ANALYSIS OF A COMBINED SIT-SIR MOSQUITO POPULATION AND HUMAN EPIDEMIOLOGY MODEL.

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ABSTRACT
Among all mosquito-transmitted diseases, dengue is one of the most widespread, causing millions on infections and thousands of deaths, especially in Latin America. Lately, the Sterile Insect Technique has been applied successfully to reduce wild mosquito populations in several regions, and therefore control the spread of mosquito-transmitted diseases such as dengue. In this paper we introduce an ODE model that combines a mosquito population model with introduced sterile males with the SIR (Susceptible, Infected, Recovered) model for human epidemics. We aim to obtain a better understanding of the evolution of a dengue pandemic in an area where the Sterile Insect Technique (SIT) is used by taking into account both the mosquito and human population. We will calculate the reproduction number R_0 for this model, and show that the use of the SIT will lead to a lower total number of human infections than in a similar situation with no mosquito controls. We also perform numerical simulations to illustrate our results.

KEYWORDS: Differential equations, SIR model, Sterile/Incompatible Insect Technique, reproduction number R_0, next generation matrices.

MSC: 92D30

RESUMEN
En este trabajo introducimos un modelo de EDOs que combina las ecuaciones de un modelo de poblaciones de mosquitos con el modelo epidemiológico SIR (Susceptibles, Infectados, Recuperados). Nuestro objetivo es mejorar la comprensión de la evolución de una epidemia de dengue en una región donde se aplica la Técnica del Insecto Estéril (SIT), teniendo en cuenta tanto la población de mosquitos como la población humana. Se calcula el coeficiente de reproducción R_0 para este modelo, y se demuestra que el uso de la SIT lleva a un total de infecciones humanas menor que en una población similar sin un método de control de vectores.

PALABRAS CLAVE: Ecuaciones diferenciales, modelo SIR, Técnica del Insecto Estéril/Incompatible, número de reproducción R_0, matrices de la siguiente generación.

1. INTRODUCTION

Mosquitoes of the *Aedes* genus are vectors for multiple infectious viral diseases, most notably dengue, which causes millions of infections and thousands of deaths each year, especially in Latin America; according to Panamerican Health Organization (PAHO) statistics [17], during epidemiological weeks $1-49$ of 2023, there have been approximately 4.2 million reported cases, including more than 6500 severe cases and 2050 deaths. Dengue has only two licensed vaccines, with limited application: Dengvaxia [18] and Qdenga [7]. This shortage of effective, widely available vaccines means the most efficient method for preventing and controlling outbreaks of these arboviruses is to lower or eliminate the vector hosts, that is, the mosquito populations. However, the use of insecticides over a prolonged period of time has issues, for example increased resistance to insecticides [19]. Therefore in recent years alternative control methods have been employed to control

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mosquito populatios, and one of these methods is the Sterile/Incompatible Insect Technique (SIT-IIT).

The SIT was first proposed by Raymond C. Bushland and Edward F. Knipling [13] in the 1950s. It is a technique that has been employed to eliminate and control different pests and vectors, for example screw worms and fruit flies (see [6] for a detailed list of SIT trials and programs). It consists of the breeding and release of a large number of sterilized males into the wild. This increases the probability that females in the already present population will mate with the introduced sterile males, thus producing no viable offspring. Over time, this leads to a progressive reduction of the total population. In the case of the Incompatible Insect Technique (IIT) for mosquitoes, the released mosquitoes are not sterilized chemically or with radiation exposure, but rather infected with bacteria of the genus *Wolbachia*, which shortens their lifespan and reduces their capacity for transmitting dengue [11]. In addition, the Wolbachia bacteria cause cytoplasmic incompatibility: an uninfected female that mates with a *Wolbachia* infected male will not have viable offspring [21]. On the other direction, Wolbachia is inherited from infected female mosquitos to their descendants, regardless of the infection status of the male mosquito they mate with. When only Wolbachia-bearing incompatible males are released, the IIT is equivalent to the SIT; when both male and female Wolbachia-infected mosquitoes are released, the expected result is that the existing population will be replaced by the Wolbachia-infected population [10].

The SIT/IIT has already been applied with success to reduce mosquito populations in several regions, such as Polynesia and Reunion Island (see e.g. [22], [16]). Pilot field studies in Cuba and Northern Italy showed the reduction of egg densities and mosquito populations' fertility after applying the SIT [4] [8]. More recently, combined SIT/IIT releases have been done in China [25] and Thailand [12], while the release of Wolbachia-infected mosquitoes in the city of Yogyakarta, Indonesia, led to a reduction of 77% in the number of dengue cases [23].

2. MODELLING

The classic SIR model for humans divides the human population in three compartments: susceptible, infectious and recovered. When a susceptible individual comes into contact with an infectious individual, there's a chance the susceptible individual will become infected; on the other hand, infected individuals have a chance of either recovering or dying of the disease. The equations of the classic SIR model are as follows [9]:

$$\begin{cases} \frac{dS}{dt} = -\beta \frac{IS}{N} \\ \frac{dI}{dt} = \beta \frac{IS}{N} - \gamma I \\ \frac{dR}{dt} = \gamma I \\ S(0), I(0), R(0) \geq 0 \\ N = S(t) + I(t) + R(t) = S(0) + I(0) + R(0) \end{cases} \tag{2.1}$$

In this model, the total human population is denoted by $N = S + I + R$; we note that $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, so the human population is considered to be constant over time. This means that the dynamics of the epidemic are considered to be much faster than the birth and death of the human population. The fraction $\beta \frac{IS}{N}$ represents the transmissions from infected to susceptible individuals, where β is the transmission rate, while γ is the recovery rate.

In [15], the authors perform a systematic review of mathematical models of dengue transmission and vector control methods during the decade $2010 - 2020$. Although the reviewed models encompass a wide variety of objectives and modelling methods, a majority of the models are deterministic models that either focus on either the mosquito populations with ODE/PDE systems, or use SIR/SEIR epidemiology models for the dengue transmission. Some models that include both mosquito populations and SIR equations are [3] and [11], as well as [20] and [14] in more recent years. The main differences between these works and ours is the

introduction of a strong Allee effect (which will be described further below) following the work of Strugarek, Bossin and Dumont [22], and including both the aquatic phase and the male mosquitos, as opposed to only including the female mosquitos, divided into susceptible and infected compartments, and using a logistic growth model [20].

Our model consists of seven ordinary differential equations, four of them for the female and male adult mosquitos and three of them for the susceptible, infected and recovered humans. We assume that the dengue mortality in humans is low enough to disregard. We include vital dynamics for the human population in our model by setting equal birth and death rates, so that the human population renews while its total remains constant over time. We also assume there is a single strain of dengue present, and that the recovered humans become immune to this strain for the rest of their lives. Additionally, we assume that the mosquitos, once infected, become carriers for the rest of their lives (i.e. there's no Recovered compartment for the mosquitos) and that a dengue infection does not impact the mosquito mortality rate. The transmission of dengue from infected to susceptible individuals can only happen when a infected female mosquito bites a susceptible human, or when a susceptible female mosquito bites an infected human; male mosquitos do not bite humans.

We take the system of equations (2.1) in our previous work [1], without the diffusion operators, as the base for modelling the mosquito population dynamics:

$$\begin{aligned}
&\frac{dE}{dt} = b(1-\frac{E}{K})F - (\nu_E+\mu_E)E,\\
&\frac{dM}{dt} = (1-r)\nu_E E - \mu_M M,\\
&\frac{dF}{dt} = r\nu_E E(1-e^{-A(M+\gamma_s M_s)})\frac{M}{M+\gamma_s M_s} - \mu_F F,\\
&\frac{dM_s}{dt} = u - \mu_s M_s.
\end{aligned} \tag{2.2}$$

In this system, the mosquito population is divided into the following compartments: the aquatic phase (eggs, larvae and pupae) is denoted E, while M and F denote respectively the number of adult wild males and adult females which have been fertilized; M_s is the number of sterile mosquitoes which are released, the release function being denoted $u = u(t)$.

We assume that the dynamic of the egg compartment is fast, which boils down to replacing the first equation by

$$\frac{dE}{dt} = 0 \Rightarrow E = \frac{KbF}{K(\nu_E+\mu_E)+bF}$$

We substitute E in the second and third equations and include the SIR equations, taking into account the female mosquito-human interactions, to arrive at our model:

$$\begin{cases}
F = F_s + F_i, N = S + I + R\\
\frac{dF_i}{dt} = \alpha\frac{F_s I}{N} - \mu_F F_i\\
\frac{dF_s}{dt} = \frac{Kbr\nu_E MF\left(1-e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} - \alpha\frac{F_s I}{N} - \mu_F F_s\\
\frac{dM}{dt} = \frac{(1-r)\nu_E KbF}{K(\mu_E+\nu_E)+bF} - \mu_M M\\
\frac{dM_s}{dt} = u - \mu_{M_s} M_s\\
\frac{dS}{dt} = -\beta\frac{F_i S}{N} + \delta N - \delta S\\
\frac{dI}{dt} = \beta\frac{F_i S}{N} - \gamma I - \delta I\\
\frac{dR}{dt} = \gamma I - \delta R
\end{cases} \tag{2.3}$$

The variables and parameters of our model are listed below. First we will list the variables:

- F_s Susceptible female mosquitos
- F_i Dengue infected female mosquitos
- $F = F_s + F_i$ Total female mosquitos
- M Wild male mosquitos
- M_s Introduced sterile male mosquitos
- S, I, R Susceptible, Infected and Recovered humans respectively
- $N = S + I + R$ Total humans
- $u = u(t)$ Control function for sterile males' release

The parameters of our hybrid model are the following:

- b Female mosquito birth rate
- r Proportion of eggs that are female
- K is an upper bound for the environmental capacity of the aquatic phase, taking also into account intraspecies competition
- ν_E is the transition rate from the aquatic phase to the adult phase
- μ_M, μ_{M_s} Wild and sterile male mosquito death rates respectively
- μ_F Female mosquito death rate
- γ_s Competitiveness of the introduced sterile male mosquitos compared to their wild counterparts
- A Allee effect parameter
- γ Human recovery rate
- β Transmission rate from infected female mosquito to susceptible human
- α Transmission rate from infected human to susceptible female mosquito
- δ Human replacement rate

The fractions $\beta\frac{F_i S}{N}$ and $\alpha\frac{F_s I}{N}$ represent the transmissions from infected female mosquitos to susceptible humans and from infected humans to susceptible female mosquitos, with respective transmission rates α and β. We assume that the birth and death rates for the human population are both equal to δ, so that we have an inflow of newborns into the susceptible compartment at a rate of δN and an outflow of deaths in each compartment at rates $\delta S, \delta I, \delta R$ respectively. Just like in the classic model 2.1, we have $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$, so the human population remains constant over time; we note however that unlike 2.1 where $\frac{dR}{dt} \geq 0$, the inclusion of the birth and death rates for the human population means that $\frac{dR}{dt}$ can be negative in our model if I is close to zero.

The term $(1 - e^{-A(M+\gamma_s M_s)})$ has been introduced to model a strong Allee effect. The strong Allee effect describes a relation between a population's size (or density) and its growth rate: when the population size is below a certain threshold, its growth rate will be negative. For mosquito populations, if the proportion of fertile males is close to zero, then it will be very difficult for a female to find a fertile male to mate with during her whole life, and therefore a strong Allee effect is present.

The competitiveness parameter γ_s represents the ability for sterile males to mate with females, compared to their wild counterparts; for example, $\gamma_s = 1$ would mean both sterile and wild males are evenly matched when competing for females, while $\gamma_s < 1$ means wild mosquito males have an advantage over the introduced sterile males. This is reflected by the fraction $\frac{M}{M+\gamma_s M_s}$, which corresponds to the adjusted probability that a female mates with a wild male.

We remark that we have changed the Allee effect parameter β and the competitiveness parameter γ in [1] to A and γ_s respectively in order to avoid confusion and keep β and γ as parameter names in the SIR part of our model. All parameters are taken as constant over time and strictly positive; in particular, the proportion of female eggs r, the mosquito transition rate ν_e, the mosquito death rates μ_M, μ_{M_s}, μ_F, the human recovery rate γ and the human replacement rate δ all lie in the interval $(0, 1)$, due to their biological interpretations.

3. CALCULATION OF THE BASIC REPRODUCTION NUMBER R_0

To calculate the basic reproduction number R_0 we follow the strategy developed in [5]. The idea is to study the stability of the disease free equilibrium for a reduced system obtained by only considering the infected states. The condition $R_0 < 1$ guarantees the stability of this disease free equilibrium, which mean that a small introduction of infected individuals will not generate an epidemic.

We begin by finding the infection free steady state: $F_i = 0$, $F = F_s = F^*$, $M = M^*$, $S = N$, $I = R = 0$. We then focus on the infection subsystem:

$$\begin{cases} \frac{dF_i}{dt} = \alpha \frac{F_s I}{N} - \mu_F F_i \\ \frac{dI}{dt} = \beta \frac{F_i S}{N} - \gamma I - \delta I \end{cases}$$

We perform a linearization around the infection free steady state $F_i = 0, I = 0$. After substituting $F_s = F^*$ on the first equation and $S = N$ on the second equation, taking the partial derivatives with respect to F_i and I and evaluating at $F_i = 0, I = 0$, we get the following linearized infection subsystem:

$$\begin{cases} \frac{dF_i}{dt} = \frac{\alpha F^*}{N} I - \mu_F F_i \\ \frac{dI}{dt} = \beta F_i - (\gamma + \delta) I \end{cases}$$

We decompose the right hand side into a transmission matrix T, which contains the terms involved in the transmission of the disease, and a transition matrix Σ, which contains the terms involved in the transition out of the infected status, to either death or recovery:

$$T = \begin{pmatrix} 0 & \frac{\alpha F^*}{N} \\ \beta & 0 \end{pmatrix}, \Sigma = \begin{pmatrix} -\mu_F & 0 \\ 0 & -(\gamma + \delta) \end{pmatrix},$$

The next generation matrix is defined as $K = -T\Sigma^{-1}$, and in our model it equals

$$K = \begin{pmatrix} 0 & \frac{\alpha F^*}{N(\gamma+\delta)} \\ \frac{\beta}{\mu_F} & 0 \end{pmatrix}$$

Thus R_0, which is the dominant eigenvalue of K [5], equals $\sqrt{\frac{\alpha\beta F^*}{\mu_F(\gamma+\delta)N}}$.

Let's consider now a constant control function $u(t) = c > 0$. The equation for the sterile males with $M_s(0) = 0$ has the explicit solution $M_s(t) = \frac{c}{\mu_s}(1 - e^{-\mu_s t})$, which tends to $M_s^* = \frac{c}{\mu_s}$ when $t \to +\infty$. For an infection-free equilibrium, $\frac{dF}{dt} = 0$ reduces to

$$\frac{Kbr\nu_E M^* F^* \left(1 - e^{-A(M^* + \gamma_s M_s^*)}\right)}{(K(\mu_E + \nu_E) + bF)(M^* + \gamma_s M_s^*)} - \mu_F F^* = 0 \tag{3.1}$$

while $\frac{dM}{dt} = 0$ is equivalent to

$$\frac{(1-r)\nu_E KbF^*}{K(\mu_E+\nu_E)+bF^*} - \mu_M M^* = 0 \Rightarrow M^* = \frac{(1-r)\nu_E KbF^*}{\mu_M(K(\mu_E+\nu_E)+bF^*)}$$

Lemma 1 *Let* $g(F,M,M_s) = \frac{Kbr\nu_E MF\left(1-e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} - \mu_F F$. *Then* $g(F,M,M_s)$ *is decreasing in* M_s *for all* $M, F > 0$.

Proof: Setting aside the positive factors that do not depend on M_s, we only need to analyze the monotony of $\frac{1-e^{-A(M+\gamma_s M_s)}}{M+\gamma_s M_s}$. Let $f(x) = \frac{1-e^{-A(M+x)}}{M+x}$: we have that

$$f'(x) = \frac{(AMe^{-AM} + Axe^{-AM} + e^{-AM})e^{-Ax} - 1}{(M+x)^2}$$

Therefore it is enough to prove that $(AMe^{-AM} + Axe^{-AM} + e^{-AM})e^{-Ax} - 1 < 0$, for all $A, M, x > 0$. From the classic inequality $e^x \geq x+1$, $\forall x \in \mathbb{R}$ (with equality only if $x = 0$) we can deduce $xe^{-x} < 1 - e^{-x}$, $\forall x > 0$. We can apply this inequality twice to get

$$\begin{aligned}(AMe^{-AM} + Axe^{-AM} + e^{-AM})e^{-Ax} - 1 &< e^{-Ax}(1 - e^{-AM} + Axe^{-AM} + e^{-AM}) - 1 \\ &= (Axe^{-AM} + 1)e^{-Ax} - 1 \\ &< (Ax+1)e^{-Ax} - 1 < 0.\end{aligned}$$

Proposition 1 *If the initial conditions* $M(0), F(0)$ *satisfy* $0 < M(0) < \frac{K(1-r)\nu_E}{\mu_M}$, $0 < F(0) < \frac{Kr\nu_E}{\mu_F}$, *then there exists* $c > 0$ *such that if* $M_s(t) > \frac{c}{\mu_s}$, *then* $g(F(t), M(t), M_s(t)) < 0$ *for all* $t > 0$ *large enough.*

Proof: Since $\frac{dM}{dt} = \frac{(1-r)\nu_E KbF}{K(\mu_E+\nu_E)+bF} - \mu_M M < K(1-r)\nu_E - \mu_M M$ and $M(0) < \frac{K(1-r)\nu_E}{\mu_M}$, then $\frac{dM}{dt} < 0$ in a neighborhood of $M = \frac{K(1-r)\nu_E}{\mu_M}$, and therefore $M(t)$ must stay below $\frac{K(1-r)\nu_E}{\mu_M}$ for all $t > 0$. Likewise, $\frac{dF}{dt} = \frac{Kbr\nu_E MF\left(1-e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} - \mu_F F < Kr\nu_E - \mu_F F$, and since $F(0) < \frac{Kr\nu_E}{\mu_F}$, $F(t) < \frac{Kr\nu_E}{\mu_F}$ for all $t > 0$.

On the other hand, in a similar manner to Lemma 4.6 in [1], we have that for $M = 0$, $F > 0$, $\frac{dM}{dt} > 0$, and for $M, F > 0$

$$\frac{dF}{dt} = \frac{Kbr\nu_E MF\left(1 - e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} - \mu_F F$$

is decreasing in M_s as proven above, and increasing in M, since $F > 0$ and $\frac{M}{M+\gamma_s M_s}$, $1 - e^{-A(M+\gamma_s M_s)}$ are both increasing in M. Therefore the mosquito-related half of 2.3 is a monotone system, and nonnegative initial conditions will remain nonnegative for all $t > 0$.

We have thus proven that $F(t), M(t)$ are uniformly bounded for all $t > 0$, and when $M_s \to +\infty$,

$$\frac{Kbr\nu_E M\left(1 - e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} \to 0$$

uniformly on $(F, M) \in [0, \frac{Kr\nu_E}{\mu_F}] \times [0, \frac{K(1-r)\nu_E}{\mu_M}]$. Then by taking $M_s = c^*$ large enough, we can assure $\frac{Kbr\nu_E M\left(1-e^{-A(M+\gamma_s M_s)}\right)}{(K(\mu_E+\nu_E)+bF)(M+\gamma_s M_s)} < \frac{\mu_F}{2}$, and thus for $c = \mu_s c^*$, we have $M_s(t) \to c^*$ when $t \to +\infty$, and $g(F(t), M(t), M_s(t)) < -\frac{\mu_F}{2}F < 0$, for all $t > 0$ large enough as desired.

From $\frac{dM}{dt} = 0$ we have $M^* = \frac{(1-r)\nu_E KbF^*}{\mu_M(K(\mu_E+\nu_E)+bF^*)} < \frac{K(1-r)\nu_E}{\mu_M}$. Isolating F^*, we get $F^* = \frac{K(\nu_E+\mu_E)\mu_M M^*}{K(1-r)\nu_E - \mu_M M^*}$ (since $M^* < \frac{K(1-r)\nu_E}{\mu_M}$ then F^* is well defined and positive).

We first substitute M^* into 3.1 to get

$$\frac{dF}{dt} = 0 \Rightarrow \frac{r\mu_M (M^*)^2 \left(1 - e^{-A(M^* + \gamma_s M_s^*)}\right)}{(1-r)(M + \gamma_s M_s)} - \mu_F F^* = 0$$

We then substitute $F^* = h(M^*)$ to get $\frac{dF}{dt} = 0 \Rightarrow g(h(M^*), M^*, M_s^*) = 0$, where

$$g(h(M^*), M^*, M_s^*) = \mu_M M^* \left[\frac{rM^* \left(1 - e^{-A(M^* + \gamma_s M_s^*)}\right)}{(1-r)(M^* + \gamma_s M_s^*)} - \frac{K(\nu_E + \mu_E)}{K(1-r)\nu_E - \mu_M M^*} \right]. \tag{3.2}$$

Proposition 2 *Let* $g(h(M), M, M_s) = \mu_M M \left[\frac{rM\left(1 - e^{-A(M + \gamma_s M_s)}\right)}{(1-r)(M + \gamma_s M_s)} - \frac{K(\nu_E + \mu_E)}{K(1-r)\nu_E - \mu_M M} \right]$.
Assume that for the given parameters, $g(h(M), M, 0)$ *has three nonnegative roots* $0, M^-, M^+$*, with* $M^- < M^+$*. Then* $g(h(M), M, 0)$ *is bistable, and* $\frac{dF}{dt} = g(h(M), M, 0)$ *has* $0, M^+$ *as stable equilibria and* M^- *as an unstable equilibrium.*

Proof: We have that $g(h(M), M, 0) = \mu_M M \left[\frac{r(1 - e^{AM})}{(1-r)} - \frac{K\mu_F(\nu_E + \mu_E)}{K(1-r)\nu_E - \mu_M M} \right]$. $M = 0$ is always a root for all parameters; set $g_1(M) = \frac{r(1 - e^{AM})}{(1-r)}$, $g_2(M) = \frac{K\mu_F(\nu_E + \mu_E)}{K(1-r)\nu_E - \mu_M M}$. Then for $M > 0$, $g_1(M), g_2(M)$ are both increasing, but $g_2(M)$ is convex and $g_1(M)$ is concave, therefore $g_1(M) - g_2(M) = 0$ has at most two positive roots, and if there are two positive roots M^-, M^+ then neither of them is a double root. Since $g(h(M), M, 0) \sim -\frac{\mu_M \mu_F (\nu_E + \mu_E)}{(1-r)\nu_E} M$ when $M \to 0^+$, then $g < 0$ when $M \in (0, M^-)$, $g > 0$ when $M \in (M^-, M^+)$ and $g'(0), g'(M^+) \neq 0$, therefore g is bistable, $0, M^+$ are stable equilibria, and M^- is an unstable equilibrium.

It follows that as the value of $u(t) = c$ increases, then the limit M_s^* increases; for values of c small enough so that $g(h(M), M, c/\mu_s)$ remains bistable, the largest stable positive root M^* of $g(h(M), M, c/\mu_s)$ decreases as c increases, due to the monotonicity of g respect to M_s. Since $M^* = \frac{(1-r)\nu_E K b F^*}{\mu_M (K(\mu_E + \nu_E) + bF^*)}$ is increasing with respect to F^*, decreasing M^* also decreases F^*, which in turn decreases $R_0 = \sqrt{\frac{\alpha\beta F^*}{\mu_F(\gamma + \delta)N}}$. Under the hypothesis of Proposition 1 for the initial conditions, eventually for c large enough, all nonnegative equilibria have $M^* = F^* = 0$. This means increasing the release of sterile males will decrease the value of R_0, until for a large enough release the only equilibrium is the mosquito-free equilibrium, at which point $R_0 = 0$.

4. NUMERICAL SIMULATIONS

The values of most mosquito-related parameters are taken from Table 3 in [22] and are listed below, in Table 1.

Parameter	A	b	r	μ_E	ν_E	μ_F	μ_M	γ_s	μ_s
Value	10^{-2}	10	0.49	0.03	0.05	0.04	0.1	1	0.12
Units		day^{-1}		day^{-1}	day^{-1}	day^{-1}	day^{-1}		day^{-1}

Table 1: Values of the mosquito parameters used for the numerical simulations. These values are taken from [22].

The parameter K, which gives an upper bound for the environmental carrying capacity of the aquatic phase, is very hard to estimate in field conditions; we will take $K = 5000$ for our numerical simulations. We take the dengue transmission rate and recovery rate parameters from the vector-host model in [24]. Additionally, we will set $\delta = 0.001$ as the human replacement rate.
All numerical simulations were performed in MATLAB R2018a, using the `ode45` solver to find numerically the solutions of our ODE system. We will first find the solutions of our ODE system, focusing on the mosquito

Parameter	α	β	γ	δ
Value	1.255	1.361	0.224	0.001

Table 2: Values of the dengue parameters used for the numerical simulations. These values are taken from [24].

variables (F, M, M_s), with zero infected mosquitos and humans, and different constant values for our release function $u(t)$. Our initial conditions will be $F(0) = F_s(0) = M(0) = 1000$, $F_i(0) = M_s(0) = I = R = 0$, $N = S = 10000$, and the values of the release function $u(t)$ will be $u(t) = 0, 1000, 10000$ respectively.

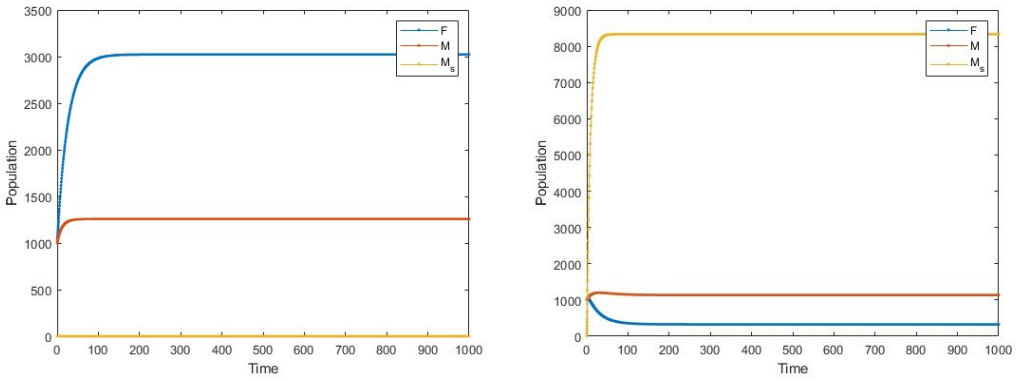

Figure 1: Mosquito variables, sterile male release $u(t) = 0$ on the left and $u(t) = 1000$ on the right.

Figures 1 and 2 show the evolution of the mosquito populations over time for sterile male releases of $u(t) = 0$, $u(t) = 1000$ and $u(t) = 10000$ respectively. We observe that in the absence of sterile males the mosquito population reaches an equilibrium $(F, M, M_s) \approx (3023, 1258, 0)$, that for a low release $(u(t) = 1000)$ a smaller equilibrium $(F, M, M_s) \approx (327, 1136, 0)$ is reached, and for a high enough release $(u(t) = 10000)$ we succeed in leading the wild mosquito population towards extinction.

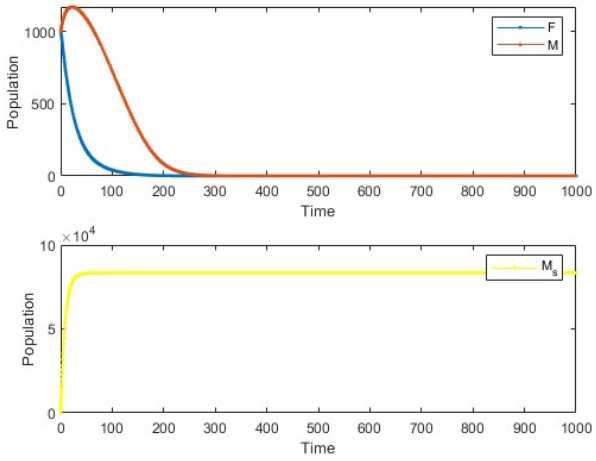

Figure 2: Mosquito variables, sterile male release $u(t) = 10000$.

Next we will take as initial conditions $F(0) = F_i(0) = M(0) = 1000$, $F_s(0) = M_s(0) = R = 0$, $N = 10000$, $I = 1000$. Most of the initial conditions are the same as in the previous simulations, but now we have a nonzero amount of initially infected mosquitos and humans. Once again, the values of the release function $u(t)$ will be $u(t) = 0, 1000, 10000$ respectively, and for this set of simulations we will focus on the human variables (S, I, R).

Figure 3 shows, in the absence of a sterile male release, the evolution of the human variables over time on the left, while the right graph zooms in on the susceptible and infected variables S, I. We note that the variables converge towards a nonzero equilibrium $(S, I, R) \approx (198, 44, 9758)$, which indicates the dengue outbreak becomes endemic, with the transfer rates between the S, I, R compartments balancing each other.

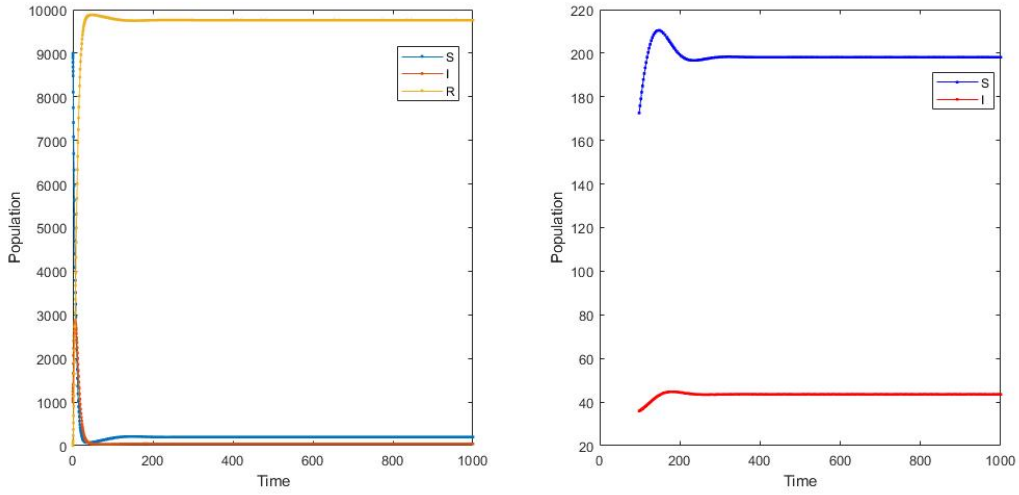

Figure 3: Human variables, sterile male release $u(t) = 0$.

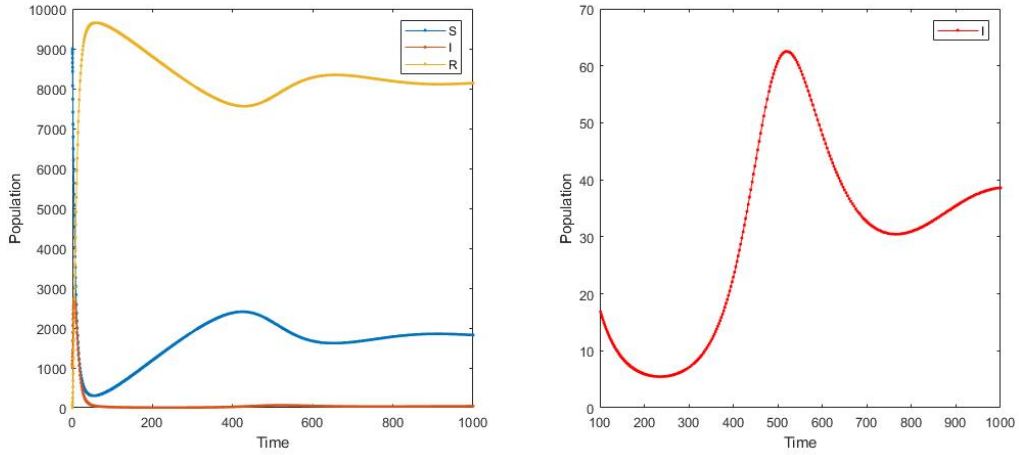

Figure 4: Human variables, sterile male release $u(t) = 1000$.

Likewise, figure 4 shows, for a sterile male release of $u(t) = 1000$, the evolution of the human variables

over time on the left, while the right graph zooms in on the infected variable I. Compared to the previous simulation, we note that this sterile male release is not enough to eradicate the mosquito population, and consequently the number of infected humans bounces back before converging to a equilibrium $(S, I, R) \approx (1823, 39, 8138)$ with a slightly lower infected value. On the other hand, the equilibrium values for the susceptible and recovered values are significantly higher and lower respectively.

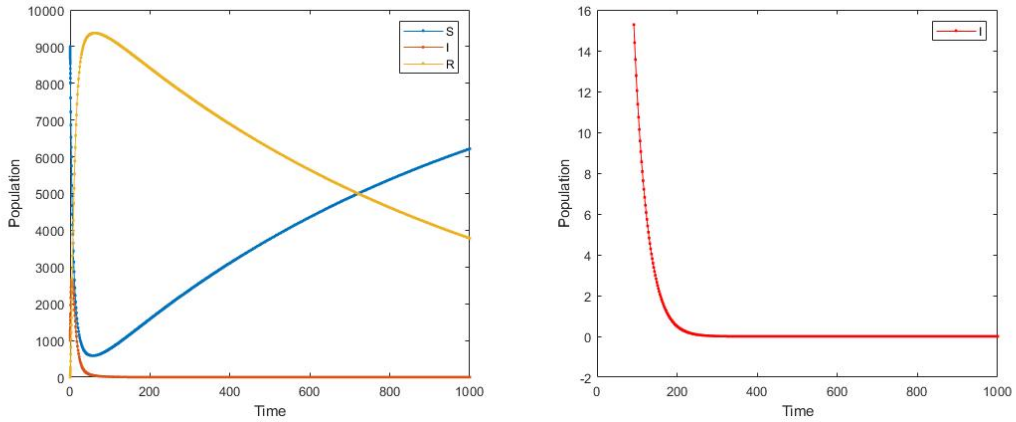

Figure 5: Human variables, sterile male release $u(t) = 10000$.

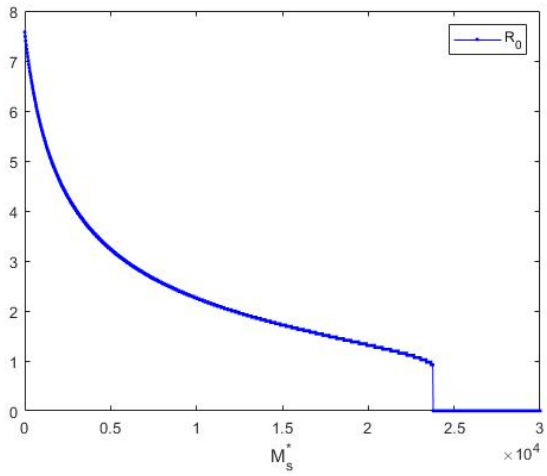

Figure 6: Graph of the reproduction number R_0 as a function of the sterile male release M_s^*.

Finally, for a sterile male release of $u(t) = 10000$, figure 5 shows that the infected variable I converges to zero, which in turn will lead the recovered variable R and the susceptible variable S to converge to 0 and N respectively. We conclude our numerical simulations with a graph of the reproduction number R_0 in terms of the sterile male equilibrium M_s^*, shown in figure 6. We note that R_0 is indeed decreasing as a function of M_s^*, and it drops off to zero after reaching a left limit of $R_0 \approx 0.9246$ for $M_s^* = 23754$; this indicates that $M_s^* = 23754$ is close to the critical value M_s^* for which the function $g(F^*, M^*, M_s^*)$ crosses from being a bistable function with three nonnegative roots to having the zero-mosquito equilibrium as its only nonnegative root.

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5. CONCLUSIONS

In this work we have presented a hybrid model than combines a model for a mosquito population on which the Sterile Insect Technique is applied, with the classic SIR epidemiology model. We show that increasing the release of sterile male mosquitoes leads to the extinction of the wild mosquito population. We find an explicit formula for the basic reproduction number R_0 that links the spread of the epidemic with the larger stable equilibrium of the female mosquito population, which is reduced by increasing the density of the sterile male release. We conclude that by linking the mosquito population equations with the SIR epidemiology equations, we get a more direct picture where it is clearer than the release of sterile males can have an impact not only on the mosquito population, but also on the spread of an arbovirus epidemic.

The numerical simulations performed in this work are just a preliminary exploration for a very simplistic release function $u(t) = c$ constant, and we would like to investigate more realistic release functions, such as a discrete instant release $u(t) = c$ for $t = 0$ and $u(t) = 0$ for $t > 0$, or periodic discrete releases, for which we can pose optimal control problems and hopefully obtain optimality conditions, in a similar manner as [2]. We expect that as long as the solutions of the sterile male equation $\frac{dM_s}{dt} = u(t) - \mu_{M_s} M_s$ are such that $M_s(t)$ is kept at a high enough value for a significant period of time, then the main results of this work, namely the extinction of the wild mosquito population and the decrease of the reproduction number R_0, should still hold.

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