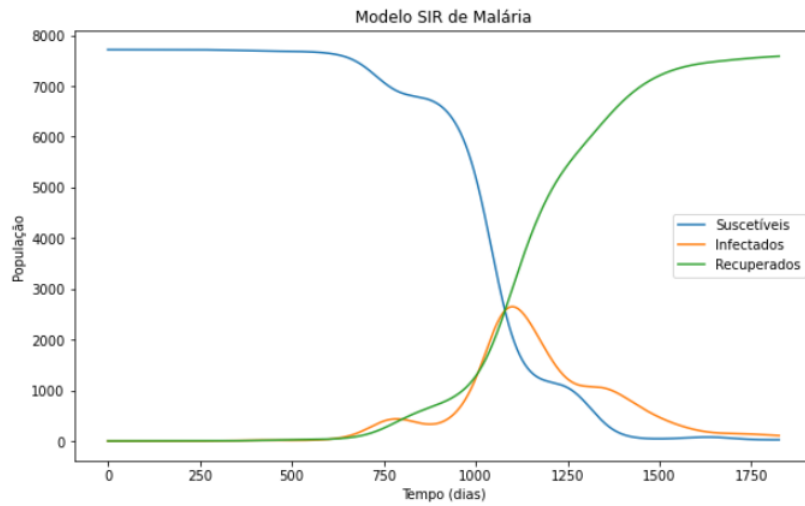


Figure 12 – SIR com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $k = 5$

Nesse caso, $\mathcal{R}_0 = 2.41$, e apesar de não ser possível verificar no tempo máximo dos 5 anos, nesse caso o número de infectados consegue se estabilizar, oscilando em aproximadamente 50 humanos e 9000 mosquitos infectados. Aumentando k para 10, $\mathcal{R}_0 = 4.82$, e a infecção atinge seu máximo de forma ainda mais rápida:

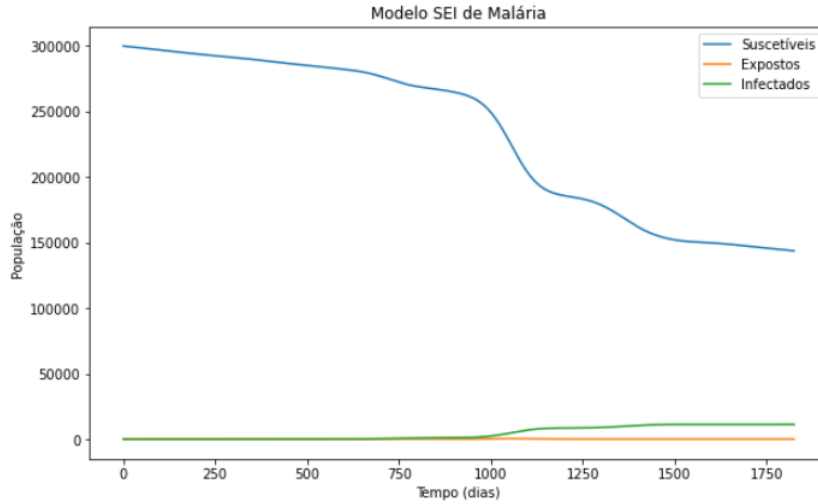


Figure 13 – SEI com $T' = 25.6^\circ C$, $A = 12.5$ ($^\circ C^2$ dias) $^{-1}$, $B = 15$ ($^\circ C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 450$ mm, $k = 5$

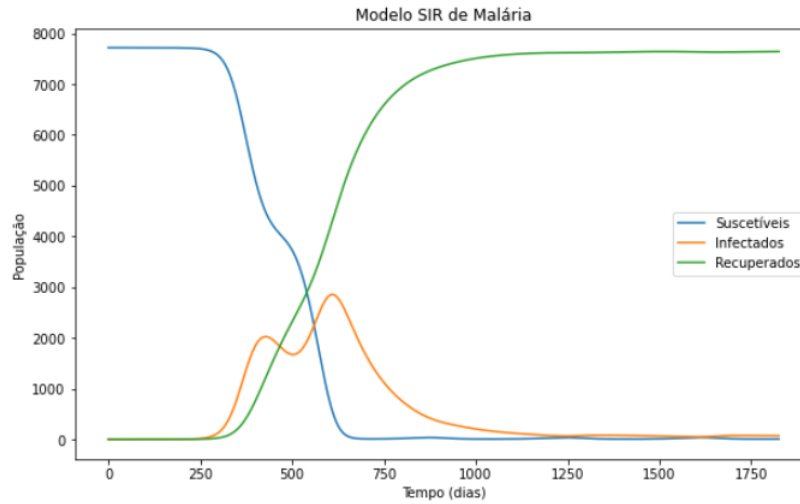


Figure 14 – SIR com $T' = 25.6^\circ C$, $A = 12.5$ ($^\circ C^2$ dias) $^{-1}$, $B = 15$ ($^\circ C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 450$ mm, $k = 10$

Com esses resultados, é possível perceber que o desmatamento acarretando em aproximação entre hospedeiro e vetor causa um alto impacto na dinâmica da malária, dado que mesmo com um único indivíduo infectado, a doença se estabelece e atinge um nível de infecção humana de cerca de 40% da população conforme a proporção de picadas causando infecção aumenta. Agora, podemos encontrar qual é o valor de I_H no equilíbrio dependendo de k :

Iniciando o plot a partir de k que deixa $\mathcal{R}_0 = 1$, é possível ver que o equilíbrio endêmico da população humana se aproxima de 64 conforme k se aproxima de 10. Mais especificamente, quando $k = 10$, $I_H^* \approx 63.49$ ⁸. Analisando o equilíbrio de suscetíveis conforme k aumenta, é

⁸ A elaboração dos gráficos em função de k podem ser encontrados em https://github.com/RaphaLevy/TCC/blob/main/Modelagem_com_Dinamica_Pop/Plota_Equilibrio_e_R0.ipynb. O cálculo dos equilíbrios pode ser encontrado em https://github.com/RaphaLevy/TCC/blob/main/Modelagem_com_

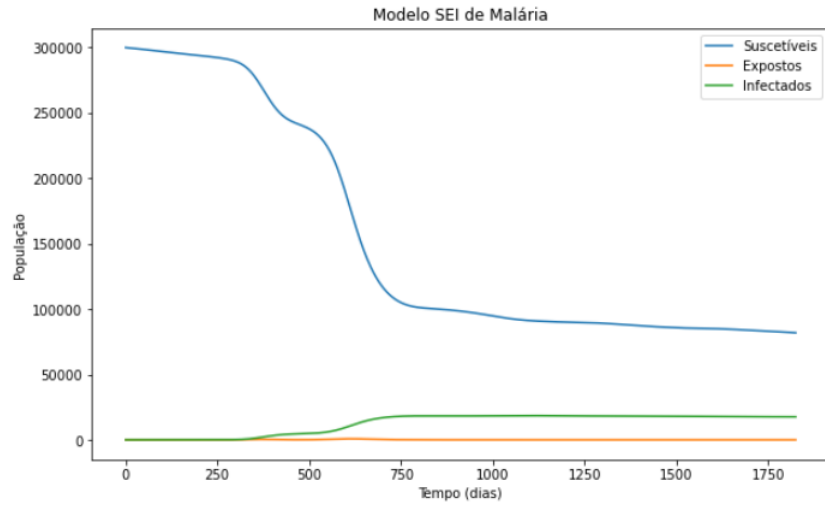


Figure 15 – SEI com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $k = 10$

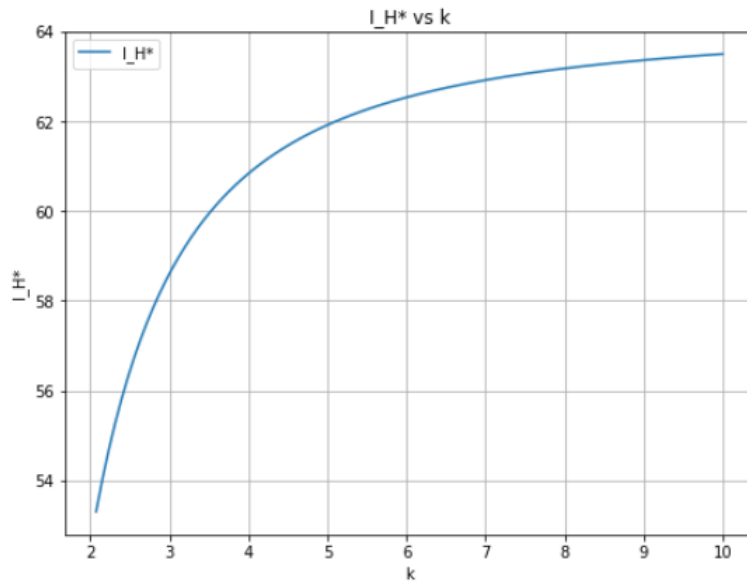
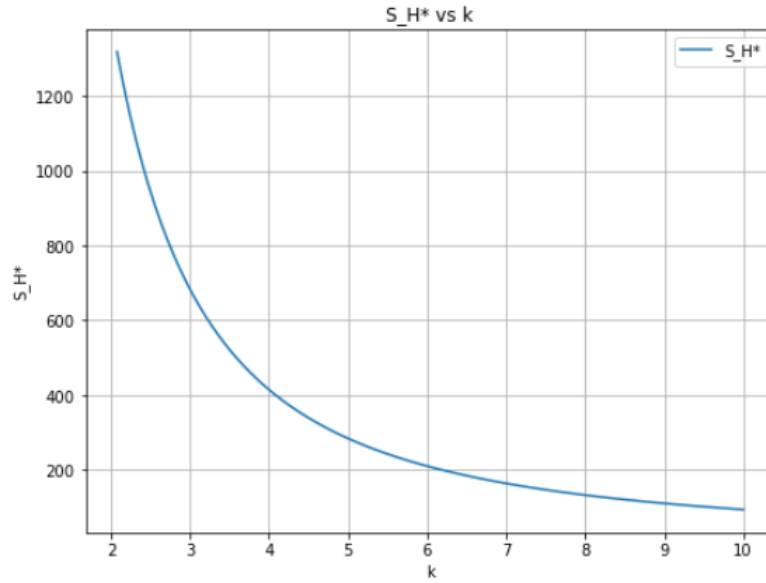
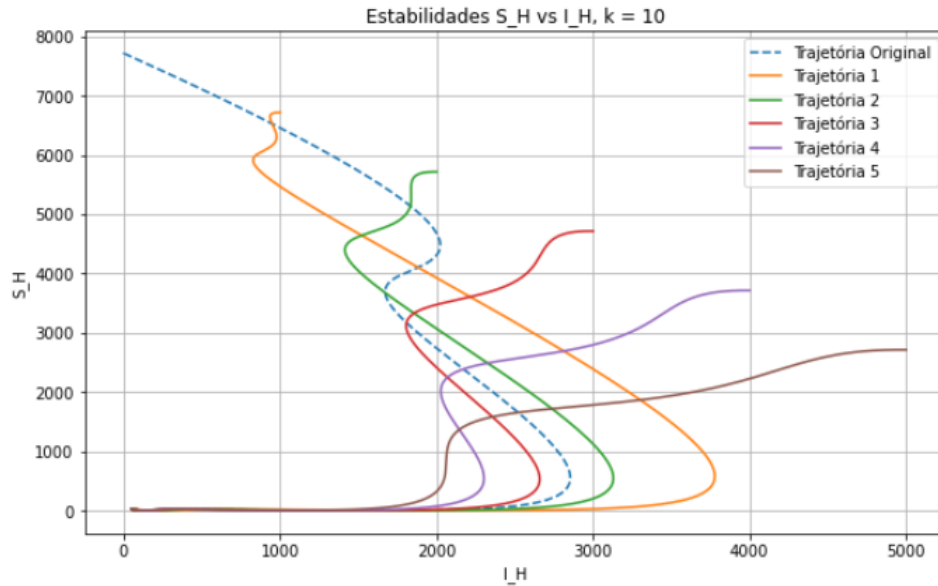


Figure 16 – I_H^* em função de k

possível ver o equilíbrio decaindo rapidamente de N quando $k = 0$ para aproximadamente 5000 indivíduos quando $k = 1$. Analisando nos valores de k tais que $\mathcal{R}_0 \geq 1$:

Figure 17 – S_H^* em função de k

Nesse caso, a população de suscetíveis tende a aproximadamente 95 conforme k se aproxima de 10. Tendo calculado os equilíbrios de S_H e I_H , foi possível fazer uma análise de estabilidade global. Como estamos interessados em analisar o equilíbrio endêmico, utilizei $k = 10$:

Figure 18 – Equilíbrio global $S_H^* \times I_H^*$ para $k = 10$

Nesse caso, foram feitas 6 análises, a primeira utilizando os valores iniciais de S_H e I_H como sendo 7716 e 1, e as demais aumentando I_H em 1000 e diminuindo S_H em 1000 indivíduos. Nesse caso, é possível ver as populações de suscetíveis e infectados com um equilíbrio final de aproximadamente 8 e 70 pessoas, respectivamente. Com isso, poderíamos comparar o resultado obtido com o cálculo do equilíbrio endêmico de Adda e Bichara

([ADDA; BICHARA, 2011](#)), onde

$$S_H^* = \frac{1}{\mathcal{R}_0}$$

$$I_H^* = \frac{\mu_H}{\mu_H + \gamma} \left(1 - \frac{1}{\mathcal{R}_0}\right)$$

Através desse cálculo, a população de suscetíveis e infectados no equilíbrio foi de aproximadamente 1601 e 51, respectivamente. Notavelmente, esses valores estão destoantes dos obtidos através do cálculo numérico. Contudo, é necessário considerar a principal diferença entre as equações propostas para S e I nesse Trabalho e no artigo de Adda e Bichara, que é o uso de I_M na taxa de infecção β , dada a dinâmica do modelo acoplado de SIR e SEI nesse caso, que não está sendo considerada no trabalho de Adda e Bichara.

4 Discussion

Throughout the work, a SIR/SEI transmission model for malaria was developed and analyzed to complement the original methodology by Parham & Michael. The model incorporated epidemiological factors of disease transmission and complemented them with human demographic dynamics and external environmental factors such as temperature and precipitation, including deforestation, considered in the multiplicative factor for the infection-causing bite proportions.

Given the significant focus on modeling transmission and analyzing the environmental impacts, requiring various adaptations to make it more realistic and compatible with disease dynamics in the environment, the in-depth analysis of the effects of socioeconomic factors, as well as the identification of disease prevention and control strategies, ended up beyond the scope of the undergraduate thesis.

Regarding what was accomplished, a key takeaway from the work is that this model is highly sensitive to the parameters used. Small modifications are sufficient for the model to reach equilibrium or for populations to tend toward $\pm\infty$.

Indeed, what was particularly noted when modifying parameters A , B , and C was that using the values indicated in the works of Parham & Michael (PARHAM; MICHAEL, 2010) and Eikenberry & Gummel (OKUNEYE; GUMEL, 2017), in the case $A = -0.03$, $B = 1.31$, $C = -4.4$, no epidemic occurred. Starting the modeling with the other parameters used in Figure 7, with $k = 1$, $\mathcal{R}_0 = 0.0207$, a value much lower than previously verified. Even with $k = 10$, $\mathcal{R}_0 = 0.207$, leading to mosquito population extinction in all cases and making the existence of endemic equilibrium unviable.

Another point that could be perceived was that in many cases, malaria takes a long time to reach endemic equilibrium. Therefore, although it was not possible to study the application of disease control strategies, it can be concluded that long-term measures may not be as effective, as the environmental conditions present at the beginning of this analysis would no longer be the same when the measure is applied.

As future work, it would be possible to compare the general methodology used by the referenced authors with the methodology used in this work and identify what could be modified so that, using the original parameters, the model still reaches endemic equilibrium.

Furthermore, as verified in the equilibrium calculations, something else that could be

studied as a continuation of the work is the analysis of its evolution over time since they are given by oscillatory functions, such as the biting rate, birth rate, mortality rate, survival probability, and the infection rate of exposed individuals, which vary depending on temperature and precipitation factors, as presented previously.

5 Conclusion

Throughout the development of the undergraduate thesis, different modifications to the dynamics of malaria transmission in the Amazon were explored in order to align the modeling more closely with the natural history of the disease in this environment. The ultimate goal was to understand how ecological impacts in the region affect interactions between the vector and the host.

With the obtained results, it was possible to perceive the effect that increased contact between humans and mosquitoes due to deforestation can have on malaria dynamics, based on the proportion of bites causing infection. Furthermore, it was observed how, depending on the original parameters provided, a much higher proximity between vector and host would be required for the disease to become endemic in the Amazon region. As verified, this contact could be double the normal amount, and yet it is still insufficient for the disease to become an epidemic.

To further align the methods used with observed behaviors in reality, the application of a stochastic transmission model, incorporating constantly changing environmental variables, could be ideal. However, for the proposed purpose, the deterministic model used was sufficient to highlight the disease's sensitivity to climate and environmental changes, allowing a clear and focused analysis of interactions between vector and host. This provides a solid foundation for investigating the implications of environmental changes on malaria transmission and for future research and improvements in the model.

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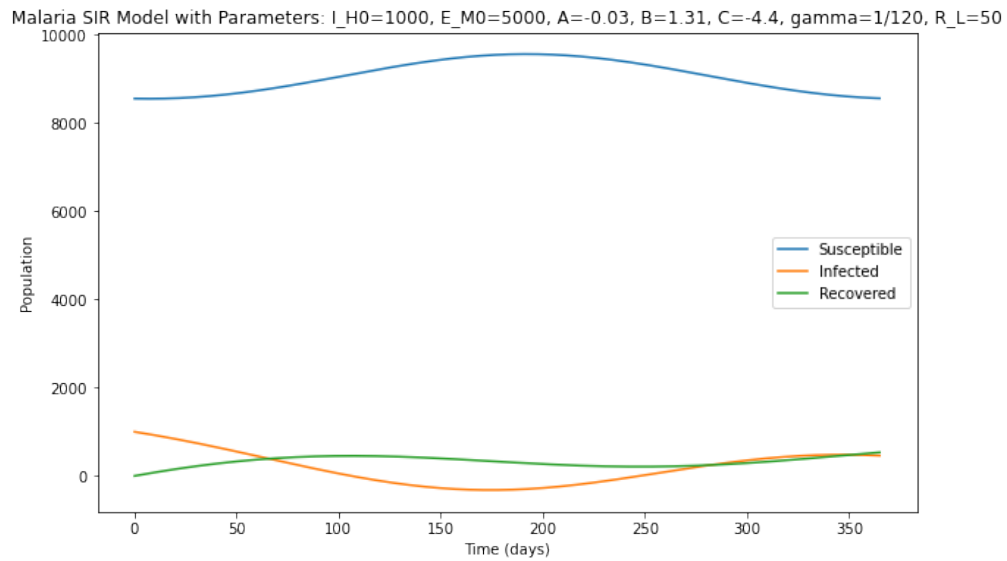
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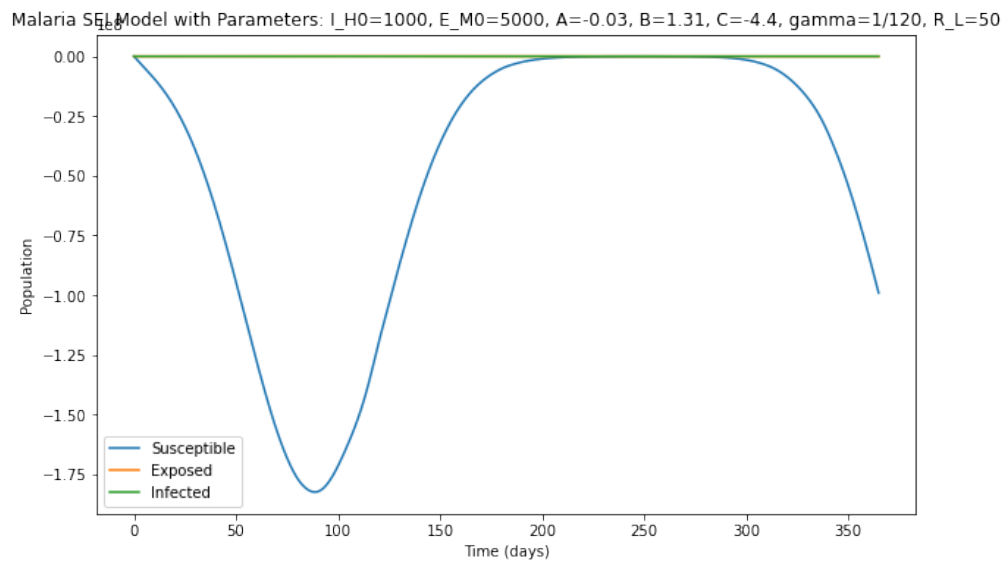
Appendix

APPENDIX A – Results

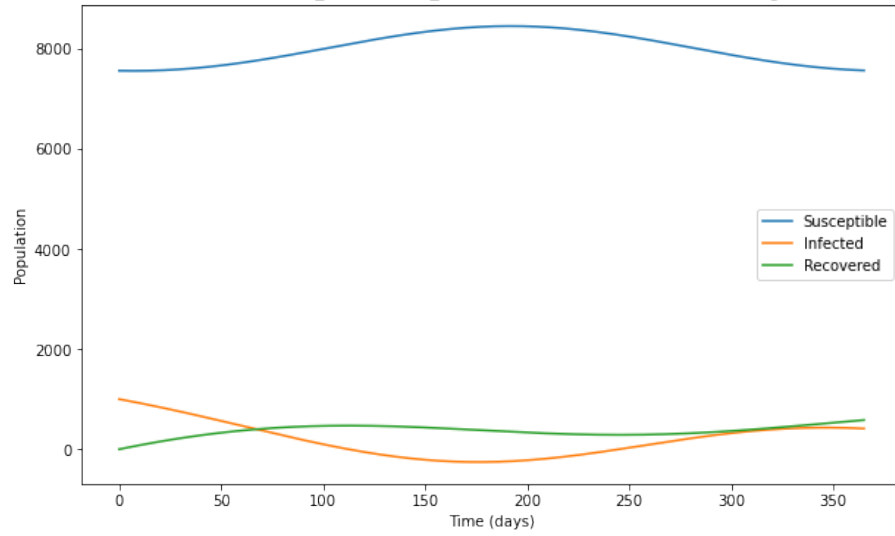
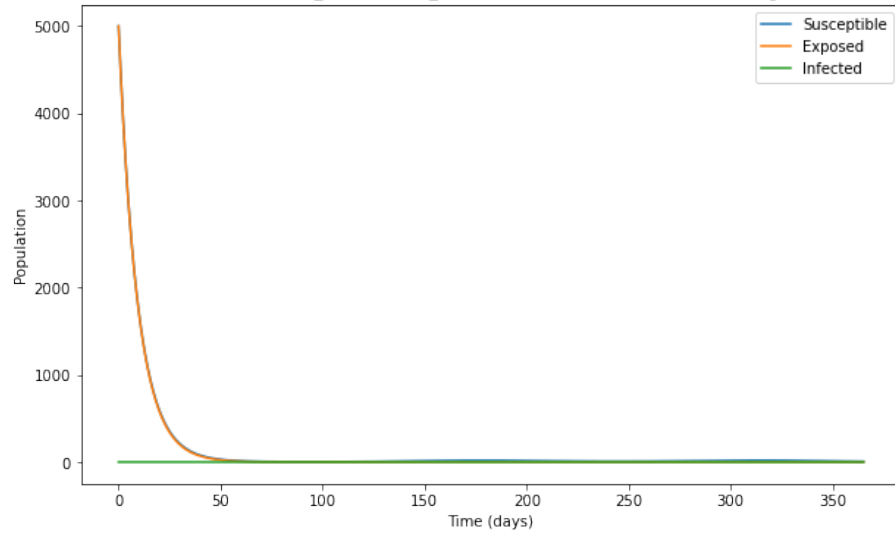
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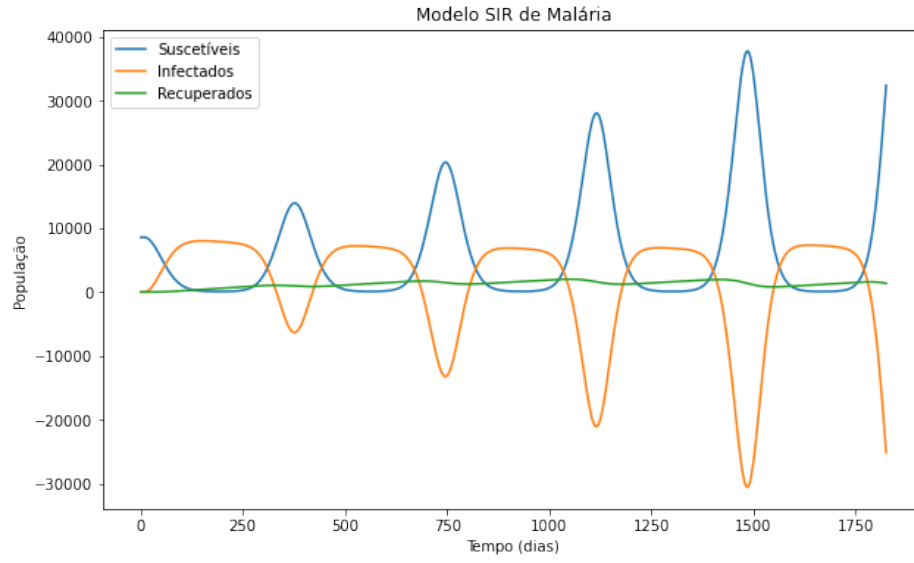


Appendix 1: Original SIR with corrected T and R

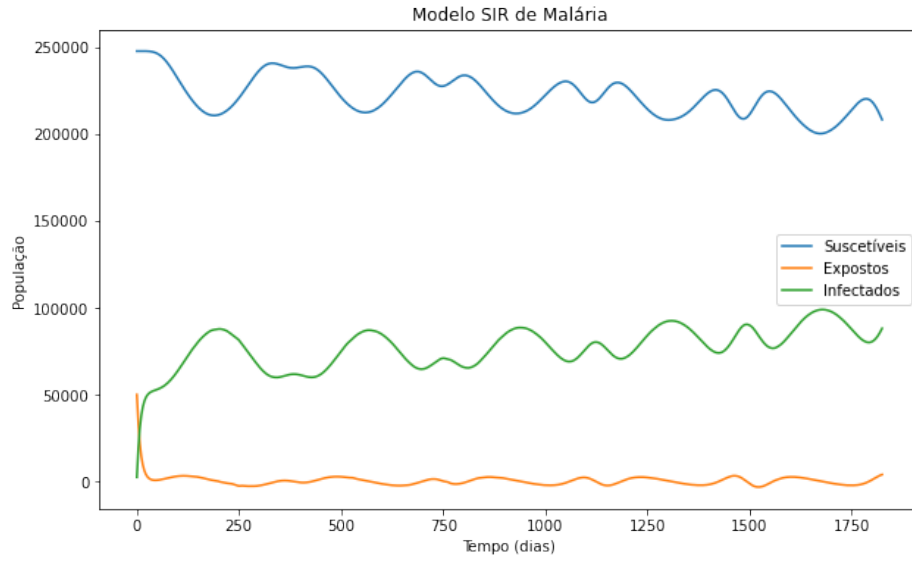


Appendix 2: Original SEI with corrected T and R

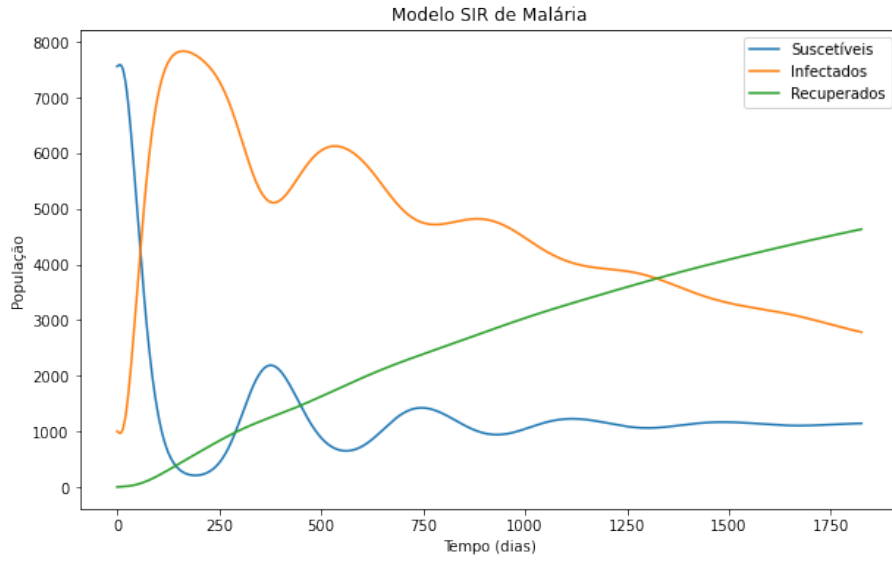
Malaria SIR Model with Parameters: $I_{H0}=1000$, $E_{M0}=5000$, $A=-0.03$, $B=1.31$, $C=-4.4$, $\gamma=1/120$, $R_L=450$ Appendix 3: Original SIR with corrected R_L Malaria SEI Model with Parameters: $I_{H0}=1000$, $E_{M0}=5000$, $A=-0.03$, $B=1.31$, $C=-4.4$, $\gamma=1/120$, $R_L=450$ Appendix 4: Original SEI with corrected R_L



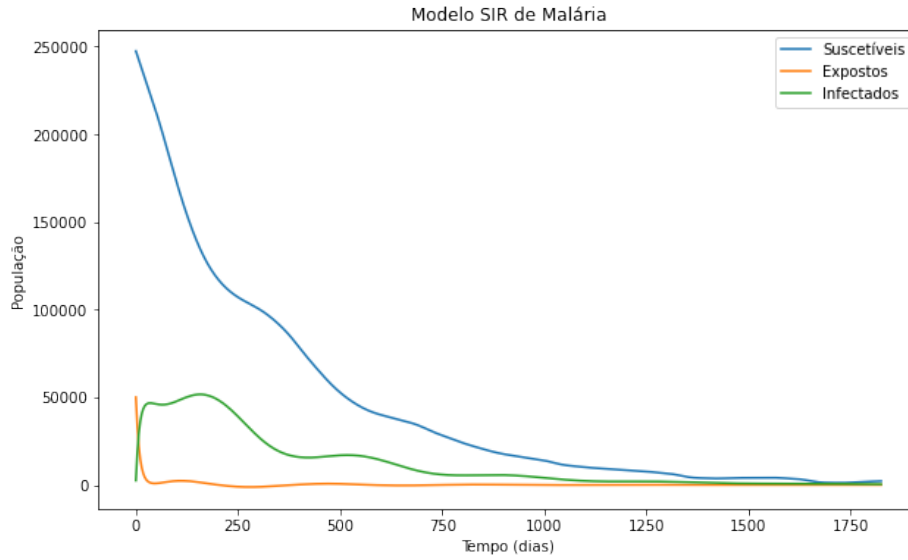
Apêndice 5: SIR com $T' = 26.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$



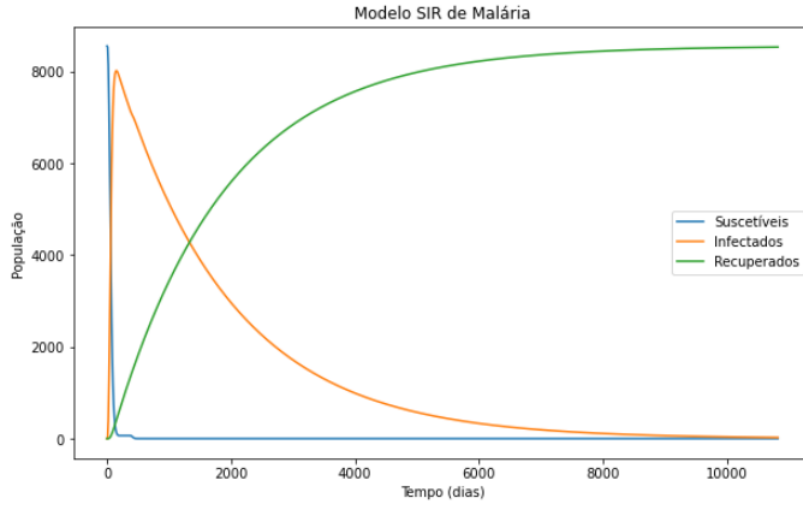
Apêndice 6: SEI com $T' = 26.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$



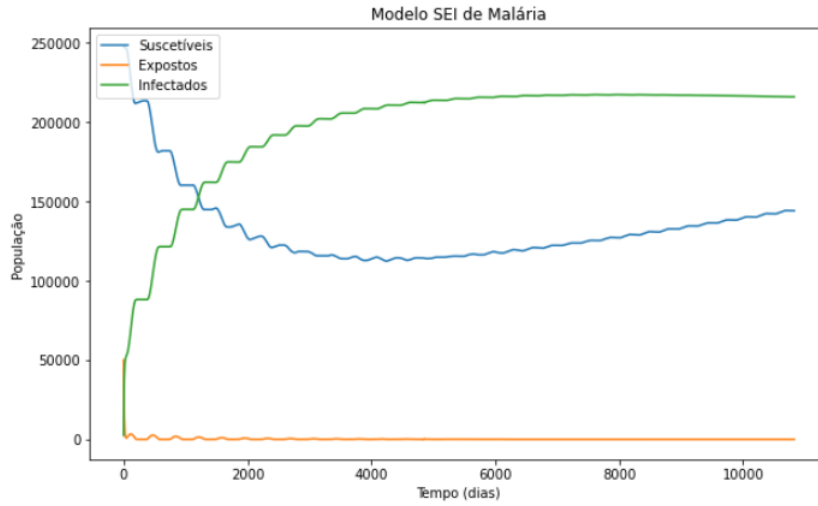
Apêndice 7: SIR com $T' = 26.4^{\circ}C$, $A = 0$ ($^{\circ}C^2$ dias) $^{-1}$, $B = 15$ ($^{\circ}C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 312\text{mm}$



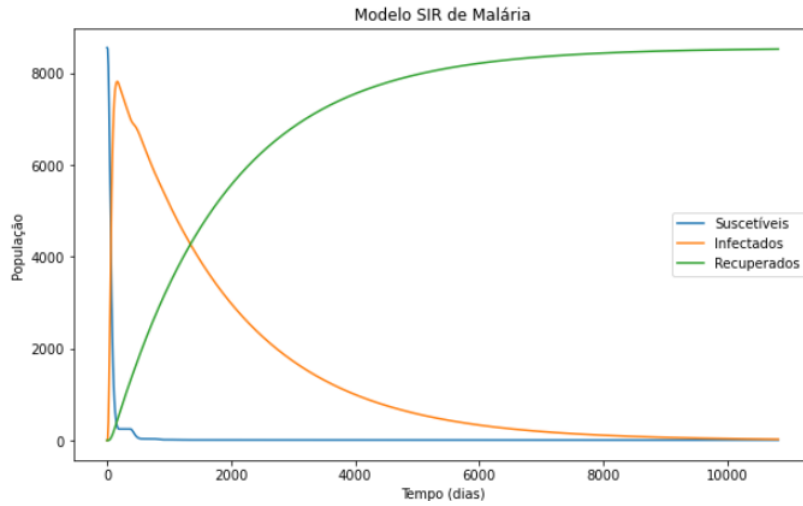
Apêndice 8: SEI com $T' = 26.4^{\circ}C$, $A = 0$ ($^{\circ}C^2$ dias) $^{-1}$, $B = 15$ ($^{\circ}C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 312\text{mm}$



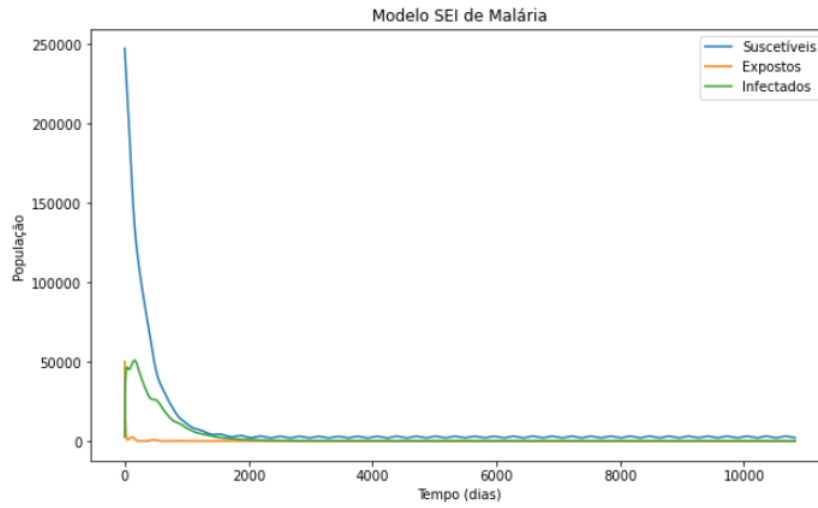
Apêndice 9: SIR com $T' = 26.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$



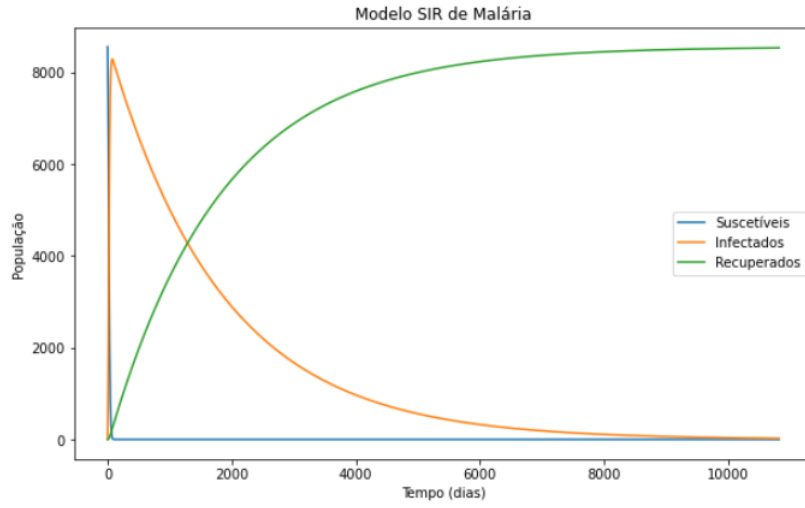
Apêndice 10: SEI com $T' = 26.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$



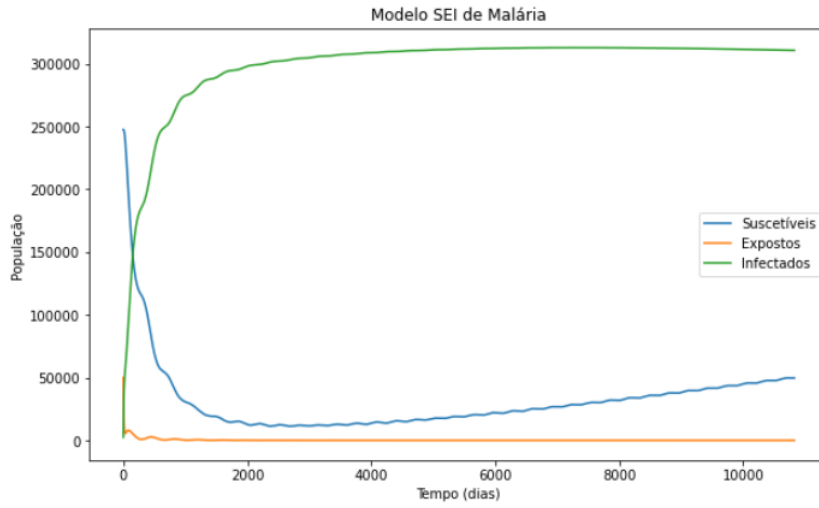
Apêndice 11: SIR com $T' = 26.4^{\circ}C$, $A = 0$ ($^{\circ}C^2$ dias) $^{-1}$, $B = 15$ ($^{\circ}C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 312\text{mm}$



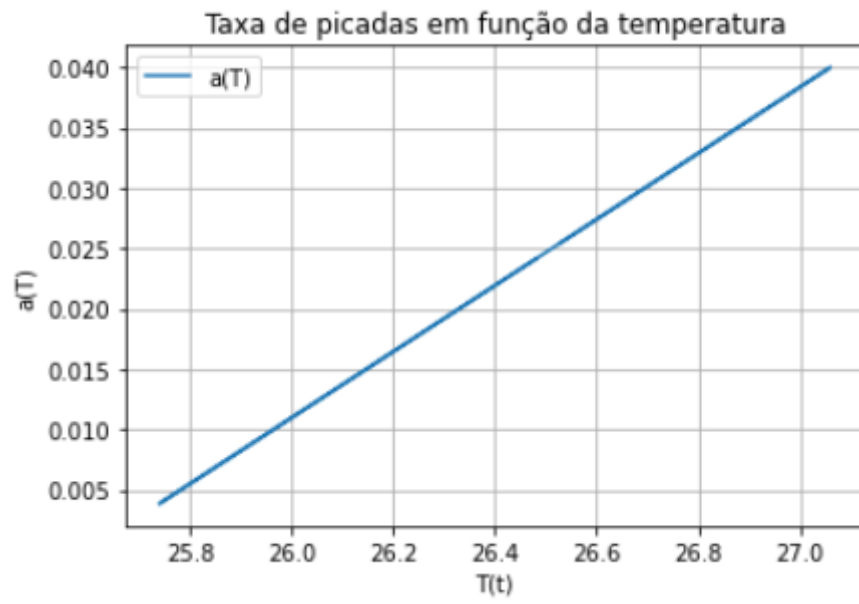
Apêndice 12: SEI com $T' = 26.4^{\circ}C$, $A = 0$ ($^{\circ}C^2$ dias) $^{-1}$, $B = 15$ ($^{\circ}C$ dias) $^{-1}$, $C = -48.78$ (dias) $^{-1}$, $R_L = 312\text{mm}$



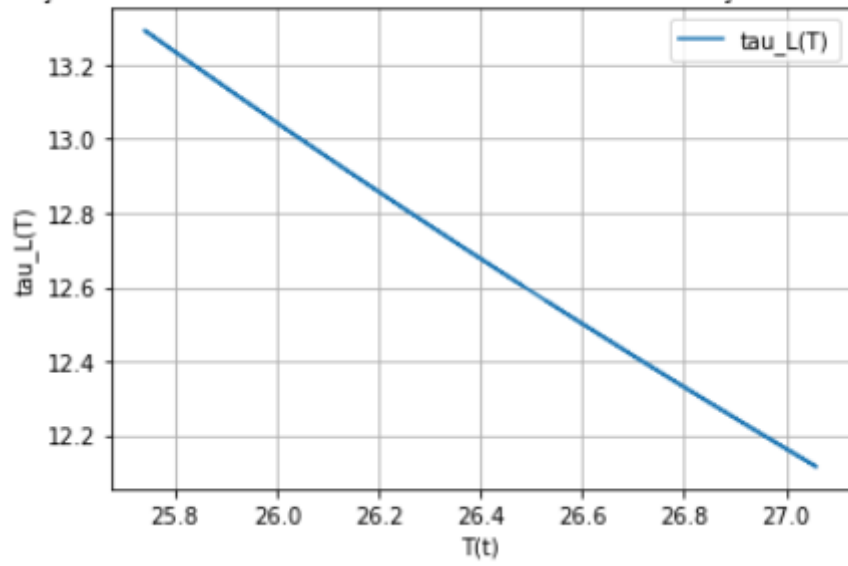
Apêndice 13: SIR com $T' = 27.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$



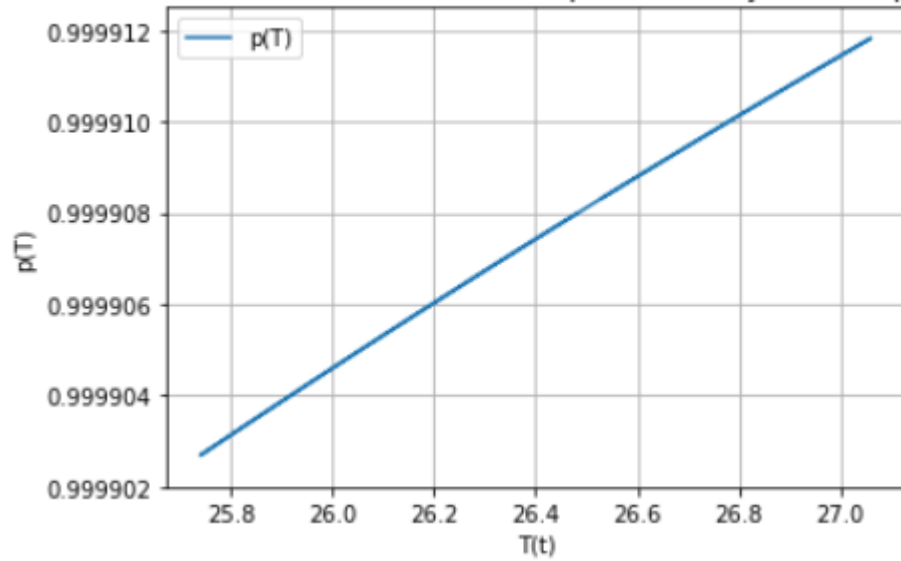
Apêndice 14: SEI com $T' = 27.4^{\circ}C$, $A = 317.925 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 312\text{mm}$

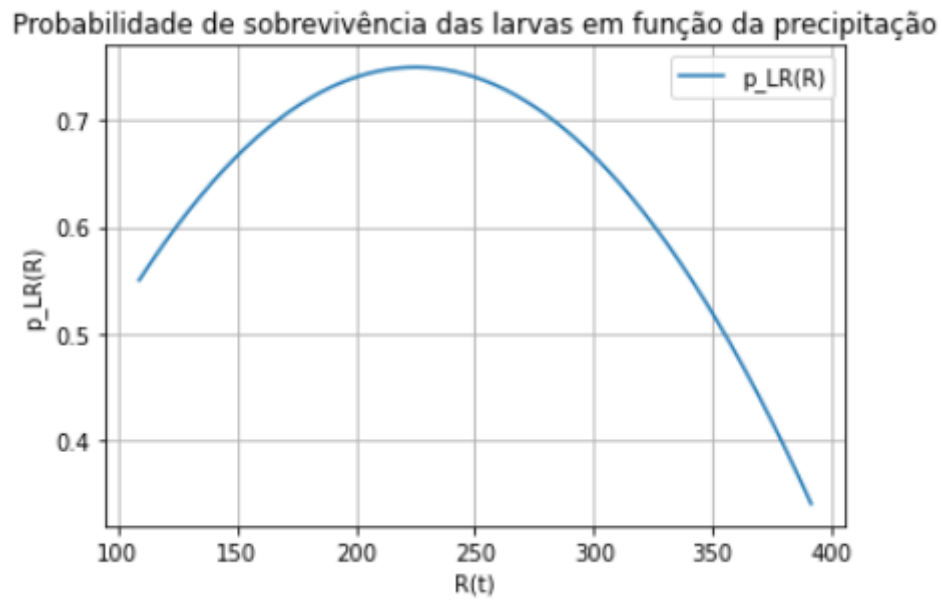
Apêndice 15: Taxa de picadas ($a(T)$)Apêndice 16: Taxa de infecção de expostos ($b_3(T)$)

Duração da fase de desenvolvimento das larvas em função da temperatura

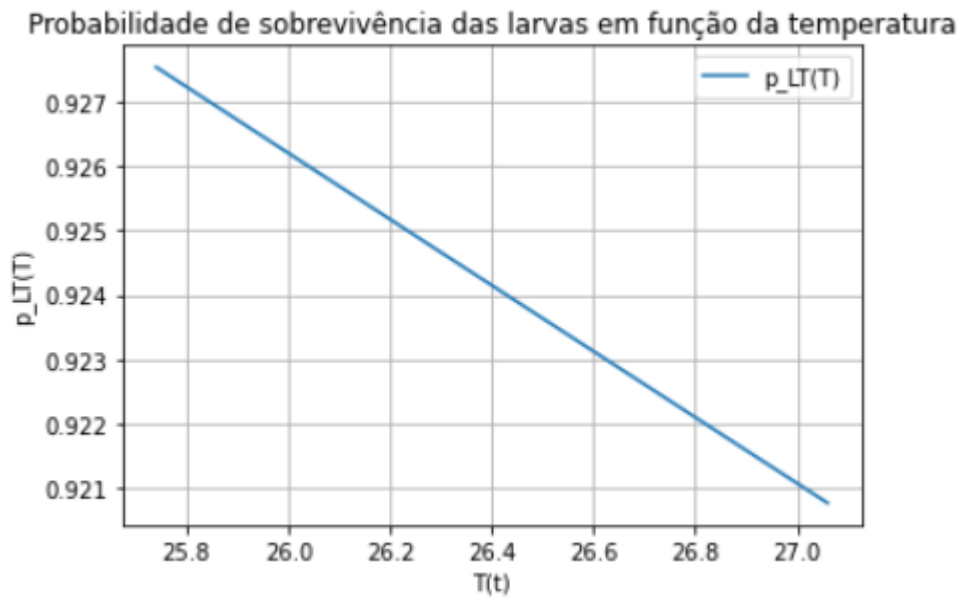
Apêndice 17: Duração da fase de desenvolvimento das larvas ($\tau_L(T)$)

Taxa diária de sobrevivência dos mosquitos em função da temperatura

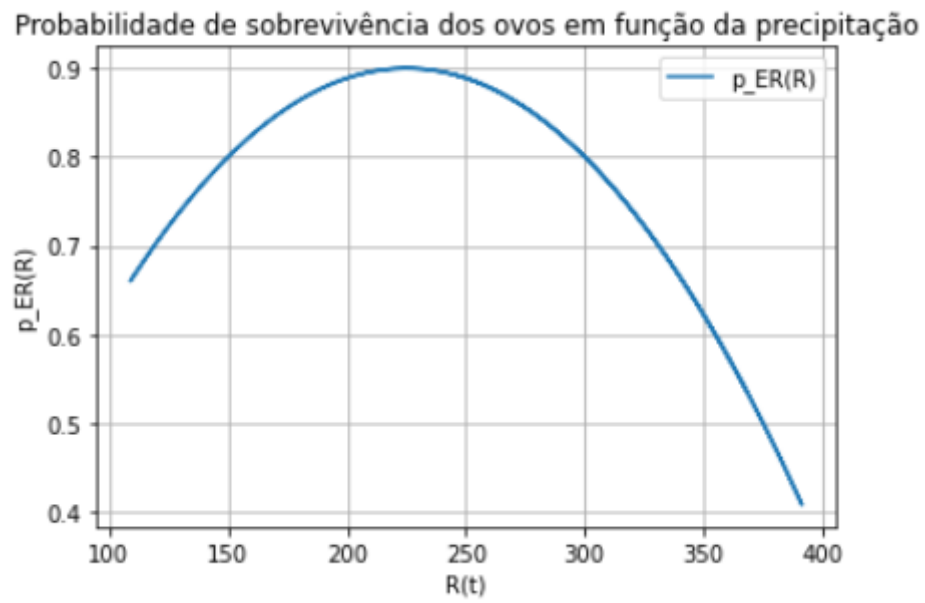
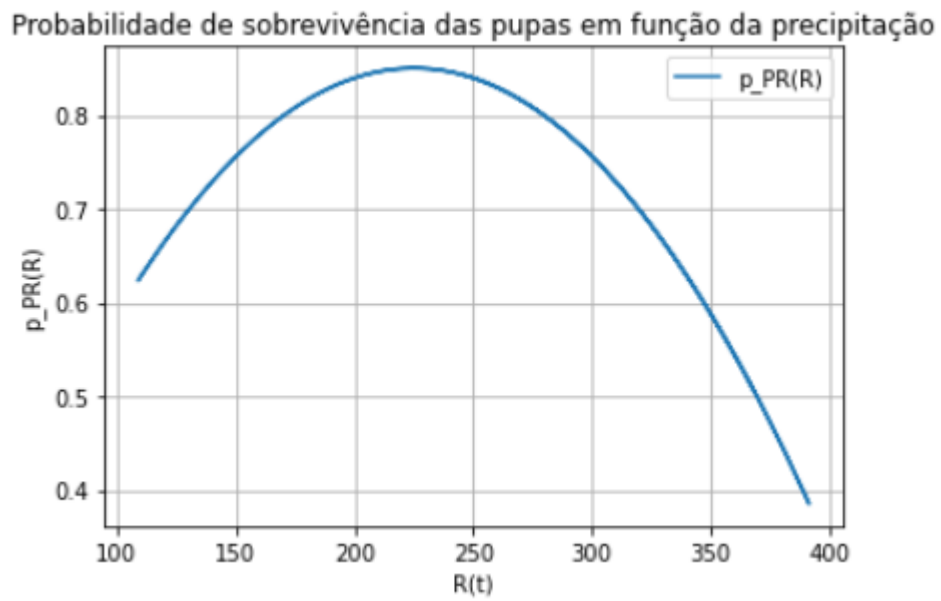
Apêndice 18: Taxa diária de sobrevivência dos mosquitos ($p(T)$)

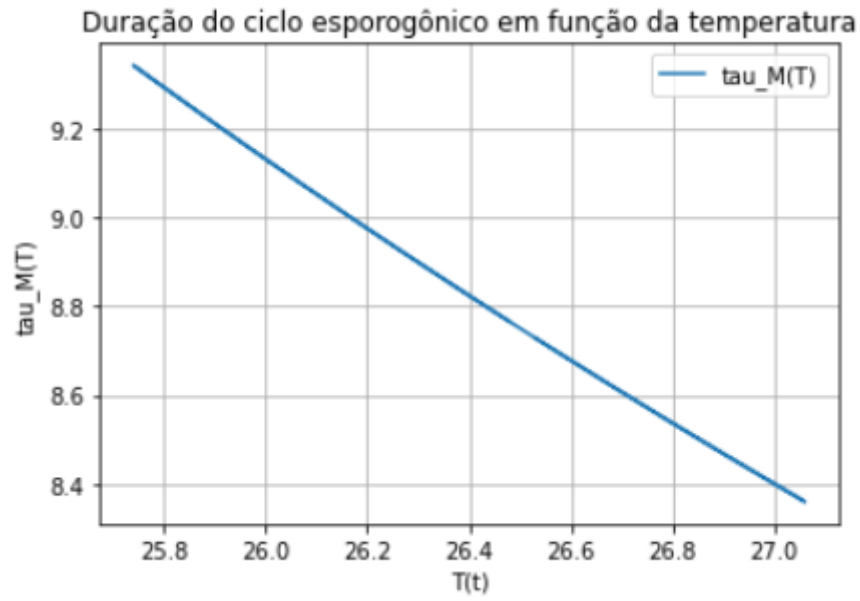
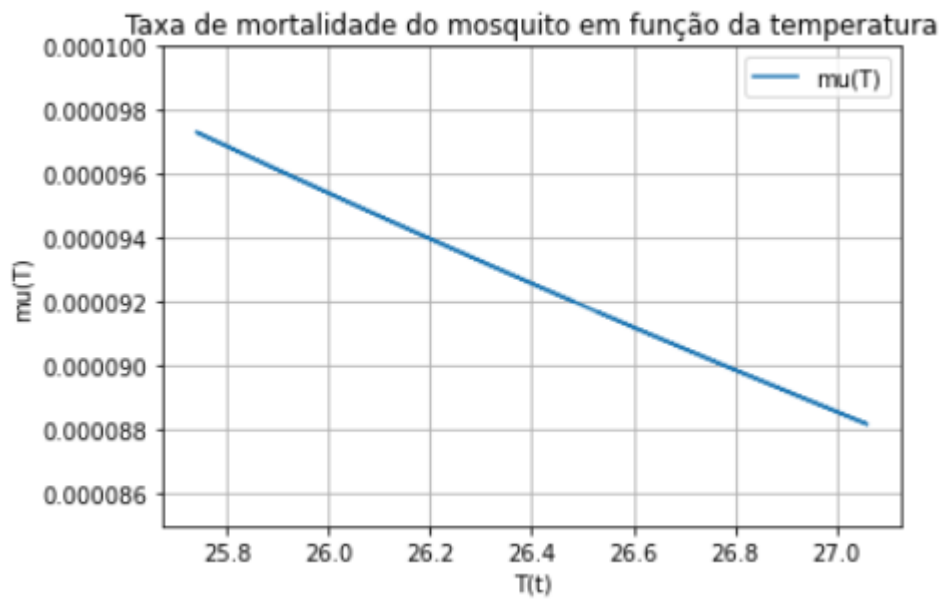


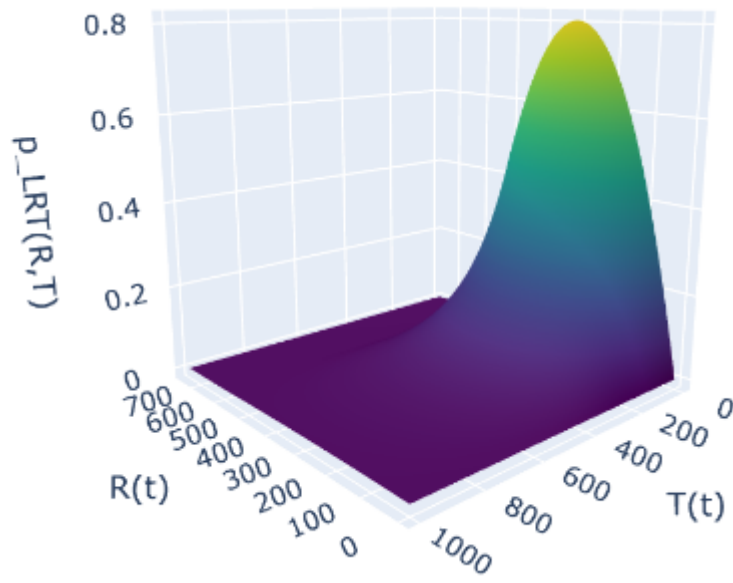
Apêndice 19: Probabilidade de sobrevivência das larvas ($p_{LR}(R)$)



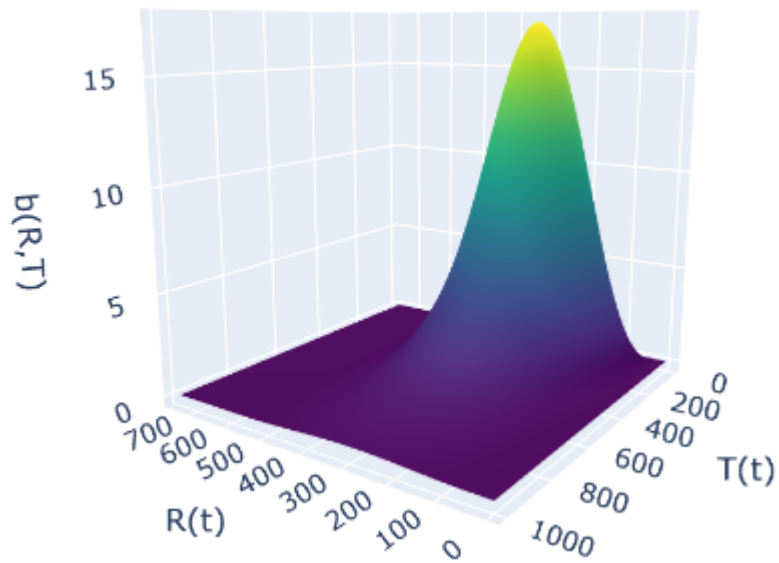
Apêndice 20: Probabilidade de sobrevivência das larvas ($p_{LT}(T)$)

Apêndice 21: Probabilidade de sobrevivência dos ovos ($p_{ER}(R)$)Apêndice 22: Probabilidade de sobrevivência das pupas ($p_{PR}(R)$)

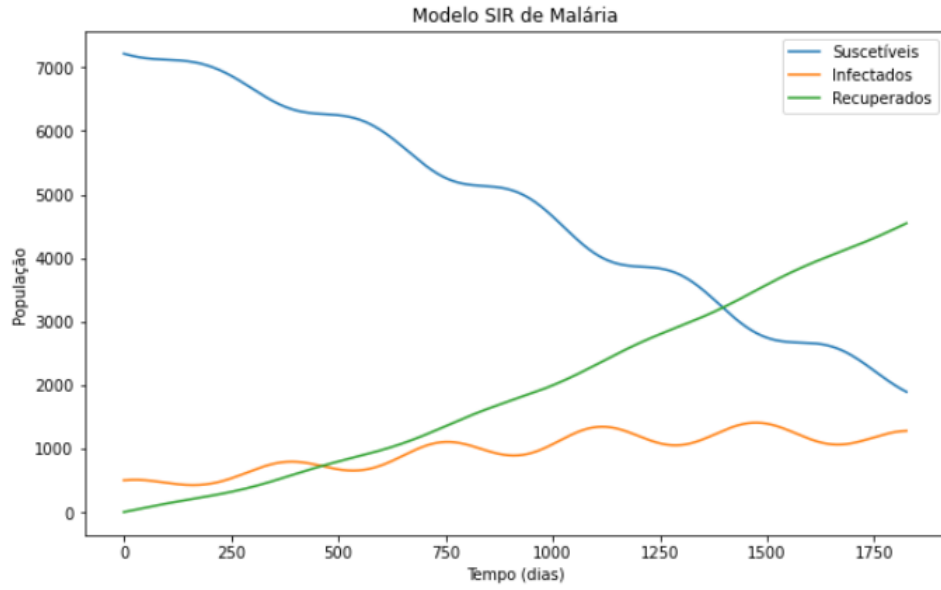
Apêndice 23: Duração do ciclo esporogônico ($\tau_M(T)$)Apêndice 24: Taxa de mortalidade do mosquito ($\mu(T)$)



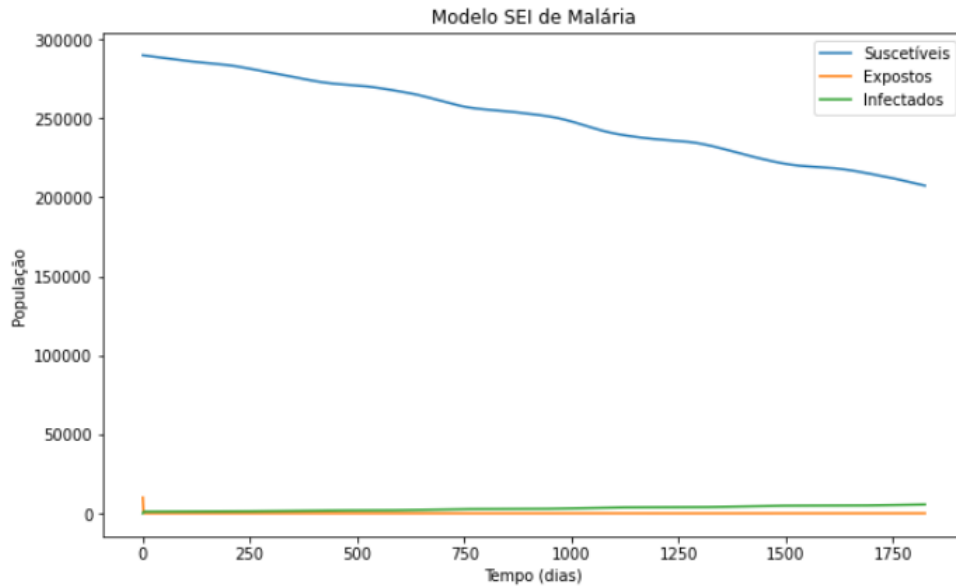
Apêndice 25: Probabilidade de sobrevivência das larvas ($\tau_M(T)$)



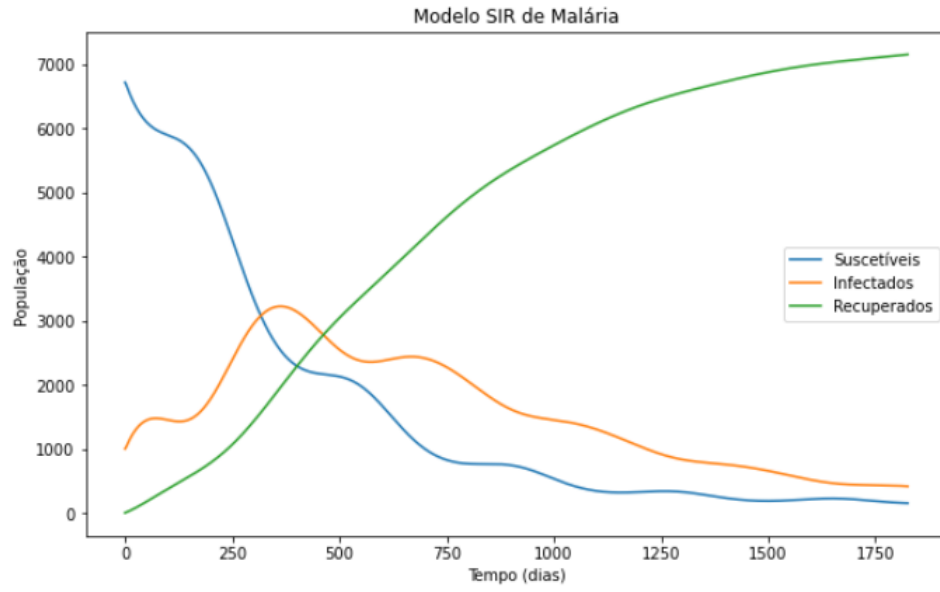
Apêndice 26: Taxa de nascimento de mosquitos ($b(R, T)$)



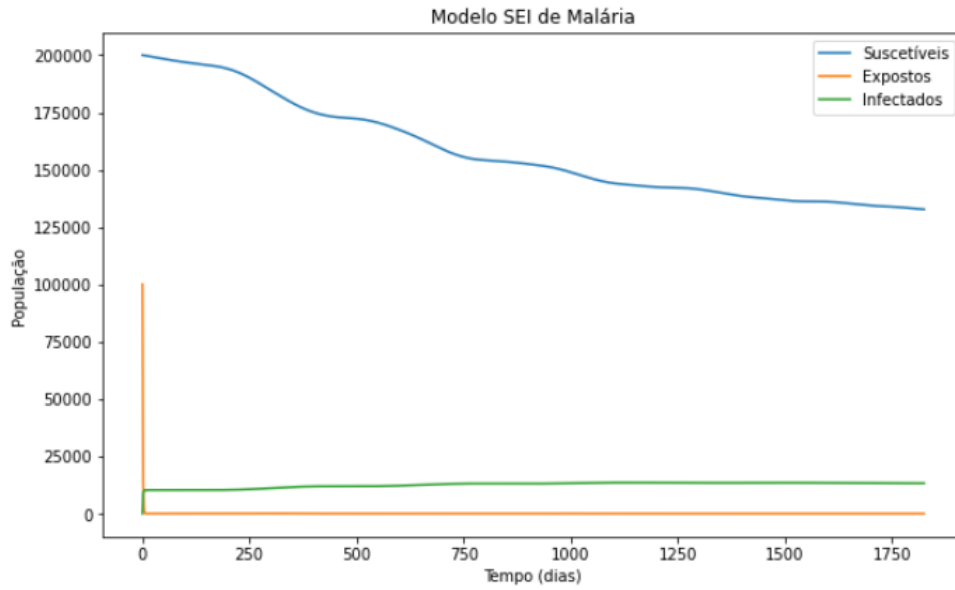
Apêndice 27: SIR com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 10000$, $I_{H0} = 500$



Apêndice 28: SEI com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 10000$, $I_{H0} = 500$



Apêndice 29: SIR com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 100000$, $I_{H0} = 1000$



Apêndice 30: SEI com $T' = 25.6^{\circ}C$, $A = 12.5 (^{\circ}C^2 \text{ dias})^{-1}$, $B = 15 (^{\circ}C \text{ dias})^{-1}$, $C = -48.78 (\text{dias})^{-1}$, $R_L = 450\text{mm}$, $E_{M0} = 100000$, $I_{H0} = 1000$