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Sterile insect technique with accidental releases of sterile females. Impact on mosquito-borne diseases control when viruses are circulating

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Abstract

The sterile insect technique (SIT) is a technique to control some vectors of diseases by releasing sterile males. However, during these releases, sterilized females can be (accidentally) released and since only females are vectors of diseases, it is important to study their impact when arthropod viruses are circulating. To that aim, we develop and study an entomological-epidemiological model, considering either permanent or periodic releases. Qualitative analysis of the continuous and periodic models is conducted. We highlight a critical sterile males release rate, Λ_M^{crit}, above which the control of wild population is always effective, using massive releases. Estimating the basic reproduction number of the epidemiological model, $\mathcal{R}_0^2$, we show that if it is above a certain threshold, $\mathcal{R}_{0,*}^2$, that depends on the basic offspring number, $\mathcal{N}$, and the release rate of sterile females, the epidemiological risk can only be controlled using (very) massive releases. Otherwise, we can estimate the basic reproduction number of the SIT epidemiological model, $\mathcal{R}_{0,SIT}^2$, that shapes the stability property of the (periodic) disease-free equilibrium. We show that it might be possible to take $\mathcal{R}_{0,SIT}^2$ below 1 using non-massive, but large enough, releases. However, practically, it seems more efficient to consider massive releases, followed by small releases once the vector population is small enough.

In addition to SIT, we also recommend mechanical control, i.e. the reduction of breeding sites, that greatly improves the efficacy of SIT, in terms of duration or size of the releases.

Our results reveal that outside an epidemic period, the release of sterile females is not an issue, as long as the sterile males release rate is greater than Λ_M^{crit}. Within an epidemic period, we show that sterile females releases do not really impact the SIT efficiency, as long as the release rate, Λ_F, is lower than a critical value, Λ_F^{crit}, that depends on the mosquito and epidemiological threshold parameters, $\mathcal{N}$, and $\mathcal{R}_0^2$. To illustrate numerically our theoretical results, we consider Dengue parameters. We estimate all thresholds and also the effective reproduction number, $\mathcal{R}_{eff}^2$, and highlight the importance of early permanent or periodic SIT control to prevent or mitigate the risk of a Dengue epidemic, with and without sterile females releases.

Keywords: Mosquito-borne disease, sterile insect technique, sterile female, vector control, mechanical control, monotone system, epidemiological system, impulsive differential equation, threshold parameters, dengue

1. Introduction

Vector-borne diseases (VBD), including mosquito-borne diseases, are still considered as serious threats to the health of societies around the world. The major global VBD of humans include malaria, dengue, lymphatic filariasis, schistosomiasis, chikungunya, onchocerciasis, Chagas disease, leishmaniasis, Zika, yellow fever and Japanese encephalitis. According to WHO [22], the major VBD together account for around 17% of the estimated global burden of communicable diseases and claim more than 700 000 lives every year. The burden is highest in tropical and subtropical areas. More than 80% of the global population live in areas at risk from at least one major VBD. Many of these VBD are co-endemic, and it is estimated that more than half of the world's population live in areas where two or more VBD are present [22].

Despite the progress in knowledge on VBD, for most of them, the major problem is the absence of effective drugs and vaccines. In addition, climate changes and travels have increased the area where these diseases can occur, even in Europe, locally [23, 25]. That is why, in the last decades, the development of (sustainable) vector control methods, which can be broadly classified into chemical- and non–chemical-based tools, has become one of the most challenging issues to reduce the impact of human vector borne diseases.

Chemical control consists on the use of massive spraying of larvicide and/or adulticide, like Deltamethrin for mosquitoes. Even being efficient to reduce adult populations, it can be very detrimental to the environment, and also, vectors can develop resistance. This is actually the case in French West Indies [2]. Therefore, it is desirable to have alternative eco-friendly controls. Several non-chemical and eco-friendly control techniques, including the sterile insect technique (SIT), have been developed or are under development. However, the process to reach field applications is long and complex.

Modeling, and in particular mathematical modeling, has become a useful tool in human epidemiology since the pioneering works of Sir R. Ross and his malaria model [24]. Numerous models have been developed to understand the dynamics of diseases and pests, to test "in silico" the usefulness or not of control strategies (and their combination).

In this paper, we focus on the sterile insect technique within an epidemiological context. SIT is an old control technique that has been used more or less successfully on the field against various kind of pests or vectors (see [16] for various examples). The classical SIT consists of mass releases of males sterilized by ionizing radiation. The released sterile males transfer their sterile sperms to wild females, which results in a progressive decay of the targeted population. For mosquitoes, other sterilization techniques have been developed using either genetics (the release of insects carrying a dominant lethal technique, in short RIDL technique) or bacteria (wolbachia) [26]. While conceptually very simple, SIT is, in practice, difficult to conduct as it requires mass rearing and sterilization facilities, and an efficient sexing method for production of males only, at an industrial scale.

Various models have been developed for SIT, using discrete or continuous approaches. Our work is a companion paper of [5, 2], where SIT against pests and vectors were considered. In [5], we showed that SIT induces a strong Allee effect, that can be useful to derive appropriate control strategies, based on massive releases followed by small releases. In [2], where diffusion operators to model the spreading of the pest/vector were considered, we showed existence of bi-stable traveling wave solutions that can be used to derive several spatial and also locally-spatial strategies. However, these works stand within an inter-epidemic period where the objective is to reduce the wild population under a certain threshold that can be

fixed based on several factors, like sterile insects production capacity, size of the targeted area, etc.

Here we consider that a virus is circulating, and that SIT is used to reduce the epidemiological risk, i.e. to drive or to maintain the basic reproduction number below 1. As it is well-known for mosquito-borne diseases, only female mosquitoes are the vector of transmission, because they are blood feeding, and preferably on humans. Thus, their control is the goal of any vector control policy. That is why the (accidental) release of sterile females is questionable (at least for the health authorities). Indeed, during the sterilization process, in order to produce sterile males only, it is necessary to eliminate/separate the females. Up to now, the sex-separation system is mechanical as male nymphs are (in general) smaller than female nymphs. However, a certain number of female nymphs can accidentally fall in the male nymphs bucket and, then, be irradiated to become fully sterilized. Releasing a small amount of (sterile) females is not really problematic during an inter-epidemic period because they are fully sterile, but can be problematic when viruses are circulating, since sterile females can transmit viruses, because they will feed blood on humans. That is why, we consider the releases of sterile females in our model, in order to define if and how they can impact the SIT strategy, and what should be the upper bound limit for the release of sterile females. In the literature [19], releasing 4% of Aedes females is considered as acceptable. Like in [14], we will also consider mechanical control (reduction of breeding sites) and show its efficacy when it is combined with SIT control.

The outline of the paper is as follows. In the next section, we present the full epidemiological-entomological SIT model. Then, in section 3, page 6, we briefly recall the results on the entomological models developed in [5]. In section 4, page 10, following [14], we study the epidemiological model without SIT. In section 5, page 12, the full epidemiological SIT model is studied and the theoretical results discussed in terms of control strategy. The SIT epidemiological model with impulsive periodic releases is studied in section 6, page 19. Finally, numerical simulations, related to *Aedes albopictus* and dengue, are provided in section 7, page 28, to discuss the timing of SIT control strategies, with and without mechanical control, with and without release of sterile females, in order to reduce the epidemiological risk. The paper ends with a conclusion in section 8, page 37.

2. An epidemiological model with SIT releases

Our model is based on the entomological model studied in [5], and initially developed in [3]. We also follow [14], except for the aquatic stage, and add the epidemiological stages related to the wild and sterile (mature) females, F_S, F_E, F_I, S_S, S_E, and S_I, respectively. We assume that the total population of humans, $N_h = S_h + I_h + R_h$, is positive and constant. When (wild and sterile) female mosquitoes are infected, we assume that their mortality rate can be impacted (see [12, 14] for the Chikungunya case). We also take into account the extrinsic incubation period of the virus within the vector population, ν_m, which implies to consider three epidemiological states, i.e. the susceptible, exposed and infected states. The previous assumptions complexify the analysis but then the model is more realistic. We don't consider vertical transmission (from infected female mosquitoes) of the virus because it was showed in [1] that the observed percentages $(1 - 4\%)$ of vertical transmission do not play a role in the long term persistence of the virus. A novelty in our model lies in the fact that, accidentally, sterile female mosquitoes are released with sterile males. This is modeled by the parameters Λ_F in equation $(3)_1$, related to the S_S compartment. We point out that the

S_S compartment gathers both sterile females that are released and young wild females that mated with sterile males and, hence, have become fully sterile. Since all sterile females are mixed with sterile males before the release, they are considered as mated and thus do not participate in mating after their release. The model is summarized in the flow diagram 1, page 4, and the full epidemiological SIT model is defined as follows:

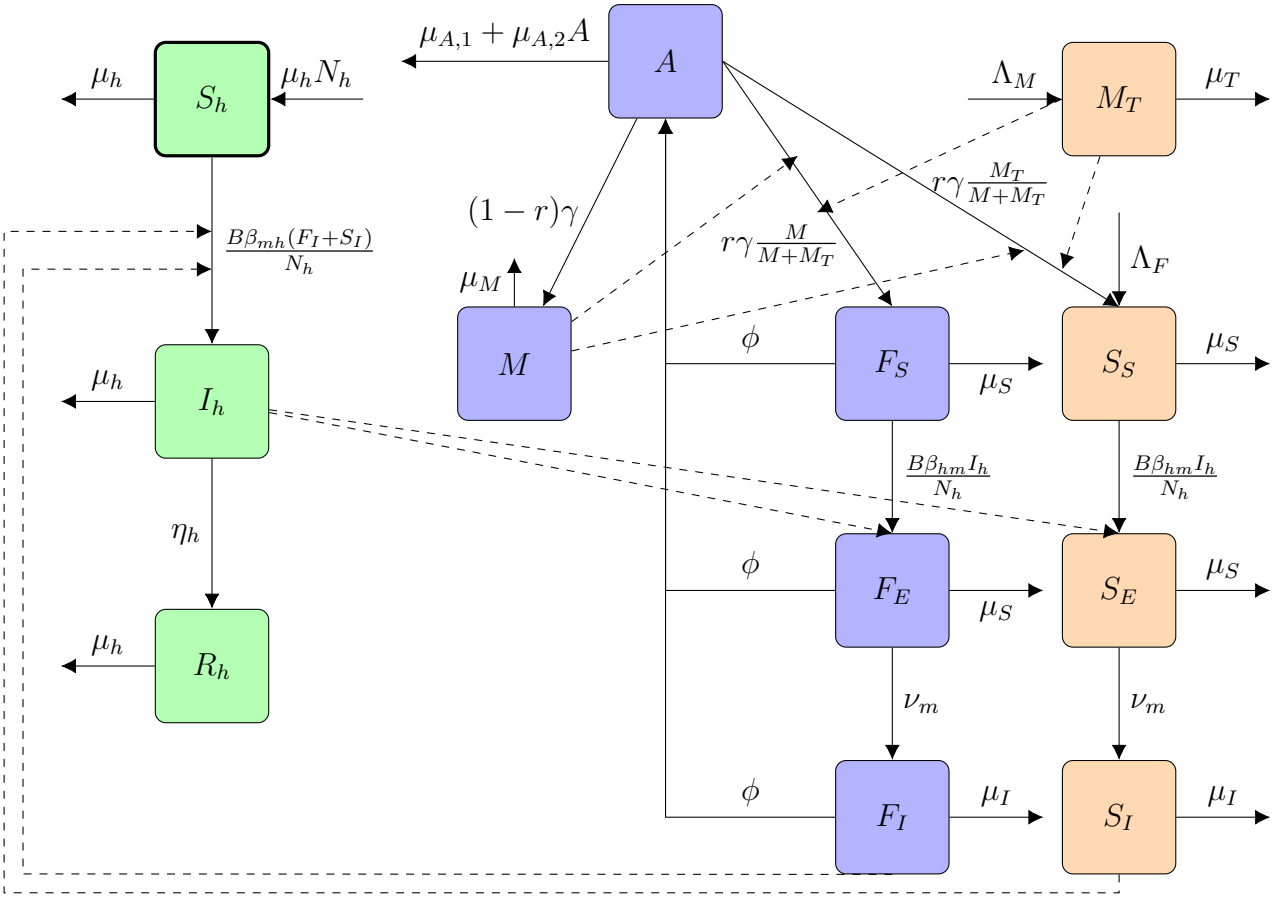

Figure 1: Flow diagram of model (1)-(4).

$$\begin{cases} \dfrac{dS_h}{dt} &= \mu_h N_h - B\beta_{mh}\dfrac{F_I+S_I}{N_h}S_h - \mu_h S_h, \\ \dfrac{dI_h}{dt} &= B\beta_{mh}\dfrac{F_I+S_I}{N_h}S_h - (\eta_h+\mu_h)\,I_h, \\ \dfrac{dR_h}{dt} &= \eta_h I_h - \mu_h R_h, \end{cases} \tag{1}$$

$$\begin{cases} \dfrac{dA}{dt} &= \phi(F_S+F_E+F_I) - (\gamma+\mu_{A,1}+\mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= r\gamma A\dfrac{M}{M+M_T} - B\beta_{hm}\dfrac{I_h}{N_h}F_S - \mu_S F_S, \\ \dfrac{dF_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}F_S - (\nu_m+\mu_S)\,F_E, \\ \dfrac{dF_I}{dt} &= \nu_m F_E - \mu_I F_I, \end{cases} \tag{2}$$

$$\begin{cases} \dfrac{dS_S}{dt} &= \Lambda_F + r\gamma A\dfrac{M_T}{M+M_T} - B\beta_{hm}\dfrac{I_h}{N_h}S_S - \mu_S S_S, \\ \dfrac{dS_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}S_S - (\nu_m + \mu_S)\, S_E, \\ \dfrac{dS_I}{dt} &= \nu_m S_E - \mu_I S_I, \end{cases} \tag{3}$$

and

$$\frac{dM_T}{dt} = \Lambda_M - \mu_T M_T \tag{4}$$

where the parameters and state variables are described in Table 1, page 5.

Symbol	Description	Unit
S_h	Susceptible human	Individuals
I_h	Infected human	Individuals
R_h	Recovered human	Individuals
A	Aquatic stage (gathering eggs, larvae, nymph stages)	Individuals
F_S	Susceptible fertilized and eggs-laying females	Individuals
F_E	Exposed fertilized and eggs-laying females	Individuals
F_I	Infected fertilized and eggs-laying females	Individuals
S_S	Susceptible sterilized females	Individuals
S_E	Exposed sterilized females	Individuals
S_I	Infected sterilized females	Individuals
M	Wild males	Individuals
M_T	Sterile males	Individuals
$1/\mu_h$	Average lifespan of human	Day
$1/\eta_h$	Average viremic period	Day
B	Average mosquito bites	days^{-1}
β_{mh}	Transmission probability from infected mosquito	-
β_{hm}	Transmission probability from infected human	-
ϕ	Number of eggs at each deposit per capita	Day^{-1}
γ	Maturation rate from larvae to adult	Day^{-1}
$\mu_{A,1}$	Density independent mortality rate of the aquatic stage	Day^{-1}
$\mu_{A,2}$	Density dependent mortality rate of the aquatic stage	Day^{-1} Individuals $^{-1}$
r	Sex ratio	-
$1/\nu_m$	Average extrinsic incubation period (EIP)	Day
$1/\mu_S$	Average lifespan of susceptible fertilized and eggs-laying female and, susceptible sterilized female	Day
$1/\mu_I$	Average lifespan of infected fertilized and eggs-laying female and, infected sterilized female	Day
$1/\mu_M$	Average lifespan of male	Day
$1/\mu_T$	Average lifespan of sterile male	Days
Λ_M	Sterile male release rate	Individuals $\times$ Day^{-1}
Λ_F	Sterile female release rate	Individuals $\times$ Day^{-1}

Table 1: Description of parameters and state variables of model (1)-(4)

Contrary to [29], we assume a density-dependent mortality rate in the aquatic stage. This may correspond to an intra-specific competition between the larvae stages, for instance. However, the forthcoming methodology could be applied for a system where the non-linearity stands for the birth-rate, like in [14, 15, 29].

Remark 1. *In systems (2) and (3), we don't consider explicitly the (mean) mating competitiveness parameter, c_T, of the sterile males. This parameter can be less or greater than one. To take it into account, like in [14, 29], it suffices to replace M_T by $c_T M_T$ and to change the following results accordingly.*

Remark 2. *Model (1)-(4) is not really appropriate for the RIDL approach. Indeed, with the RIDL control technique, sterile females can lay eggs that will hatch and enter the aquatic stage, resulting in an increase in the density-dependent mortality. However, all RIDL pupae are supposed not to survive into the adulthood. Note also that, so far, the RIDL approach has only been tested on Aedes aegypti.*

3. The wild insect sub-models

The wild insect population model has been studied in [5]. Here, we just recall results obtained in [5].

3.1. The wild insect sub-model without SIT

It is straightforward to deduce from system (2) that dynamics of wild insect without SIT is modelled by system (5):

$$\begin{cases} \dfrac{dA}{dt} &= \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= r\gamma A - \mu_S F_S. \end{cases} \tag{5}$$

Remark 3. *System (5) is very simple: it implies that all emerging immature individuals will become either males, either females (after mating), assuming implicitly that $0 < r < 1$. Since we consider a mosquito population, we have $r \approx 0.5$, such that there are always adults of both sex, considering at least either $A(0) > 0$ or $F_S(0) > 0$.*

The basic offspring number related to model (5) is

$$\mathcal{N} = \frac{r\gamma\phi}{\mu_S(\gamma + \mu_{A,1})}. \tag{6}$$

Setting the right-hand side of (5) to zero we obtain the extinction equilibrium $\mathbf{0}_{\mathbb{R}^3} = (0,0,0)^T$ and the equilibrium $E^* = (A^*, M^*, F_S^*)^T$ given by

$$\begin{cases} A^* &= \dfrac{(\gamma + \mu_{A,1})}{\mu_{A,2}}(\mathcal{N} - 1), \\ M^* &= \dfrac{(1-r)\gamma A^*}{\mu_M} = \dfrac{(1-r)\gamma}{\mu_M}\dfrac{(\gamma + \mu_{A,1})}{\mu_{A,2}}(\mathcal{N} - 1), \\ F_S^* &= \dfrac{r\gamma A^*}{\mu_S} = \dfrac{r\gamma}{\mu_S}\dfrac{(\gamma + \mu_{A,1})}{\mu_{A,2}}(\mathcal{N} - 1). \end{cases} \tag{7}$$

The inequalities between vectors are considered here in their usual coordinate-wise sense. Clearly, $E^* > \mathbf{0}_{\mathbb{R}^3}$ if and only if $\mathcal{N} > 1$. We summarize these results with some more details related to basins of attraction of equilibria in the following theorem.

Theorem 1 ([5]). *Model (5) defines a forward dynamical system on* $\mathcal{D} = \{x \in \mathbb{R}^3 : x \geq \mathbf{0}_{\mathbb{R}^3}\}$. *Furthermore,*

1) If $\mathcal{N} \leq 1$ *then* $\mathbf{0}_{\mathbb{R}^3}$ *is globally asymptotically stable on* $\mathcal{D}$.
2) If $\mathcal{N} > 1$ *then* E^* *is stable with basin of attraction*

$$\mathcal{D} \setminus \{x = (A, M, F_S)^T \in \mathbb{R}^3_+ : A = F_S = 0\},$$

and $\mathbf{0}_{\mathbb{R}^3}$ *is unstable with the non negative* $M-$*axis being a stable manifold.*

Proof. See [5, Theorem 1] where $F = F_S$ and $\mu = \mu_S$. □

3.2. The wild insect sub-model with SIT

We now assume that $\mathcal{N} > 1$. We take into account the constant release of sterile males M_T by adding to model (5) an equation for M_T, and also the accidental release of sterile females by considering the release rate, Λ_F, in the compartment S_S. Altogether, the SIT model becomes

$$\begin{cases} \dfrac{dA}{dt} &= \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= \dfrac{M}{M+M_T} r\gamma A - \mu_S F_S, \\ \dfrac{dS_S}{dt} &= \Lambda_F + \dfrac{M_T}{M+M_T} r\gamma A - \mu_S S_S, \\ \dfrac{dM_T}{dt} &= \Lambda_M - \mu_T M_T. \end{cases} \tag{8}$$

Remark 4. *In system (8), the term* $\dfrac{M}{M+M_T}$ *does not appear in the deposit rate component, in the aquatic/immature component, contrary to some models (see [8, 34], for instance) where we have*

$$\frac{dA}{dt} = \phi \frac{M}{M+M_T} F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A. \tag{9}$$

Implicitly, in (9), it is assumed that daily deposit rate always depends on the proportion of fertile males over the total male population: this is not true. Mosquito females, like Aedes spp, have 3 spermathecae that allow to stock sperms such that in general one mating is sufficient to deposit eggs along their lifespan. Biologically, after emergence, immature or virgin females will enter the mature/fertile females compartment, F_S, only when they mate with fertile/wild males, and, then, are able to deposit viable (hatching) eggs along their lifespan. This is exactly what is modeled in system (8): the income rate in the mature (sterile) female compartment, F_S (S_S), takes into account the fact that immature females have a probability $\dfrac{M}{M+M_T}\left(\dfrac{M_T}{M+M_T}\right)$ *to mate with wild (sterile) males.*

Using (9) is very convenient from a mathematical point of view because all nonlinear terms appear in the same equation, but, biologically, it is false. At some point, it could

be, mathematically, equivalent to consider (9), but we believe that it is better to stick to the biological reality in order to make the model understandable for entomologists, even if, mathematically, it is a bit more challenging.

Assuming t large enough, we may assume that $M_T(t)$ has reached its equilibrium value, $M_T^* := \dfrac{\Lambda_M}{\mu_T}$. In fact, from a practical point of view, the value Λ_M/μ_T can be reached with massive constant and continuous releases of $2\Lambda_M$ during $t = \dfrac{\ln(2)}{\mu_T}$, see also [5, 6]. Hence, model (8) reduces to

$$\left\{\begin{array}{rcl} \dfrac{dA}{dt} &=& \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &=& (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &=& \dfrac{M}{M + M_T^*} r\gamma A - \mu_S F_S, \\ \dfrac{dS_S}{dt} &=& \Lambda_F + \dfrac{M_T^*}{M + M_T^*} r\gamma A - \mu_S S_S, \end{array}\right. \tag{10}$$

where parameters and state variables are described in Table 1, page 5. Since the state variable S_S does not appear in the first three equations of system (10), it suffices to study the following sub-system

$$\left\{\begin{array}{rcl} \dfrac{dA}{dt} &=& \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &=& (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &=& \dfrac{M}{M + M_T^*} r\gamma A - \mu_S F_S. \end{array}\right. \tag{11}$$

In fact we recover the system studied in [5]. Knowing the dynamics of A and M, the dynamics of S_S is deduced by using the fourth equation of system (10).

Let us set

$$Q = \frac{\mu_{A,2}\mu_M}{(\gamma + \mu_{A,1})(1-r)\gamma}, \quad \Lambda_M^{crit} = \frac{\mu_T(\sqrt{\mathcal{N}} - 1)^2}{Q}. \tag{12}$$

Following [5, Theorem 3], we derive existence of equilibria for $0 < \Lambda_M \leq \Lambda_M^{crit}$, and also stability results for system (11), such that we can deduce the following results for system (8):

Theorem 2. *System (8) defines a forward dynamical system on $\mathcal{D}' := \{x \in \mathbb{R}^5 : x \geq 0_{\mathbb{R}^5}\}$. Moreover,*

(1) If $\Lambda_M > \Lambda_M^{crit}$, then equilibrium $\boldsymbol{TE} = \left(0_{\mathbb{R}^3}, \dfrac{\Lambda_F}{\mu_S}, \dfrac{\Lambda_M}{\mu_T}\right)^T$ is unique and globally asymptotically stable on $\mathcal{D}'$.

(2) If $\Lambda_M = \Lambda_M^{crit}$ *then system* (8) *has two equilibria,* $\boldsymbol{TE}$ *and* $\boldsymbol{E}_\dagger = \left(A_\dagger, M_\dagger, F_{S_\dagger}, S_{S_\dagger}, \dfrac{\Lambda_M}{\mu_T}\right)^T$ *where*

$$\begin{cases} M_T^* &= \dfrac{\Lambda_M^{crit}}{\mu_T}, \\ \alpha_\dagger &= \dfrac{1}{2}\left((\mathcal{N} - 1 - QM_T^*)\right) = \sqrt{\mathcal{N}} - 1, \\ M_\dagger &= \dfrac{M_{T_1}}{\alpha_\dagger} = \dfrac{\sqrt{\mathcal{N}} - 1}{Q} > 0, \\ A_\dagger &= \dfrac{\mu_M}{(1-r)\gamma} M_\dagger = (\gamma + \mu_{A,1})\dfrac{\sqrt{\mathcal{N}} - 1}{\mu_{A,2}} > 0, \\ F_{S_\dagger} &= \dfrac{(\gamma + \mu_{A,1} + \mu_{A,2}A_\dagger)A_\dagger}{\phi} > 0, \\ S_{S_\dagger} &= \dfrac{1}{\mu_S}\left(\Lambda_F + r\gamma A_\dagger \dfrac{M_T^*}{M_\dagger + M_T^*}\right) > \dfrac{\Lambda_F}{\mu_S}. \end{cases} \tag{13}$$

The set

$$\{(A, M, F_S, S_S, M_T)^T \in \mathbb{R}^5 : 0_{\mathbb{R}^3} \leq (A, M, F_S)^T < (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

is in the basin of attraction of $\boldsymbol{TE}$*, while the set*

$$\{(A, M, F_S, S_S, M_T)^T \in \mathbb{R}^5 : (A, M, F_S)^T \geq (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

is in the basin of attraction of $\boldsymbol{E}_\dagger$*.*

(3) If $0 < \Lambda_M < \Lambda_M^{crit}$*, then system* (8) *has three equilibria* $\boldsymbol{TE}$*,* $\boldsymbol{E}_1 = \left(A_1, M_1, F_{S_1}, S_{S_1}, \dfrac{\Lambda_M}{\mu_T}\right)^T$ *and* $\boldsymbol{E}_2 = \left(A_2, M_2, F_{S_2}, S_{S_2}, \dfrac{\Lambda_M}{\mu_T}\right)^T$ *where*

$$\begin{cases} M_T^* &= \dfrac{\Lambda_M}{\mu_T}, \\ \alpha_\pm &= \dfrac{1}{2}\left((\mathcal{N} - 1 - QM_T^*) \pm \sqrt{((\mathcal{N} - 1 - QM_T^*)^2 - 4M_T^*Q))}\right), \\ M_1 &= \dfrac{M_T^*}{\alpha_+} > 0, \\ M_2 &= \dfrac{M_T^*}{\alpha_-} > 0, \\ A_{1,2} &= \dfrac{\mu_M}{(1-r)\gamma} M_{1,2} > 0, \\ F_{S_{1,2}} &= \dfrac{(\gamma + \mu_{A,1} + \mu_{A,2}A_{1,2})A_{1,2}}{\phi} > 0, \\ S_{S_{1,2}} &= \dfrac{1}{\mu_S}\left(\Lambda_F + r\gamma A_{1,2}\dfrac{M_T^*}{M_{1,2} + M_T^*}\right) > \dfrac{\Lambda_F}{\mu_S} \end{cases} \tag{14}$$

and $(A_1, M_1, F_{S_1})^T < (A_2, M_2, F_{S_2})^T$*. The set*

$$\{(A, M, F_S, S_S, M_T)^T \in \mathbb{R}^5 : 0_{\mathbb{R}^3} \leq (A, M, F_S)^T < (A_1, M_1, F_{S_1})^T\}$$

is in the basin of attraction of $\boldsymbol{TE}$ *while the set*

$$\{(A, M, F_S, S_S, M_T)^T \in \mathbb{R}^5 : (A, M, F_S)^T > (A_1, M_1, F_{S_1})^T\}$$

is in the basin of attraction of $\boldsymbol{E}_2$.

Remark 5. *If mechanical control is included within the SIT control strategy, this will increase the density-dependent mortality rate,* $\mu_{A,2}$*, in the aquatic compartments, such that, according to* (12)*,* Λ_M^{crit} *will decay. In other words, mechanical control is helpful to decrease the amount of sterile males to release.*

The bifurcation diagram in Fig. 2, page 10, summarizes Theorem 1 and Theorem 2 when $\mathcal{N} > 1$: the blue (red) solid line represents (globally) stable equilibrium, while the blue dotted line represents unstable equilibrium.

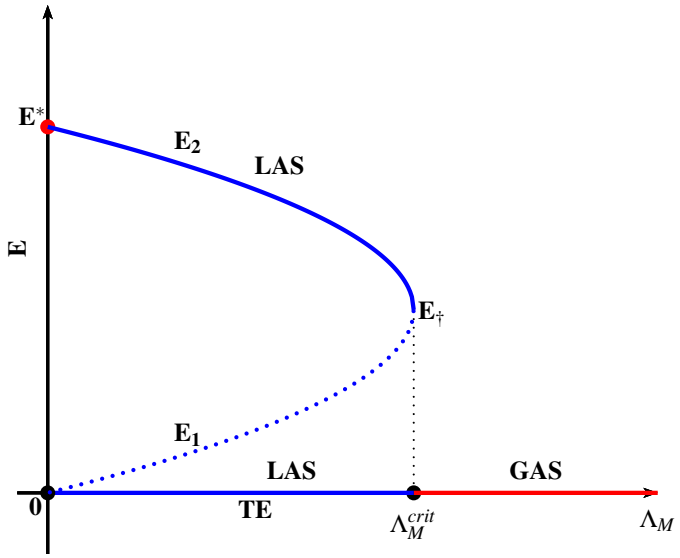

Figure 2: Bifurcation diagram at equilibrium with respect to the values of Λ_M for system (8) and system (10)

4. The vector-borne epidemiological model without SIT

Most of the analysis follows [14], where a vector-borne epidemiological model was studied. Of course only the case $\mathcal{N} > 1$ is worth of investigations and we will consider this assumption for the rest of the paper. The model is given as a system of ordinary differential equations as follows:

$$\begin{cases} \dfrac{dS_h}{dt} &= \mu_h N_h - B\beta_{mh}\dfrac{F_I}{N_h}S_h - \mu_h S_h, \\ \dfrac{dI_h}{dt} &= B\beta_{mh}\dfrac{F_I}{N_h}S_h - (\eta_h + \mu_h)\, I_h, \\ \dfrac{dR_h}{dt} &= \eta_h I_h - \mu_h R_h, \end{cases} \tag{15}$$

where $N_h = S_h + I_h + R_h = N_h(0) > 0$, and

$$\begin{cases} \dfrac{dA}{dt} &= \phi(F_S + F_E + F_I) - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= r\gamma A - B\beta_{hm}\dfrac{I_h}{N_h}F_S - \mu_S F_S, \\ \dfrac{dF_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}F_S - (\mu_S + \nu_m)F_E, \\ \dfrac{dF_I}{dt} &= \nu_m F_E - \mu_I F_I. \end{cases} \tag{16}$$

Let $x(t) = (S_h(t), I_h(t), R_h(t), A(t), M(t), F_S(t), F_E(t), F_I(t))^T$. Let us consider the set

$$\mathcal{D}'' = \mathbb{R}^8_+ = \{x \in \mathbb{R}^8 : x \geq 0_{\mathbb{R}^8}\}.$$

The following result holds true.

Lemma 1. 1. *The set*

$$\Gamma_{\mathcal{N}>1} = \left\{x \in \mathbb{R}^8_+ : S_h + I_h + R_h = N_h; A \leq A^*; M \leq M^*; F_S + F_E + F_I \leq F_S^*\right\}$$

is positively invariant for system (15)-(16) where $(A^*, M^*, F_S^*)^T$ is given by (7). That is, any solution that starts in $\Gamma_{\mathcal{N}>1}$ will remain there.

2. *System (15)-(16) defines a dynamical system on $\mathcal{D}''$.*

Proof. See Appendix A. □

4.1. Equilibria of system (15)-(16)

In this section, we deal with the computation of trivial and non-trivial equilibria of system (15)-(16). It is straightforward to obtain the following results, so its proof is omitted.

Proposition 1. 1. *System (15)-(16) admits a trivial disease-free equilibrium, $TDFE = (N_h, \mathbf{0}_{\mathbb{R}^7})^T$, that always exists.*

2. *System (15)-(16) admits a disease-free equilibrium, $DFE = (N_h, 0, 0, A^*, M^*, F_S^*, 0, 0)^T$, whenever $\mathcal{N} > 1$ and where A^*, M^*, F_S^* are defined in (7).*

4.2. Stability analysis of the DFE

Using the next generation matrix (NGM) approach, see e.g. [30], we derive the basic reproduction number related to system (15)-(16), that is

$$\mathcal{R}_0^2 = \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{mh}}{\mu_I}\frac{B\beta_{hm}}{\eta_h + \mu_h}\frac{F_S^*}{N_h}. \tag{17}$$

We further summarize stability results of the non trivial disease-free equilibrium in the following

Theorem 3. 1. *If $\mathcal{R}_0^2 \leq 1$, then the DFE is globally asymptotically stable.*

2. *If $\mathcal{R}_0^2 = 1$, then system (15)-(16) has a forward bifurcation.*

3. *If $\mathcal{R}_0^2 > 1$, then the DFE is unstable.*

Proof. See Appendix B, page 44. □

4.3. Endemic Equilibrium of system (15)-(16)

Long but straightforward computations lead to

Proposition 2. *When $\mathcal{R}_0^2 > 1$, there exists a unique endemic equilibrium*

$$EE = (S_h^\sharp, I_h^\sharp, R_h^\sharp, A^\sharp, M^\sharp, F_S^\sharp, F_E^\sharp, F_I^\sharp)^T$$

which is locally asymptotically stable.

Proof. See Appendix C, page 47. □

Remark 6. *Again, if mechanical control is considered, then the size of the female population, F_S^*, will decrease, such that $\mathcal{R}_0^2$ will also decrease.*

5. The vector-borne epidemiological model with SIT

In this section, we consider that constant and permanent SIT releases are done as a control tool. Hence, following (10), the dynamics of human and mosquito populations are described by system (18)-(19):

$$\begin{cases} \dfrac{dS_h}{dt} &= \mu_h N_h - B\beta_{mh}\dfrac{F_I + S_I}{N_h}S_h - \mu_h S_h, \\ \dfrac{dI_h}{dt} &= B\beta_{mh}\dfrac{F_I + S_I}{N_h}S_h - \eta_h I_h - \mu_h I_h, \\ \dfrac{dR_h}{dt} &= \eta_h I_h - \mu_h R_h, \end{cases} \tag{18}$$

$$\begin{cases} \dfrac{dA}{dt} &= \phi(F_S + F_E + F_I) - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= \dfrac{M}{M + M_T^*}r\gamma A - B\beta_{hm}\dfrac{I_h}{N_h}F_S - \mu_S F_S, \\ \dfrac{dF_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}F_S - (\nu_m + \mu_S)F_E, \\ \dfrac{dF_I}{dt} &= \nu_m F_E - \mu_I F_I, \\ \dfrac{dS_S}{dt} &= \Lambda_F + \dfrac{M_T^*}{M + M_T^*}r\gamma A - B\beta_{hm}\dfrac{I_h}{N_h}S_S - \mu_S S_S, \\ \dfrac{dS_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}S_S - (\nu_m + \mu_S)S_E, \\ \dfrac{dS_I}{dt} &= \nu_m S_E - \mu_I S_I. \end{cases} \tag{19}$$

We provide qualitative results of system (18)-(19). Let us set

$$u^* = \frac{\Lambda_F + r\gamma A^*}{\mu_S}.$$

5.1. Boundedness of solutions and existence of disease-free equilibria

Based on Lemma 1, page 11, it is straightforward to obtain the following result:

Lemma 2 (Boundedness of solutions)**.** *The set*

$$\Gamma_{\mathcal{N}>1,SIT} = \Big\{x \in \mathbb{R}^{11}_{+} : S_h + I_h + R_h = N_h; (A, M)^T \leq (A^*, M^*)^T; F_S + F_E + F_I \leq F_S^*; \\ S_S + S_E + S_I \leq u^*\}$$

is positively invariant for system (18)-(19) where $(A^*, M^*, F_S^*)^T$ is given by (7).

Proof. Taking into account Lemma 1, page 11, it remains to prove that $S_S + S_E + S_I \leq u^*$. By adding the last three equations of system (19) and using the fact that $A \leq A^*$, one obtains:

$$\frac{d(S_S + S_E + S_I)}{dt} \leq \Lambda_F + r\gamma A^* - \mu_S(S_S + S_E + S_I) = \mu_S(u^* - (S_S + S_E + S_I)). \tag{20}$$

Using $G = (S_S + S_E + S_I) - u^*$ and f the right-hand side of (20), we have

$$\nabla G \cdot f|_{S_S+S_E+S_I=u^*} = 0. \text{ Hence } S_S + S_E + S_I \leq u^*.$$

This ends the proof. □

Using Theorem 2, page 8, we deduce:

Proposition 3 (Trivial and non-trivial disease-free equilibria)**.** *Let Λ_M^{crit} defined by (12), page 8.*

1. *If $\Lambda_M \in (0, \Lambda_M^{crit})$, then system (18)-(19) has two non-trivial disease-free equilibria $DFE_{1,2} = (N_h, 0, 0, A_{1,2}, M_{1,2}, F_{S_{1,2}}, 0, 0, S_{S_{1,2}}, 0, 0)^T$ with $(A_1, M_1, F_{S_1})^T < (A_2, M_2, F_{S_2})^T$ and $A_{1,2}$, $M_{1,2}$, $F_{S_{1,2}}$, $S_{S_{1,2}}$ are given in Theorem 2.*
2. *If $\Lambda_M = \Lambda_M^{crit}$, then system (18)-(19) has a non-trivial disease-free equilibrium $DFE_\dagger = (N_h, 0, 0, A_\dagger, M_\dagger, F_{S_\dagger}, 0, 0, S_{S_\dagger}, 0, 0)^T$ where $A_\dagger$, $M_\dagger$, $F_{S_\dagger}$, $S_{S_\dagger}$ are given in Theorem 2.*
3. *If $\Lambda_M > \Lambda_M^{crit}$, system (18)-(19) has a unique equilibrium, $TDFE = \left(N_h, \boldsymbol{0}_{\mathbb{R}^7}, \frac{\Lambda_F}{\mu_S}, 0, 0\right)^T$.*

Following Theorem 2, in the disease-free case, equilibrium DFE_1 is unreachable because it is always unstable. Therefore, the meaningful disease-free equilibrium of system (18)-(19) is

$$DFE_{SIT_c} = \begin{cases} DFE_2, & \text{when} \quad \Lambda_M \in (0, \Lambda_M^{crit}), \\ DFE_\dagger, & \text{when} \quad \Lambda_M = \Lambda_M^{crit}, \\ TDFE, & \text{when} \quad \Lambda_M > \Lambda_M^{crit}. \end{cases} \tag{21}$$

Using again the NGM-approach, the basic reproduction number of system (18)-(19) is

$$\mathcal{R}^2_{0,SIT_c} = \begin{cases} \dfrac{\nu_m}{\nu_m + \mu_S} \dfrac{B\beta_{mh}}{\mu_I} \dfrac{B\beta_{hm}}{\eta_h + \mu_h} \dfrac{(F_{S_2} + S_{S_2})}{N_h}, & \text{when} \quad \Lambda_M \in (0, \Lambda_M^{crit}), \\ \dfrac{\nu_m}{\nu_m + \mu_S} \dfrac{B\beta_{mh}}{\mu_I} \dfrac{B\beta_{hm}}{\eta_h + \mu_h} \dfrac{(F_{S_\dagger} + S_{S_\dagger})}{N_h}, & \text{when} \quad \Lambda_M = \Lambda_M^{crit}, \\ \dfrac{\nu_m}{\nu_m + \mu_S} \dfrac{B\beta_{mh}}{\mu_I} \dfrac{B\beta_{hm}}{\eta_h + \mu_h} \dfrac{\Lambda_F}{\mu_S N_h}, & \text{when} \quad \Lambda_M > \Lambda_M^{crit}. \end{cases} \tag{22}$$

Remark 7. *In the case of non-massive release, that is* $\Lambda_M \in (0, \Lambda_M^{crit})$, $\mathcal{R}^2_{0,SIT_c}$ *has two parts: the first part*

$$\mathcal{R}^2_{0,SIT_c,W} = \frac{\nu_m}{\nu_m + \mu_S} \frac{B\beta_{mh}}{\mu_I} \frac{B\beta_{hm}}{\eta_h + \mu_h} \frac{F_{S_{2,\dagger}}}{N_h},$$

related to the wild susceptible females and the second part,

$$\mathcal{R}^2_{0,SIT_c,S} = \frac{\nu_m}{\nu_m + \mu_S} \frac{B\beta_{mh}}{\mu_I} \frac{B\beta_{hm}}{\eta_h + \mu_h} \frac{S_{S_{2,\dagger}}}{N_h},$$

related to the sterile susceptible females. The main question is: when $\mathcal{R}^2_{0,SIT_c,W} < 1$, *is it possible that releases of sterile females imply* $\mathcal{R}^2_{0,SIT_c} > 1$*?*

Remark 8. *Since* $F_{S_{2,\dagger}} + S_{S_{2,\dagger}} = \dfrac{r\gamma A_{2,\dagger} + \Lambda_F}{\mu_S}$ *and* $F_S^* = \dfrac{r\gamma A^*}{\mu_S}$, *it is interesting to observe that*

$$\mathcal{R}^2_{0,SIT_c} = \mathcal{R}_0^2 \begin{cases} \dfrac{r\gamma A_2 + \Lambda_F}{r\gamma A^*}, & \text{when} \quad \Lambda_M \in (0, \Lambda_M^{crit}), \\ \dfrac{r\gamma A_\dagger + \Lambda_F}{r\gamma A^*}, & \text{when} \quad \Lambda_M = \Lambda_M^{crit}, \\ \dfrac{\Lambda_F}{r\gamma A^*}, & \text{when} \quad \Lambda_M > \Lambda_M^{crit}, \end{cases} \tag{23}$$

where F_S^* *is defined in* $(7)_3$, *page 6. Thus clearly, if* Λ_F *is too large, i.e.* $\Lambda_F > r\gamma A^*$, *we always have* $\mathcal{R}^2_{0,SIT_c} > \mathcal{R}^2_0$, *such that the epidemiological risk increases,* $\mathcal{R}^2_{0,SIT_c}$ *might become larger than* 1. *Note also that, since* $A_{2,\dagger} < A^*$, *it follows that*

$$\mathcal{R}^2_{0,SIT_c} \leq \mathcal{R}^2_0$$

if and only if Λ_F *is sufficiently small, i.e.*

$$\Lambda_F < \begin{cases} r\gamma \left(A^* - A_2\right), & \text{when} \quad \Lambda_M \in (0, \Lambda_M^{crit}), \\ r\gamma \left(A^* - A_\dagger\right), & \text{when} \quad \Lambda_M = \Lambda_M^{crit}, \\ r\gamma A^*, & \text{when} \quad \Lambda_M > \Lambda_M^{crit}. \end{cases} \tag{24}$$

This result shows that for small releases, the constraint on the sterile females release rate is strong.

Remark 9.

- *Since* S_{S_2} *or* $S_{S_\dagger}$ *is an increasing function of* Λ_F, *it is straightforward to deduce that* $\mathcal{R}^2_{0,SIT_c}$ *increases with respect to* Λ_F.
- *Using* (14), *it is straightforward to show that* M_2 *is decreasing with respect to* Λ_M, *such that* A_2, F_{S_2} *are decreasing functions of* Λ_M *too. Thus, using* (22), *we deduce that* $\mathcal{R}^2_{0,SIT_c}$ *decreases with respect to* Λ_M.

5.2. Stability analysis of the disease-free equilibria

Using [30, Theorem 2] and a comparison argument, the stability properties of the biological disease-free equilibrium $DFE_{SIT_c} \in \{DFE_2, DFE_\dagger, TDFE\}$ is summarized as follows.

Theorem 4. *The following results hold true for system (18)-(19).*

1. *Assume* $\Lambda_M \in (0, \Lambda_M^{crit})$.
   (a) *If* $\mathcal{R}^2_{0,SIT_c} < 1$, *then* DFE_2, *defined in Proposition 3, is locally asymptotically stable.*
   (b) *If* $\mathcal{R}^2_{0,SIT_c} > 1$, *then* DFE_2 *is unstable.*
2. *Assume* $\Lambda_M = \Lambda_M^{crit}$).
   (a) *If* $\mathcal{R}^2_{0,SIT_c} < 1$, *then* $DFE_\dagger$, *defined in Proposition 3, is locally asymptotically stable.*
   (b) *If* $\mathcal{R}^2_{0,SIT_c} > 1$, *then* $DFE_\dagger$ *is unstable.*
3. *Assume* $\Lambda_M > \Lambda_M^{crit}$.
   (a) *If* $\mathcal{R}^2_{0,SIT_c} < 1$, *then* $TDFE$, *defined in Proposition 3, is globally asymptotically stable.*
   (b) *If* $\mathcal{R}^2_{0,SIT_c} > 1$, *then* $TDFE$ *is unstable.*

Thanks to Remark 8, page 14, when $\Lambda_M > \Lambda_M^{crit}$, $\mathcal{R}^2_{0,SIT_c} < 1 \Longleftrightarrow \Lambda_F \in [0, \Lambda_F^{crit})$ where

$$\Lambda_F^{crit} = \frac{r\gamma(\gamma + \mu_{A,1})(\mathcal{N} - 1)}{\mu_{A,2}\mathcal{R}_0^2}. \tag{25}$$

In fact, in the disease-free context, with $\Lambda_F \in [0, \Lambda_F^{crit})$, system (18)-(19) may exhibit a bistable dynamics. Indeed, using a similar approach as in the proof of Theorem 3 together with Theorem 2, it is straightforward to establish:

Theorem 5. *Consider system (18)-(19) with* $\Lambda_F \in [0, \Lambda_F^{crit})$.

1. *Assume that* $\Lambda_M \in (0, \Lambda_M^{crit})$. *If* $\mathcal{R}^2_{0,SIT_c} < 1$, *then equilibria* DFE_2 *and* $TDFE$ *are locally asymptotically stable (LAS). Moreover, the set*

   $$\{(S_h, I_h, R_h, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}_+^{11} : (A, M, F_S)^T < (A_1, M_1, F_{S_1})^T\}$$

   belongs to the basin of attraction of TDFE while the set

   $$\{(S_h, I_h, R_h, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}_+^{11} : (A, M, F_S)^T > (A_1, M_1, F_{S_1})^T\}$$

   belongs to the basin of attraction of DFE_2.
2. *Assume that* $\Lambda_M = \Lambda_M^{crit}$. *If* $\mathcal{R}^2_{0,SIT_c} < 1$, *then equilibria* $DFE_\dagger$ *and* $TDFE$ *are locally asymptotically stable (LAS). Moreover, the set*

   $$\{(S_h, I_h, R_h, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}_+^{11} : (A, M, F_S)^T < (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

   belongs to the basin of attraction of TDFE while the set

   $$\{(S_h, I_h, R_h, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}_+^{11} : (A, M, F_S)^T \geq (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

   belongs to the basin of attraction of $DFE_\dagger$.

However, the previous result does not give information on if and how SIT can impact $\mathcal{R}^2_{0,SIT_c}$.

5.3. Impact of insect releases on the SIT basic reproduction number

As stated in Remark 8, page 14, $\mathcal{R}^2_{0,SIT_c} \leq \mathcal{R}^2_0$ iff Λ_F is sufficiently small. However, this does not necessarily imply that there exists $\Lambda_M > 0$ such that $\mathcal{R}^2_{0,SIT_c} < 1$. Assume that $\Lambda_M \in (0, \Lambda_M^{crit})$. Using formula (14), we derive

$$A_2 = \frac{1}{2}A^* \left(1 - \frac{Q\Lambda_M}{\mu_T(\mathcal{N}-1)}\right)\left(1 + \sqrt{1 - \frac{4Q\Lambda_M\mu_T}{\left(\mu_T(\mathcal{N}-1) - Q\Lambda_M\right)^2}}\right) > 0. \tag{26}$$

Assuming $\Lambda_F \geq 0$ and using (26) and $(23)_{1,2}$, we deduce that

$$\mathcal{R}^2_{0,SIT_c} = \begin{cases} \dfrac{1}{2}\mathcal{R}_0^2\left(1 - \dfrac{Q\Lambda_M}{\mu_T(\mathcal{N}-1)}\right)\left(1 + \sqrt{1 - \dfrac{4Q\Lambda_M\mu_T}{\left(\mu_T(\mathcal{N}-1) - Q\Lambda_M\right)^2}}\right) + \dfrac{\Lambda_F}{\Lambda_F^{crit}}, & \text{when} \quad \Lambda_M \in (0, \Lambda_M^{crit}), \\ \dfrac{1}{2}\mathcal{R}_0^2\left(1 - \dfrac{Q\Lambda_M}{\mu_T(\mathcal{N}-1)}\right) + \mathcal{C} = \mathcal{R}_0^2\left(\dfrac{\sqrt{\mathcal{N}}-1}{\mathcal{N}-1}\right) + \dfrac{\Lambda_F}{\Lambda_F^{crit}}, & \text{when} \quad \Lambda_M = \Lambda_M^{crit}. \end{cases} \tag{27}$$

Clearly, if $\Lambda_F > \Lambda_F^{crit}$, this means that $\mathcal{R}^2_{0,SIT_c} > 1$, such that whatever the size of the releases of massive males, the epidemiological risk cannot be controlled.
We now assume $\Lambda_F < \Lambda_F^{crit}$, and we set

$$\mathcal{R}^2_{0,*} = \frac{\mathcal{N}-1}{\sqrt{\mathcal{N}} - 1 + \dfrac{\mu_{A,2}\Lambda_F}{r\gamma(\gamma + \mu_{A,1})}}. \tag{28}$$

We derive the following result

Theorem 6. *Assume $0 \leq \Lambda_F < \Lambda_F^{crit}$. Consider system (18)-(19) and set*

$$\Lambda^*_{M,\mathcal{R}_0^2,\mathcal{C}} = \frac{\mu_T(\mathcal{N}-1)}{Q}\left(1 - \frac{\mathcal{R}_0^4 + \left(1 - \dfrac{\Lambda_F}{\Lambda_F^{crit}}\right)^2(\mathcal{N}-1)}{\mathcal{R}_0^4 + \mathcal{R}_0^2\left(1 - \dfrac{\Lambda_F}{\Lambda_F^{crit}}\right)(\mathcal{N}-1)}\right). \tag{29}$$

1. *If $\mathcal{R}_0^2 \geq \mathcal{R}^2_{0,*}$, then for $\Lambda_M > \Lambda_M^{crit}$, the equilibrium TDFE is globally asymptotically stable.*
2. *If $1 < \mathcal{R}_0^2 < \mathcal{R}^2_{0,*}$, then the following results hold true:*
   - *When $\Lambda_M > \Lambda_M^{crit}$, the equilibrium TDFE is globally asymptotically stable.*
   - *When $\Lambda_M = \Lambda_M^{crit}$, then $\mathcal{R}^2_{0,SIT_c} < 1$, $DFE_\dagger$ and TDFE are locally asymptotically stable. The set*

     $$\{(S, I, R, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}^{11}_+ : (A, M, F_S)^T < (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

     belongs to the basin of attraction of TDFE while the set

     $$\{(S, I, R, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}^{11}_+ : (A, M, F_S)^T \geq (A_\dagger, M_\dagger, F_{S_\dagger})^T\}$$

     belongs to the basin of attraction of $DFE_\dagger$.

- *when* $\Lambda_M > \Lambda^*_{M,\mathcal{R}_0^2,\mathcal{C}}$, *then* $\mathcal{R}^2_{0,SIT_c} < 1$, *and the equilibria* DFE_2 *and* $TDFE$ *are locally asymptotically stable. Moreover, the set*

$$\{(S, I, R, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}^{11}_+ : (A, M, F_S)^T < (A_1, M_1, F_{S_1})^T\}$$

belongs to the basin of attraction of TDFE while the set

$$\{(S, I, R, A, M, F_S, F_E, F_I, S_S, S_E, S_I)^T \in \mathbb{R}^{11}_+ : (A, M, F_S)^T > (A_1, M_1, F_{S_1})^T\}$$

belongs to the basin of attraction of DFE_2.

Proof. We deduce from Theorem 4 that when $\Lambda_M > \Lambda_M^{crit}$ and $0 \leq \Lambda_F < \Lambda_F^{crit}$, then TDFE is GAS. When $\Lambda_M \in (0, \Lambda_M^{crit}]$ and $\mathcal{R}_0^2 > \mathcal{R}^2_{0,*}$, then $\mathcal{R}^2_{0,SIT_c} > 1$, such that SIT control is ineffective: only massive releases can be used, i.e. $\Lambda_M > \Lambda_M^{crit}$. Clearly, the larger Λ_F, the smaller $\dfrac{\mathcal{N}-1}{\sqrt{\mathcal{N}}-1+\dfrac{\mu_{A,2}\Lambda_F}{r\gamma(\gamma+\mu_{A,1})}}$. In order to be able to use "small" releases strategy, we have to define upper bounds for Λ_F, for a given Λ_M and vice-versa.

We set $\mathcal{C} = \dfrac{\Lambda_F}{\Lambda_F^{crit}}$ and $M_T^* = \dfrac{\Lambda_M}{\mu_T}$. Since $\mathcal{C} < 1$ holds true, then, for a given Λ_F, using $(27)_1$, we have to find $x = QM_T^*$ such that

$$1 - \frac{4x}{(\mathcal{N}-1-x)^2} < \left(\frac{2(1-\mathcal{C})(\mathcal{N}-1)}{\mathcal{R}_0^2\,(\mathcal{N}-1-x)} - 1\right)^2. \tag{30}$$

Setting $y = \mathcal{N} - 1 - x$, this leads, after some manipulation, to

$$1 - \frac{4(\mathcal{N}-1-y)}{y^2} < \left(\frac{2(1-\mathcal{C})(\mathcal{N}-1)}{\mathcal{R}_0^2 y} - 1\right)^2,$$

$$\frac{y^2 - 4(\mathcal{N}-1-y)}{y^2} < \frac{1}{\mathcal{R}_0^4 y^2}\left(2(1-\mathcal{C})(\mathcal{N}-1) - \mathcal{R}_0^2 y\right)^2,$$

$$\mathcal{R}_0^4 y^2 - 4\mathcal{R}_0^4(\mathcal{N}-1-y) < 4(1-\mathcal{C})^2(\mathcal{N}-1)^2 + \mathcal{R}_0^4 y^2 - 4\mathcal{R}_0^2 y(1-\mathcal{C})(\mathcal{N}-1),$$

$$\left(\mathcal{R}_0^4 + \mathcal{R}_0^2(1-\mathcal{C})(\mathcal{N}-1)\right) y < (1-\mathcal{C})^2(\mathcal{N}-1)^2 + \mathcal{R}_0^4(\mathcal{N}-1),$$

$$y < \frac{(1-\mathcal{C})^2(\mathcal{N}-1)^2 + \mathcal{R}_0^4(\mathcal{N}-1)}{\mathcal{R}_0^4 + \mathcal{R}_0^2(1-\mathcal{C})(\mathcal{N}-1)},$$

$$\mathcal{N}-1-x < \frac{(1-\mathcal{C})^2(\mathcal{N}-1)^2 + \mathcal{R}_0^4(\mathcal{N}-1)}{\mathcal{R}_0^4 + \mathcal{R}_0^2(1-\mathcal{C})(\mathcal{N}-1)},$$

$$\mathcal{N}-1-\frac{(1-\mathcal{C})^2(\mathcal{N}-1)^2 + \mathcal{R}_0^4(\mathcal{N}-1)}{\mathcal{R}_0^4 + \mathcal{R}_0^2(1-\mathcal{C})(\mathcal{N}-1)} < x,$$

that is,

$$\frac{\mu_T(\mathcal{N}-1)}{Q}\left(1 - \frac{(1-\mathcal{C})^2(\mathcal{N}-1) + \mathcal{R}_0^4}{\mathcal{R}_0^4 + \mathcal{R}_0^2(1-\mathcal{C})(\mathcal{N}-1)}\right) < \Lambda_M,$$

and we deduce (29). Then, the results follow from Theorem 5, page 15. □

Clearly the constraint on the releases size given by (29) can be strong, i.e. close to Λ_M^{crit}, such that it seems to be preferable to use massive releases, i.e. $\Lambda_M > \Lambda_M^{crit}$.

In that case, the strategy developed in [2, 5], using massive and then small releases can be adequate to reduce the epidemiological risk and maintain this risk at a lower level.

Thus, in terms of vector control: when $\mathcal{R}_0^2 \leq 1$, vector control is not necessary; when $\mathcal{R}_0^2 > 1$ and $0 \leq \Lambda_F < \Lambda_F^{crit}$, then two cases should be considered:

- when $\mathcal{R}_0^2 \geq \mathcal{R}_{0,*}^2$, then massive releases of sterile insect, i.e. $\Lambda_M > \Lambda_M^{crit}$, should be advocated.
- When $\mathcal{R}_0^2 < \mathcal{R}_{0,*}^2$, then small, but large enough ($\Lambda_{M,\mathcal{R}_0^2,\mathcal{C}}^* < \Lambda_M \leq \Lambda_M^{crit}$), releases of sterile insects could be useful to control the disease. However, since $\Lambda_{M,\mathcal{R}_0^2,\mathcal{C}}^*$ is close to Λ_M^{crit}, from a practical point of view, it is preferable to consider massive releases of sterile insects too.

We summarize all qualitative results of system (18)-(19) related to the disease free equilibria in Table 2, page18.

$\mathcal{N}$	$\mathcal{R}_0^2$	Λ_F	$\mathcal{R}_0^2$	Λ_M	Observations
≤ 1					$TDFE$ is GAS
> 1	≤ 1				Releases of sterile insects are useless because the DFE is already GAS
	> 1	$\geq \Lambda_F^{crit}$		$> \Lambda_M^{crit}$	Only massive releases could be efficient such that $TDFE$ is GAS
		$< \Lambda_F^{crit}$	$\geq \mathcal{R}_{0,*}^2$	$> \Lambda_M^{crit}$	$TDFE$ is GAS
			$< \mathcal{R}_{0,*}^2$	$> \Lambda_M^{crit}$	$TDFE$ is GAS
				$= \Lambda_M^{crit}$	$\mathcal{R}_{0,SIT_c}^2 < 1$, $TDFE$ and $DFE_\dagger$ are both stable
				$> \Lambda_{M,\mathcal{R}_0^2,\mathcal{C}}^*$	$\mathcal{R}_{0,SIT_c}^2 < 1$, $TDFE$ and DFE_2 are both stable

Table 2: Summary table of the qualitative analysis of system (18)-(19)

Remark 10. *According to Table 2, when $\mathcal{R}_0^2 < \mathcal{R}_{0,*}^2$, we recover bifurcation diagram 2, page 10: it suffices to replace $\boldsymbol{TE}$ by $TDFE$ and $\boldsymbol{E}_{2,\dagger}$ by $DFE_{2,\dagger}$.*

5.4. About the effective reproduction number

SIT control is a long term strategy: it means that to lower the epidemiological risk before entering the risky season (when DENV starts circulating), SIT has to be started far before. However, if the basic reproduction number is estimated (even roughly), several options can be used. Indeed, before the rainy season starts, and following (17), page 11, to get $\mathcal{R}_0^2 < 1$, it suffices to reduce the size of the female population under the following threshold

$$F_{epi}^* = \frac{(\nu_m + \mu_S)}{\nu_m} \frac{\mu_I (\eta_h + \mu_h)}{B^2 \beta_{mh} \beta_{hm}} N_h. \tag{31}$$

If SIT is used before the risky season, then the strategy of massive releases followed by small releases can be used: first, massive releases to reduce the initial size below F_{epi}^*, for instance,

followed by small releases to maintain it small [5], that is $F_S(t) + S_S(t) < F^*_{epi}$, for all $t > t_{DENV}$, where t_{DENV} is the time where infected individuals (by Dengue) are introduced, i.e. when the outbreak starts. However, we will highlight the fact that the starting time, t_S, of the SIT control is important relative to t_{DENV}, even if $\mathcal{R}^2_{0,SIT_c} < 1$. That is why, it is important to consider the effective reproduction number, $\mathcal{R}_{eff}(t)$,that is defined as follow

$$\mathcal{R}_{eff}(t) = \frac{\nu_m}{\nu_m + \mu_S} \frac{B^2 \beta_{mh} \beta_{hm}}{\mu_I (\eta_h + \mu_h)} \frac{F_S(t) + S_S(t)}{N_h}. \tag{32}$$

In particular, we will estimate $\mathcal{R}_{eff}$ at time t_{DENV}. Clearly, if $\mathcal{R}_{eff}(t_{DENV}) < 1$ and $\mathcal{R}^2_{0,SIT_c} < 1$, then no epidemics will occur. In contrary, even if $\mathcal{R}^2_{0,SIT_c} < 1$ but $\mathcal{R}_{eff}(t_{DENV}) > 1$ then an outbreak will occur.

Last, combining SIT with Mechanical control will also improve the previous results in terms of size of the releases or duration of the releases in order to have $\mathcal{R}_{eff}(t_{DENV}) < 0.5$, to avoid any outbreak. This will be illustrated later.

6. The SIT periodic releases case

Assuming that the releases are periodic, for example with weekly period. Then epidemiological model is defined by the system (18)-(33)-(34):

$$\begin{cases} \frac{dA}{dt} &= \phi(F_S + F_E + F_I) - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \frac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \frac{dF_S}{dt} &= \frac{M}{M+M_T} r\gamma A - B\beta_{hm}\frac{I_h}{N_h}F_S - \mu_S F_S, \\ \frac{dF_E}{dt} &= B\beta_{hm}\frac{I_h}{N_h}F_S - (\nu_m + \mu_S)F_E, \\ \frac{dF_I}{dt} &= \nu_m F_E - \mu_I F_I, \\ \frac{dS_S}{dt} &= \frac{M_T}{M+M_T} r\gamma A - B\beta_{hm}\frac{I_h}{N_h}S_S - \mu_S S_S, \\ \frac{dS_E}{dt} &= B\beta_{hm}\frac{I_h}{N_h}S_S - (\nu_m + \mu_S)S_E, \\ \frac{dS_I}{dt} &= \nu_m S_E - \mu_I S_I, \\ \frac{dM_T}{dt} &= -\mu_T M_T, \end{cases} \tag{33}$$

$$\begin{cases} S_h(n\tau_+) = S_h(n\tau), \\ I_h(n\tau_+) = I_h(n\tau), \\ R_h(n\tau_+) = R_h(n\tau), \\ A(n\tau_+) = A(n\tau), \\ M(n\tau_+) = M(n\tau), \\ F_S(n\tau_+) = F_S(n\tau), \\ F_E(n\tau_+) = F_E(n\tau), \\ F_I(n\tau_+) = F_I(n\tau), \\ S_S(n\tau_+) = S_S(n\tau) + \tau\Lambda_F, \\ S_E(n\tau_+) = S_E(n\tau), \\ S_I(n\tau_+) = S_I(n\tau), \\ M_T(n\tau_+) = M_T(n\tau) + \tau\Lambda_M \end{cases} \tag{34}$$

where τ is the period of the releases.

Remark 11. *Practically, we will consider a total amount of sterile insects released at time $n\tau$, M_{total}. Among these releases, a fraction ε of sterile females will be released, such that $\varepsilon M_{total} = \tau\Lambda_F$, and $(1-\varepsilon)M_{total} = \tau\Lambda_M$.*

When $\varepsilon = 0$, we can use the approach already used in [14] to study the sterile male impulsive model. When $\varepsilon > 0$, then we have to study the previous systems.

6.1. Existence and stability of the trivial periodic disease-free solution ($TDFS_{per}$)

Let us consider the following system (35)-(36) that models the periodic release of sterile insects (both males and females):

$$\begin{cases} \dfrac{dS_S}{dt} = -\mu_S S_S, \quad \text{for} \quad t \neq n\tau, \\ \dfrac{dM_T}{dt} = -\mu_T M_T, \end{cases} \tag{35}$$

$$\begin{cases} S_S(n\tau_+) = S_S(n\tau) + \tau\Lambda_F, \\ M_T(n\tau_+) = M_T(n\tau) + \tau\Lambda_M. \end{cases} \tag{36}$$

Hence, as $t \to +\infty$, M_T and S_S converges toward the periodic solution

$$\begin{cases} M_T^{per}(t) = \dfrac{\tau\Lambda_M}{1-e^{-\mu_T\tau}} e^{-\mu_T(t-\lfloor t/\tau \rfloor \tau)}, \\ \\ S_S^{per}(t) = \dfrac{\tau\Lambda_F}{1-e^{-\mu_S\tau}} e^{-\mu_S(t-\lfloor t/\tau \rfloor \tau)}. \end{cases} \tag{37}$$

Moreover, we have that

$$\int_0^\tau S_S^{per}(x)dx = \frac{\tau\Lambda_F}{\mu_S}. \tag{38}$$

Taking into account (37), we deduce that solutions of system (18)-(33)-(34) converge in the sense of $L^\infty(0,+\infty)$ norm, to solutions of system (18)-(39)-(40). Precisely, only $M_T(t)$ is substituted by $M_T^{per}(t)$ in (33) to obtain (39):

$$
\begin{cases}
\dfrac{dA}{dt} &= \phi(F_S+F_E+F_I)-(\gamma+\mu_{A,1}+\mu_{A,2}A)A,\\
\dfrac{dM}{dt} &= (1-r)\gamma A-\mu_M M,\\
\dfrac{dF_S}{dt} &= \dfrac{M}{M+M_T^{per}}r\gamma A-B\beta_{hm}\dfrac{I_h}{N_h}F_S-\mu_S F_S,\\
\dfrac{dF_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}F_S-(\nu_m+\mu_S)F_E,\\
\dfrac{dF_I}{dt} &= \nu_m F_E-\mu_I F_I,\\
\dfrac{dS_S}{dt} &= \dfrac{M_T^{per}}{M+M_T^{per}}r\gamma A-B\beta_{hm}\dfrac{I_h}{N_h}S_S-\mu_S S_S,\\
\dfrac{dS_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}S_S-(\nu_m+\mu_S)S_E,\\
\dfrac{dS_I}{dt} &= \nu_m S_E-\mu_I S_I,
\end{cases}
\tag{39}
$$

$$
\begin{cases}
S_h(n\tau_+)=S_h(n\tau),\\
I_h(n\tau_+)=I_h(n\tau),\\
R_h(n\tau_+)=R_h(n\tau),\\
A(n\tau_+)=A(n\tau),\\
M(n\tau_+)=M(n\tau),\\
F_S(n\tau_+)=F_S(n\tau),\\
F_E(n\tau_+)=F_E(n\tau),\\
F_I(n\tau_+)=F_I(n\tau),\\
S_S(n\tau_+)=S_S(n\tau)+\tau\Lambda_F,\\
S_E(n\tau_+)=S_E(n\tau),\\
S_I(n\tau_+)=S_I(n\tau).
\end{cases}
\tag{40}
$$

It is straightforward to obtain that system (18)-(39)-(40) admits as trivial periodic disease-free solution (TDFS$_{per}$):

$$
TDFS_{per}=(N_h,\mathbf{0}_{\mathbb{R}^7},S_S^{per}(t),0,0)^T
$$

where S_S^{per} is defined in (37). Finally, we set

$$
\mathcal{T}_{0,pulse}=\frac{\nu_m}{\nu_m+\mu_S}\frac{B^2\beta_{hm}\beta_{mh}}{(\eta_h+\mu_h)\mu_I N_h}\frac{1}{\tau}\int_0^\tau S_S^{per}(x)dx=\frac{\nu_m}{\nu_m+\mu_S}\frac{B^2\beta_{hm}\beta_{mh}}{(\eta_h+\mu_h)\mu_I N_h}\frac{\Lambda_F}{\mu_S},
\tag{41}
$$

$$
\begin{cases}
\underline{\Lambda}_M=\mu_T\min\limits_{t\in[0,\tau]}M_T^{per}(t)=\dfrac{\mu_T\tau\Lambda_M}{1-e^{-\mu_T\tau}}e^{-\mu_T\tau},\\
\overline{\Lambda}_M=\mu_T\max\limits_{t\in[0,\tau]}M_T^{per}(t)=\dfrac{\mu_T\tau\Lambda_M}{1-e^{-\mu_T\tau}},
\end{cases}
\tag{42}
$$

and, using Λ_M^{crit} defined in (12), page 8, we also set

$$
M_{T,per}^{crit}=\frac{\Lambda_M^{crit}\left(e^{\mu_T\tau}-1\right)}{\mu_T}.
\tag{43}
$$

Note that following (22), when $\Lambda_M > \Lambda_M^{crit}$, then $\mathcal{R}^2_{0,SIT_c} = \mathcal{T}_{0,pulse}$ and both thresholds shape the stability of the trivial disease-free state. Moreover,

$$\mathcal{T}_{0,pulse} < 1 \quad \text{if and only if} \quad \Lambda_F < \Lambda_F^{crit}, \tag{44}$$

where Λ_F^{crit} is defined in (25). Indeed, from (41), we deduce that

$$\mathcal{T}_{0,pulse} = \frac{\mathcal{R}_0^2 \mu_{A,2}}{r\gamma(\gamma + \mu_{A,1})(\mathcal{N} - 1)} \Lambda_F. \tag{45}$$

Hence, (44) is straightforwardly deduced. We can prove the following global attractivity result.

Theorem 7. *Let $\varepsilon \geq 0$ and $M_{total} > 0$ be given. Assume that $\Lambda_F < \Lambda_F^{crit}$.*

(i) If

$$\tau \Lambda_M > M_{T,per}^{crit}, \tag{46}$$

then solutions of system (18)-(39)-(40) are such that

$$\lim_{t \to +\infty} (S(t), I(t), R(t), A(t), M(t), F_S(t), F_E(t), F_I(t), S_S(t), S_E(t), S_I(t))^T = TDFS_{per}.$$

(ii) If

$$\tau \Lambda_M = M_{T,per}^{crit}, \tag{47}$$

solutions of system (18)-(39)-(40) are such that

$$\lim_{t \to +\infty} (S(t), I(t), R(t), A(t), M(t), F_S(t), F_E(t), F_I(t), S_S(t), S_E(t), S_I(t))^T = TDFS_{per}$$

whenever

$$(A, M, F_S + F_E + F_I)^T(0) \in [\boldsymbol{0}_{\mathbb{R}^3}, \boldsymbol{E}_\dagger(\underline{\Lambda}_M)).$$

(iii) If

$$0 < \tau \Lambda_M < M_{T,per}^{crit}, \tag{48}$$

then solutions of system (18)-(39)-(40) are such that

$$\lim_{t \to +\infty} (S(t), I(t), R(t), A(t), M(t), F_S(t), F_E(t), F_I(t), S_S(t), S_E(t), S_I(t))^T = TDFS_{per}$$

whenever

$$(A, M, F_S + F_E + F_I)^T(0) \in [\boldsymbol{0}_{\mathbb{R}^3}, \boldsymbol{E}_1(\underline{\Lambda}_M)).$$

Proof. See Appendix D, page 50. □

Like in Theorem 6, we can deduce from Theorem 7 that, in the case of periodic releases, the strategy using large and small releases is useful to decay an established mosquito population and thus to lower the epidemiological risk. If the mosquito population is small or not established, then (appropriate) small releases of sterile insects are sufficient to reach elimination too.

6.2. Existence of a periodic disease-free solution (DFS_{per})

Taking into account (37), the disease-free sub-system derived from system (18)-(33)-(34) is given by system (49)-(50):

$$\begin{cases} \dfrac{dS_h}{dt} &= \mu_h(N_h - S_h), \\ \dfrac{dA}{dt} &= \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} &= (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} &= \dfrac{M}{M + M_T^{per}} r\gamma A - \mu_S F_S, \\ \dfrac{dS_S}{dt} &= \dfrac{M_T^{per}}{M + M_T^{per}} r\gamma A - \mu_S S_S, \end{cases} \tag{49}$$

$$\begin{cases} S_h(n\tau_+) = S_h(n\tau), \\ A(n\tau_+) = A(n\tau), \\ M(n\tau_+) = M(n\tau), \\ F_S(n\tau_+) = F_S(n\tau), \\ S_S(n\tau_+) = S_S(n\tau) + \tau\Lambda_F. \end{cases} \tag{50}$$

The dynamics of state variables S_h and S_S, i.e. equations $(49)_1$–$(50)_1$ and $(49)_5$–$(50)_5$, are uncoupled. Hence, we will consider in the sequel the sub-system (51):

$$\begin{cases} \dfrac{dA}{dt} = \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} = (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} = \dfrac{M}{M + M_T^{per}(t)} r\gamma A - \mu_S F_S. \end{cases} \tag{51}$$

Substituting $M_T^{per}(t)$ by $\dfrac{\overline{\Lambda}_M}{\mu_T}$ and by $\dfrac{\underline{\Lambda}_M}{\mu_T}$ in (51) leads to the lower system (52) and the upper system (53), with

$$\begin{cases} \dfrac{dA}{dt} = \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} = (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} = \dfrac{M}{M + \dfrac{\overline{\Lambda}_M}{\mu_T}} r\gamma A - \mu_S F_S \end{cases} \tag{52}$$

and

$$\begin{cases} \dfrac{dA}{dt} = \phi F_S - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} = (1-r)\gamma A - \mu_M M, \\ \dfrac{dF_S}{dt} = \dfrac{M}{M + \dfrac{\underline{\Lambda}_M}{\mu_T}} r\gamma A - \mu_S F_S. \end{cases} \tag{53}$$

Hence, one has, see e.g. [5]:

(i) When $\mathcal{N} > 1$ and $\overline{\Lambda}_M \in (0, \Lambda_M^{crit}]$, system (52) admits one or two positive equilibria $\overline{E}_{1,3D} \leq \overline{E}_{2,3D}$. In addition, if the initial data of (52) is greater or equal to $\overline{E}_{1,3D}$, then the corresponding solution is also greater or equal to $\overline{E}_{1,3D}$. Recall that M_{T_1} is given by (12). Similarly, since $0 < \underline{M}_T < \overline{M}_T \leq M_{T_1}$, we deduce that system (53) admits one or two positive equilibria $\underline{E}_{1,3D} \leq \underline{E}_{2,3D}$. Finally, the set

$$\{(A, M, F_S)^T \in \mathbb{R}^3_+ : (A, M, F_S)^T < \underline{E}_{1,3D}\}$$

belongs to the basin of attraction of $\mathbf{0}_{\mathbb{R}^3} = (0, 0, 0)^T$ for system (53), hence by comparison, its belongs to the basin of attraction of $\mathbf{0}_{\mathbb{R}^3}$ for system (51).

(ii) When $\mathcal{N} > 1$ and $\underline{\Lambda}_M > \Lambda_M^{crit}$, then the elimination equilibrium $\mathbf{0}_{\mathbb{R}^3}$ is globally asymptotically stable for system (53).

(ii) When $\mathcal{N} \leq 1$, then the elimination equilibrium $\mathbf{0}_{\mathbb{R}^3}$ is globally asymptotically stable for system (53).

Taking into account Lemma 1 together with the previous discussion, leads to Proposition 4.

Proposition 4. *If $\mathcal{N} > 1$ and $\overline{\Lambda}_M \in (0, \Lambda_M^{crit}]$, the set*

$$\Omega = \{(A, M, F_S)^T \in \mathbb{R}^3_+ : \overline{E}_{1,3D} \leq (A, M, F_S)^T \leq E^*\}$$

is positively invariant by system (51) where E^* is the positive wild equilibrium given by (7).

To establish the existence of a least one positive and periodic solution for system (51), we will use the Brouwer fixed point theorem together with comparison arguments. The following result is valid.

Theorem 8. *Assume that $\mathcal{N} > 1$ and $\overline{\Lambda}_M \in (0, \Lambda_M^{crit}]$. Then, for*

$$\overline{E}_{1,3D} \leq (A(0), M(0), F_S(0))^T \leq E^*,$$

system (18)-(39)-(40) has at least one positive τ-periodic disease-free solution $DFS_{per} = (S_h(t), 0_{\mathbb{R}^2}, A(t), M(t), F_S(t), 0_{\mathbb{R}^2}, S_S(t), 0_{\mathbb{R}^2})^T$ with

$$\overline{E}_{1,3D} \leq (A(t), M(t), F_S(t))^T \leq E^*,$$

$S_h(t) = N_h$ *and*

$$S_S(t) = \left(\frac{\tau \Lambda_F + e^{-\mu_S \tau} \displaystyle\int_{\lfloor t/\tau \rfloor \tau}^{(\lfloor t/\tau \rfloor \tau + 1)\tau} v(x) e^{\mu_S x} dx}{1 - e^{-\mu_S \tau}} + \int_{\lfloor t/\tau \rfloor \tau}^{t} v(x) e^{\mu_S x} dx \right) e^{-\mu_S (t - \lfloor t/\tau \rfloor \tau)} \tag{54}$$

where

$$v(t) = \frac{M_T^{per}(t)}{M(t) + M_T^{per}(t)} r \gamma A(t).$$

Proof. See Appendix E. □

6.3. Stability property of a periodic disease-free solution (DFS_{per})

Now, we are in position to define the basic reproduction number, $\mathcal{R}^{per}_{0,SIT}$, of system (18)-(39)-(40). Following the approach given in [32, 33], we set $X = (I_h, F_E, F_I, S_E, S_I, A, M, F_S, S_S, S_h, R_h)^T$ and the non-trivial and τ-periodic disease-free solution is

$$X^{per} = (0_{\mathbb{R}^5}, A^{per}(t), M^{per}(t), F_S^{per}(t), S_S^{per}(t), N_h, 0)^T.$$

For $i \in \{1,2,3,4,5,6,7,8,9,10,11\}$ and $t \neq n\tau$, let $\mathcal{F}_i(t,X)$ be the input rate of newly infected individuals in the ith compartment; $\mathcal{V}_i^+(t,X)$ is the input rate of individuals by others means, $\mathcal{V}_i^-(t,X)$ is the rate of transfer of individuals out of compartment i and $\mathcal{V}_i(t,X) = \mathcal{V}_i^-(t,X) - \mathcal{V}_i^+(t,X)$. That is,

$$\mathcal{F}(t,X) = \begin{pmatrix} B\beta_{mh}\dfrac{F_I+S_I}{N_h}S_h \\ B\beta_{hm}\dfrac{I_h}{N_h}F_S \\ 0 \\ B\beta_{hm}\dfrac{I_h}{N_h}S_S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}(t,X) = \begin{pmatrix} (\eta_h+\mu_h)I_h \\ (\nu_m+\mu_S)F_E \\ -\nu_m F_E + \mu_I F_I \\ (\nu_m+\mu_S)S_E \\ -\nu_m S_E + \mu_I S_I \\ -\phi(F_S+F_E+F_I) + (\gamma+\mu_{A,1}+\mu_{A,2}A)A \\ -(1-r)\gamma A + \mu_M M \\ -\dfrac{M}{M+M_T^{per}(t)}r\gamma A + B\beta_{hm}\dfrac{I_h}{N_h}F_S + \mu_S F_S \\ -\dfrac{M^{per}(t)}{M+M_T^{per}(t)}r\gamma A + B\beta_{hm}\dfrac{I_h}{N_h}S_S + \mu_S S_S \\ -\mu_h N_h + B\beta_{mh}\dfrac{F_I+S_I}{N_h}S_h + \mu_h S_h \\ -\eta_h I_h + \mu_h R_h \end{pmatrix}$$

such that system (18)-(39) can be written as follows

$$\frac{dX}{dt} = \mathcal{F}(t,X) - \mathcal{V}(t,X). \tag{55}$$

It is straightforward to prove that $\mathcal{F}(t,X)$, $\mathcal{V}(t,X)$ and system (40) satisfy assumptions $(H1)-(H6)$ of [33].

The derivatives of $\mathcal{V}$ and $\mathcal{F}$, at X^{per}, can be parted as follows

$$D\mathcal{F}(x,t) = \begin{pmatrix} F(t) & 0 \\ 0 & 0 \end{pmatrix}, \qquad D\mathcal{V}(x,t) = \begin{pmatrix} V(t) & 0 \\ \star & -M(t) \end{pmatrix},$$

where

$$F = \begin{pmatrix} 0 & 0 & B\beta_{mh} \\ B\beta_{hm}\dfrac{F_S^{per}(t)}{N_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \eta_h+\mu_h & 0 & 0 \\ 0 & \nu_m+\mu_S & 0 \\ 0 & -\nu_m & \mu_I \end{pmatrix}$$

and

$$\mathcal{M}(t) = \begin{pmatrix} -(\mu_S+\nu_m) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \nu_m & -\mu_I & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{66} & 0 & \phi & 0 & 0 & 0 \\ 0 & 0 & (1-r)\gamma & -\mu_M & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{86} & a_{87} & -\mu_S & 0 & 0 & 0 \\ 0 & 0 & a_{96} & a_{97} & 0 & -\mu_S & 0 & 0 \\ 0 & -B\beta_{mh} & 0 & 0 & 0 & 0 & -\mu_h & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_h \end{pmatrix}$$

with

$$\begin{aligned} a_{66} &= -(\gamma + \mu_{A,1} + 2\mu_{A,2}A^{per}(t)), & a_{87} &= \frac{r\gamma A^{per}(t)M_T^{per}(t)}{(M(t)+M_T^{per}(t))^2}, \\ a_{86} &= \frac{r\gamma M(t)}{M(t)+M_T^{per}(t)}, & a_{97} &= -\frac{r\gamma A^{per}(t)M_T^{per}(t)}{(M^{per}(t)+M_T^{per}(t))^2}, \\ a_{96} &= \frac{r\gamma M_T^{per}(t)}{M^{per}t)+M_T^{per}(t)}. \end{aligned}$$

Let $\Phi_{\mathcal{M}}(\tau)$ be the monodromy matrix related to the linear τ-periodic system

$$\begin{cases} \dfrac{dZ(t)}{dt} = \mathcal{M}(t)Z(t), & t \neq n\tau, \\ Z(n\tau+) = Z(n\tau), & t = n\tau. \end{cases}$$

We assume that the periodic equilibrium $X^{per} = (0, 0, A^{per}(t), M^{per}(t), F_S^{per}(t), S_S^{per}(t), N_h, 0)^T$ is locally asymptotically stable, that is

$$(H7) \qquad \rho(\Phi_{\mathcal{M}}(\tau)) < 1,$$

where $\rho(\cdot)$ is the spectral radius operator. Let us consider the sub-matrix $\mathcal{E}$ of matrix $\mathcal{M}$ defined by

$$\mathcal{E} = \begin{pmatrix} a_{66} & 0 & \phi \\ (1-r)\gamma & -\mu_M & 0 \\ a_{86} & a_{87} & -\mu_S \end{pmatrix}$$

and the monodromy matrix, $\Phi_{\mathcal{E}}(\tau)$, related to the linear τ-periodic system

$$\begin{cases} \dfrac{dQ(t)}{dt} = \mathcal{E}(t)Q(t), & t \neq n\tau, \\ Q(n\tau+) = Q(n\tau), & t = n\tau. \end{cases}$$

It is straightforward to deduce that

$$\rho(\Phi_{\mathcal{M}}(\tau)) < 1 \iff \rho(\Phi_{\mathcal{E}}(\tau)) < 1.$$

Hence, we set

$$(H7)' \qquad \rho(\Phi_{\mathcal{E}}(\tau)) < 1.$$

Unfortunately, since we do not have an explicit expression of X_{per}, we will only be able to verify assumption $(H7)$ or $(H7)'$ numerically. Similarly, the monodromy matrix related to the linear τ-periodic system

$$\begin{cases} \dfrac{dY(t)}{dt} = -VY(t), & t \neq n\tau, \\ Y(n\tau+) = Y(n\tau), & t = n\tau, \end{cases}$$

is

$$\Phi_{-V}(\tau) = \begin{pmatrix} e^{-(\eta_h+\mu_h)\tau} & 0 & 0 \\ 0 & e^{-(\nu_m+\mu_S)\tau} & 0 \\ 0 & \star & e^{-\mu_I\tau} \end{pmatrix}$$

where $\star$ is a real value. Therefore, it is straightforward to deduce that

$$(H8) \qquad \rho(\Phi_{-V}(\tau)) < 1$$

holds true.

Following [33], one can define the next infection operator $\mathcal{L}$ which is positive, continuous and compact. We also define the basic reproduction number (see for instance [33])

$$\mathcal{R}^{per}_{0,SIT} = \rho(\mathcal{L}),$$

the spectral radius of $\mathcal{L}$. The next result (see Lemma 3) shows that $\rho(\Phi_{F-V}(\tau)) - 1$ has the same sign as $\mathcal{R}^{per}_{0,SIT} - 1$.

Lemma 3. *Let X_{per} be a non-trivial periodic disease-free solution of system (18)-(39)-(40). Assume that $(H7)'$ holds true. Then, one has:*

1. $\mathcal{R}^{per}_{0,SIT} = 1$ *if and only if* $\rho(\Phi_{F-V}(\tau)) = 1$.
2. $\mathcal{R}^{per}_{0,SIT} > 1$ *if and only if* $\rho(\Phi_{F-V}(\tau)) > 1$.
3. $\mathcal{R}^{per}_{0,SIT} < 1$ *if and only if* $\rho(\Phi_{F-V}(\tau)) < 1$.

Hence, X_{per} is locally asymptotically stable if $\mathcal{R}^{per}_{0,SIT} < 1$ and unstable if $\mathcal{R}^{per}_{0,SIT} > 1$.

Proof. Assumptions (H1), (H2), (H3), (H4), (H5), (H6) and (H8) are verified. Assuming that $(H7)'$ is verified implies that (H7) holds true. Hence, conclusions of the Lemma follow from [33, Theorem 2.2]. □

As already pointed out in [14, 32], it is not possible to obtain explicitly an analytic expression for $\rho(\Phi_{F-V}(\tau))$ or $\mathcal{R}^{per}_{0,SIT}$. Since F and V are not both upper or lower triangular, according to Theorem 4.1 [21], we cannot use a time-average method to compute $\mathcal{R}^{per}_{0,SIT}$. It will be computed numerically; see for instance [21] for a procedure for the linear operator method.

Also, when $\mathcal{R}_0 > 1$, since $\mathcal{R}^{per}_{0,SIT}$ can only be estimated numerically, we cannot derive an analytical expression for the threshold $\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$ in order to ensure that $\mathcal{R}^{per}_{0,SIT} < 1$, assuming, of course, that $\Lambda_F < \Lambda_F^{crit}$. This will be evaluated numerically, using the linear operator method coupled with the bisection method. See some estimates in Tables 9, 10, 11, with and without mechanical control.

7. Numerical simulations

All numerical simulations are done using a standard finite difference method, the ode23tb solver of Matlab [20] which solves system of stiff ODEs using a trapezoidal rule and second order backward differentiation scheme (TR-BDF2) [7, 18]. Results are obtained in a couple of seconds.

We consider *Aedes albopictus* mosquito parameters used in previous publications [4, 5, 11, 12, 13]. They are summarized in Table 3.

Symbol	ϕ	$\mu_{A,1}$	$\mu_{A,2}$	r	μ_S	μ_M	μ_T	γ
Value	10	0.05	to be estimated	0.50	1/10	1/7	1/7	0.08

Table 3: *Aedes spp* entomological parameter values.

According to Table 3, the basic offspring number is $\mathcal{N} \approx 30.76$, and the critical daily release rate for sterile males is $\Lambda_M^{crit} = 1613$, without mechanical control. Above this value, the wild mosquito population will decay to $\mathbf{0}$ more or less fast depending on the size of the releases.

Following [14], we estimate $\mu_{A,2}$ thanks to the carrying capacity (of breeding sites), K. Assuming that $K = 3 \times N_h$, the aquatic stage at equilibrium is $A^* = \left(1 - \frac{1}{\mathcal{N}}\right) K$, such that to get the same equilibrium with our model leads to

$$\mu_{A,2} = \frac{\gamma + \mu_{A,1}}{K} \mathcal{N}.$$

Thus according to the value taken for N_h in Table 5, we derive $\mu_{A,2} \approx 6.667 \times 10^{-5}$, from which we deduce the following equilibrium values for the mosquito population on a domain with $N_h = 20000$ inhabitants:

A^*	M^*	F^*
58050	16254	23220

Table 4: Mosquito positive equilibrium

These will be the initial values in the following simulations. In Table 5, we consider epidemiological parameters related to a Dengue epidemic, because Dengue is circulating in La Réunion since 2019. We also assume that there is no impact of the virus on the infected vector's mean lifespan, i.e. $\mu_I = \mu_S$. Values for β_{hm}, β_{mh}, η_h and ν_m are taken from [1].

Symbol	B	β_{hm}	β_{mh}	μ_I	ν_m	μ_h	η_h	N_h
Value	1	0.375	0.375	1/10	1/8	$\frac{1}{365 \times 78}$	1/7	20000

Table 5: *DENV* epidemiological parameter values [1]

7.1. The constant and permanent SIT releases case

According to Table 5, the basic reproduction number is $\mathcal{R}_0^2 \approx 6.3476$ such that $F_{epi}^* = 3659$, and we can also estimate $\Lambda_F^{crit} \approx 366$ individuals. Note also that, when $\Lambda_F = 0$, $\mathcal{R}_{0,*}^2 \approx 6.547$.

Since $\mathcal{R}_0^2$ is close to $\mathcal{R}_{0,*}^2$, we have $\Lambda^*_{M,\mathcal{R}_0^2,\mathcal{C}} \approx 1611$, very close to $\Lambda_M^{crit} = 1613$, such that it is far better to consider massive releases, i.e. $\Lambda_M > \Lambda_M^{crit}$, like, for instance, a daily release rate of $\Lambda_M = 1650$ sterile male individuals. However, be careful, being just above the sterile threshold, implies that it will take a long time to be close from $TDFE$: the larger the release, the faster the system converges to $TDFE$.

In Table 6 we provide several computations of some threshold parameters, when $0 < \Lambda_F < \Lambda_F^{crit}$. When $\epsilon = 0.01$, that is $\Lambda_F = 17$, we have $\mathcal{R}^2_{0,SIT_c} = \mathcal{R}^2_{0,SIT_c,S} < 1$ because the release rate for sterile males is larger than Λ_M^{crit}. Once $\epsilon \geq 0.025$, sterile females are enough to make $\mathcal{R}^2_{0,SIT_c}$ above 1, because $\mathcal{R}^2_{0,SIT_c,S} > 1$.

ε	0	0.01	0.025	0.05	0.1
Λ_F	0	17	41	82	165
Λ_M	1650	1633	1609	1567	1485
$\Lambda^*_{M,\mathcal{R}_0^2,\mathcal{C}}$	1611	1613	1611	1599	1528
$\mathcal{R}^2_{0,*}$	6.547	6.074	5.512	4.744	3.729
$\mathcal{R}^2_{0,SIT_c,W}$	-	-	0.385	0.637	1.004
$\mathcal{R}^2_{0,SIT_c,S}$	-	0.0836	1.766	2.329	3.065
$\mathcal{R}^2_{0,SIT_c}$	-	0.0836	2.152	2.967	4.070

Table 6: Threshold values to lower the epidemiological risk for DENV

However, for a given $\varepsilon > 0$, as long as we consider large releases, such that $\tau\Lambda_M >> \dfrac{\Lambda_M^{crit}}{\mu_T}$, that is $(1-\varepsilon) \times M_{total} >> \dfrac{\Lambda_M^{crit}}{\mu_T}$, the wild population will decay to $\mathbf{0}$, whatever the amount of sterile females that are released.

However, for practical application, we have to be cautious because these results are only true for "long time" control. That is why, in the forthcoming simulations, we estimate $\mathcal{R}_{eff}$ at time $t = t_I$, for different starting time. It is important to understand that massive continuous or impulsive periodic SIT control, i.e. $\Lambda_M > \Lambda_M^{crit}$, with or without sterile females releases, cannot prevent from an outbreak, when the SIT starting time is too close from the outbreak starting time. The red stars in all figures indicate the minimal amount of sterile insects to release considering an infinite time to get $\mathcal{R}_{0,SIT_c} < 1$ ($\mathcal{R}^{per}_{0,SIT} < 1$)

Whatever the type of control, mechanical control is always recommended. Indeed, as showed in Table 7, page 29, the use of mechanical control allows to lower the threshold Λ_M^{crit}. In fact, when $\varepsilon = 0$, as expected, since Λ_M^{crit} depends linearly on K, the decay on Λ_M^{crit} is proportional to the level of mechanical control.

% of Mechanical control (days)	0	20%	40%	60%
Λ_M^{crit}	1613	1291	968	646
Improvement	0	20%	40%	60%

Table 7: Threshold values for the release rate to eliminate the wild population, thanks to different level of mechanical control - $\epsilon = 0$

Figs. 3, 4, and 5 show the impact of mechanical control (MC) to improve the efficiency of the SIT and also to reduce faster the population in order to get $\mathcal{R}_{eff}$ smaller than 0.5, once

the epidemic starts. With MC, SIT control time reduction can be substantial. For instance, assuming $\Lambda_M = 2000$, without MC, almost 400 days are necessary to get $\mathcal{R}_{eff} < 0.5$, while it takes only 180 days with 20% of MC, and 120 days with 40% of MC. Also, once Λ_M is ~~chosen~~ such that $\Lambda_M > \Lambda_M^{crit}$, for a sufficient long time, then $\mathcal{R}_{0,SIT} < 1$. Again, the positive impact of MC is obvious.

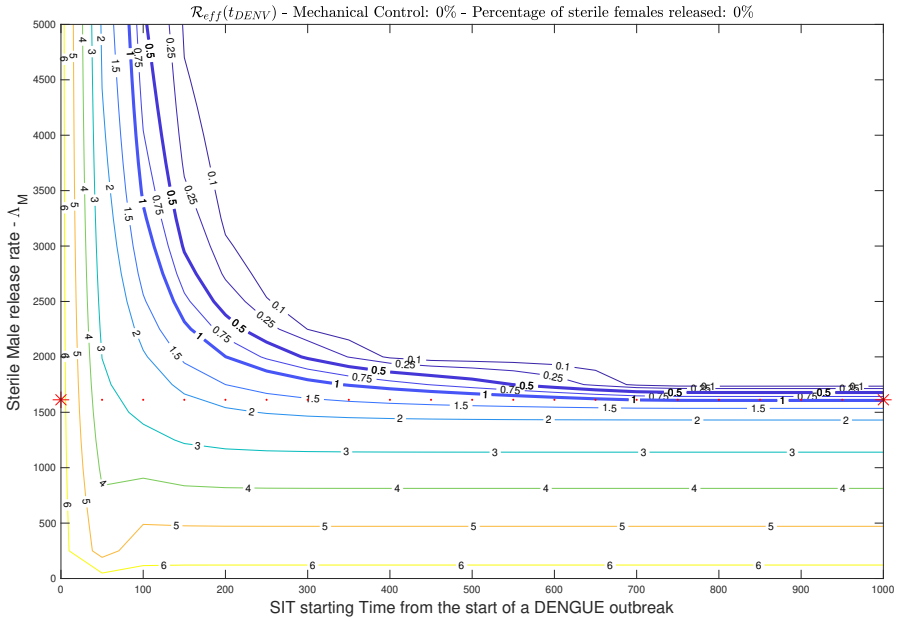

Figure 3: $\mathcal{R}_{eff}(t_I)$ vs the starting time and the level of the control - Without release of sterile females and without mechanical control

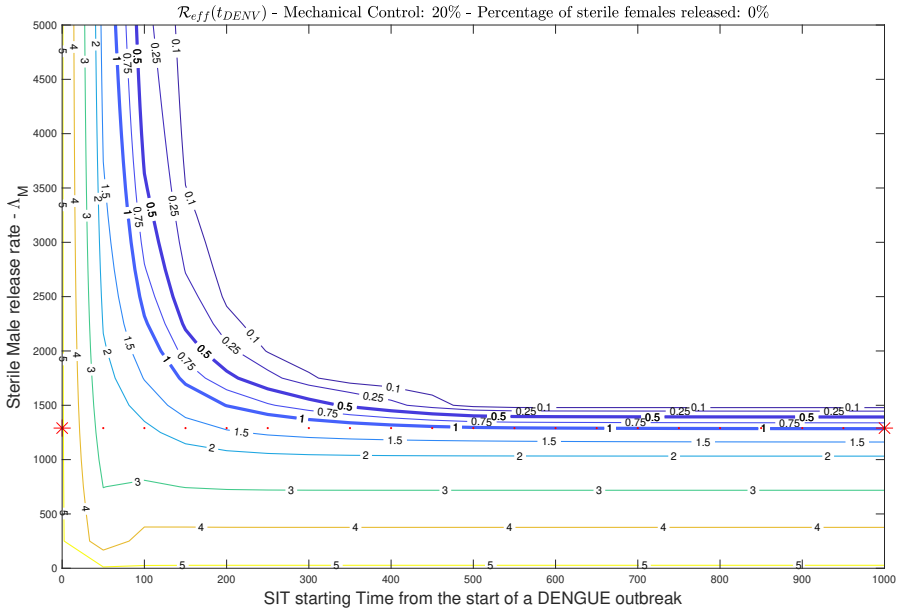

Figure 4: Impact of 20% of mechanical control on the effective reproduction number thanks to the starting time of the control - Without release of sterile females

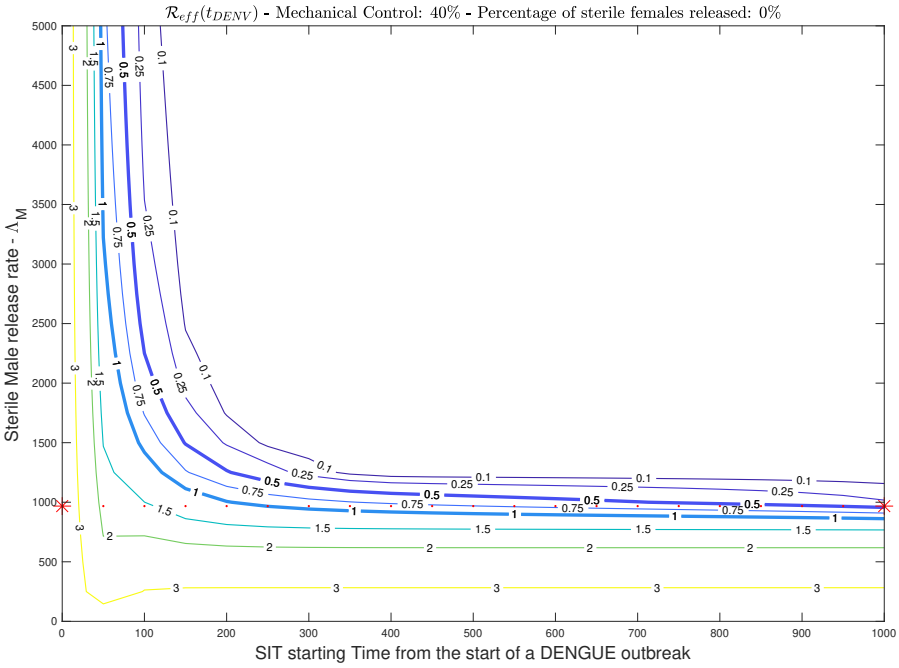

Figure 5: Impact of 40% of mechanical control on the effective reproduction number thanks to the starting time of the control - Without release of sterile females

Let us now consider the effect of releasing sterile females. In general, there is at most a small percentage of sterile females releases, but to illustrate our findings we will assume that

5% of sterile females are released: see Figs. 6-7-8. As observed in these Figs. (see the level set 0.5), the sterile male release rate has an impact on $\mathcal{R}_{eff}$, such that if the release is too large then $\mathcal{R}_{eff} > 0.5$. This makes sense because when the release rate is large, a significant amount of sterile females are released, such that they maintain $\mathcal{R}_{0,SIT}$ above 0.5. According to Fig. 6, the best release rate would be $\Lambda_M = 3000$ to get $\mathcal{R}_{eff}(t_{DENV}) < 0.5$ in the shortest time, here 250 days, approximately. The addition of MC (see Figs. 7-8) can reduce this time to 200 days for 20% of MC, and 175 days, and also, to reduce the release rate to 2500 or even 2000, for instance. This result make sense as MC lower the threshold release rate as showed in Table 8, page 32. The gain in time and in the release rate is very substantial and show again the great importance of MC, with or without sterile females releases.

% of Mechanical control (days)	0	20%	40%	60%
Λ_M^{crit}	1692	1357	992	594
Improvement	0	19.8%	41.4%	64.89%

Table 8: Threshold values for the release rate to eliminate the wild population, thanks to different level of mechanical control - $\epsilon = 0.05$

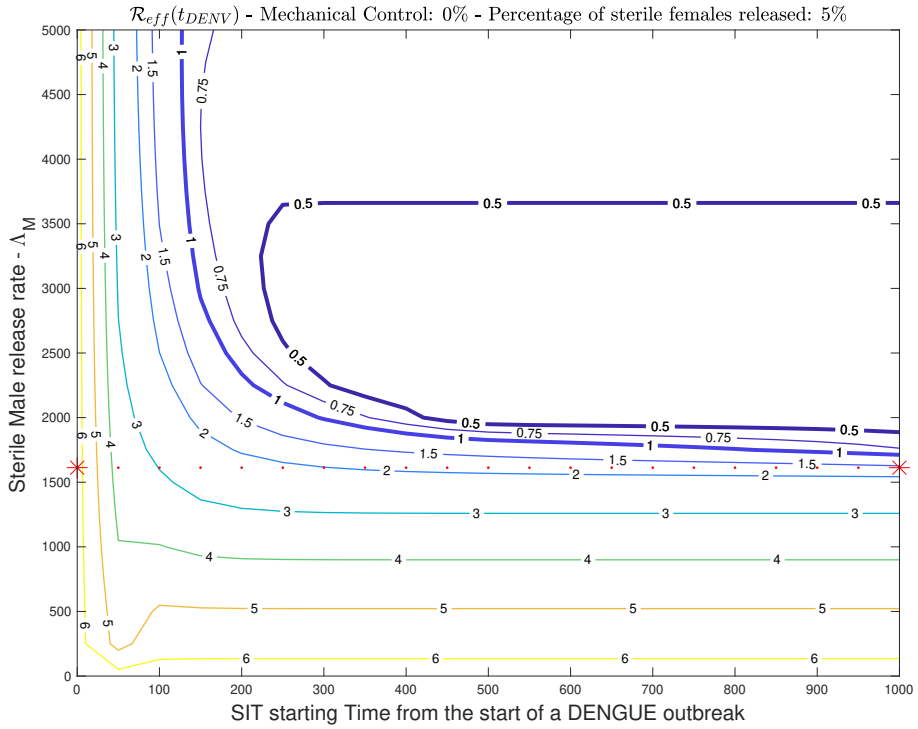

Figure 6: $\mathcal{R}_{eff}(t_I)$ vs the starting time and the level of the control including releases of sterile females

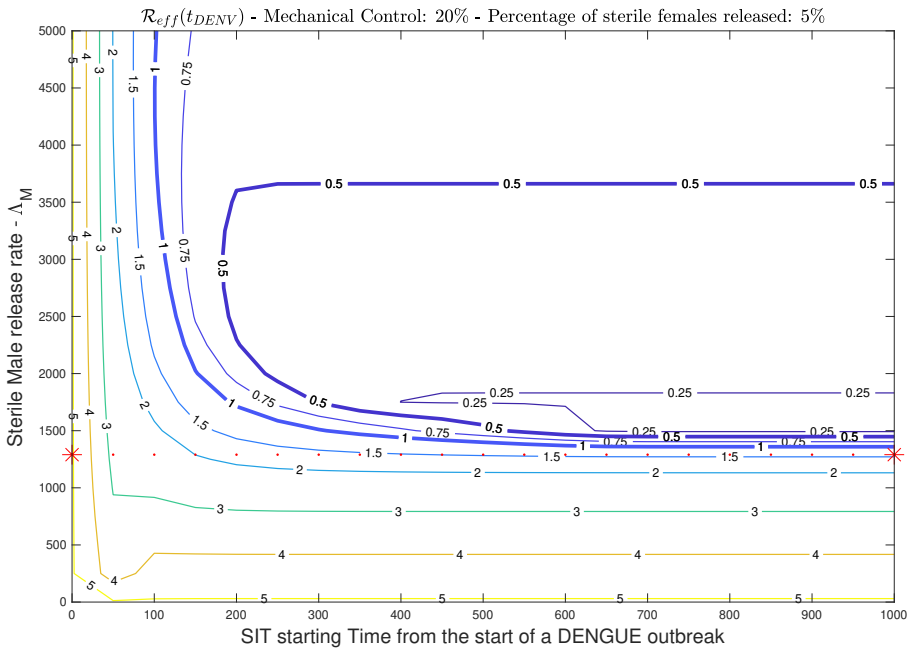

Figure 7: Impact of mechanical control on the effective reproduction number thanks to the starting time of the control - With release of sterile females.

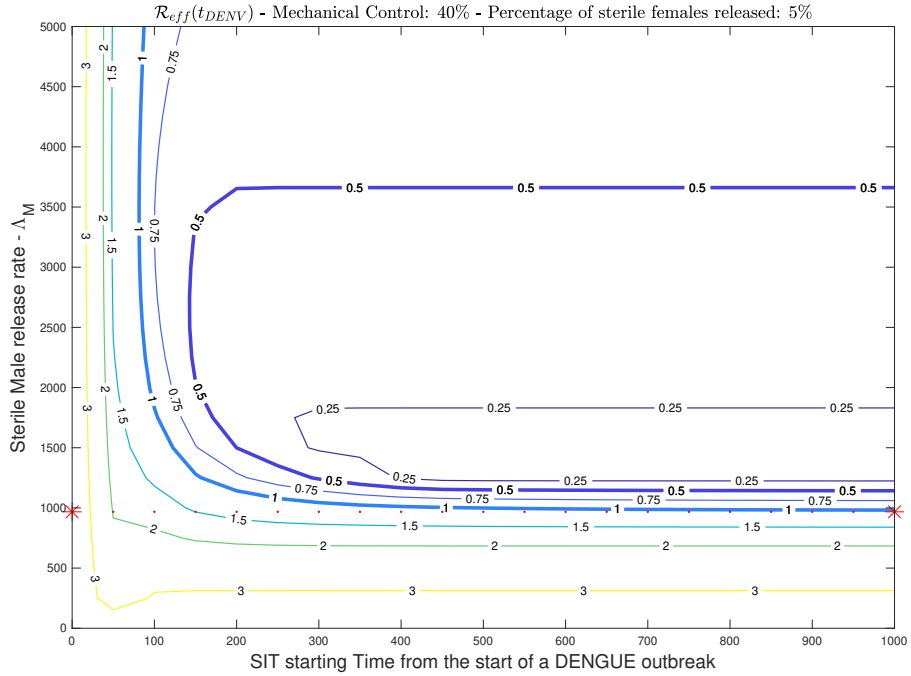

Figure 8: Impact of mechanical control on the effective reproduction number thanks to the starting time of the control - With release of sterile females

Let us discuss briefly the particular case $\Lambda_F > \Lambda_F^{crit}$. For that case, we know that

$\mathcal{R}^2_{0,SIT_c} > 1$, whatever the size of the releases. In particular, assuming that the proportion of sterile females is 5%, then releasing a total amount of 7320 sterile insects leads to release exactly 366 sterile females, i.e. $\Lambda_F \approx \Lambda_F^{crit}$, such that $\mathcal{R}^2_{0,SIT,c} > 1$. If very massive releases are used, then the percentage of sterile females within the releases have to be sufficiently small, in order to have $\Lambda_F < \Lambda_F^{crit}$. This shows that results like [19], where an admissible percentage of 4% of released sterile females is given, are useless if we don't know exactly the real sizes of the releases.

7.2. The impulsive periodic releases case

We now consider periodic impulsive releases, that is more realistic. We will estimate numerically the periodic basic reproduction number and also estimate the minimal release amount needed to decay the wild mosquito population to **0** for a given releases period, τ.

When $\varepsilon = 0$ (no release of sterile females), in Table 9, page 34, we derive numerically the threshold, $\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$, and the mean number of sterile insects, for a given τ. It is interesting to notice that for $\tau = 5$, the release rate is close to the release rate obtained for the continuous daily rate, $\Lambda^*_{M,\mathcal{R}^2_0,\mathcal{C}} = 1611$.

τ (days)	5	7	10	14
$\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$	1670	1723	1842	2080
Nb of insects per release	8350	12061	18420	29120
Mean number of sterile males over one period	11690	12061	12894	14560

Table 9: Threshold values to lower the epidemiological risk for DENV, i.e. $\mathcal{R}^{per}_{0,SIT} < 1$

We consider $\tau = 7$, because, in the field, weekly releases are usually considered. With different level of mechanical control, including also the release of sterile females (like previously, 5%), we estimate numerically the minimal amount of insect to ensure that $\mathcal{R}^{per}_{0,SIT}$ is less than one. This is based on a numerical estimate of $\mathcal{R}^{per}_{0,SIT}$, like in [14].

% of mechanical control (days)	0	20%	40%
$\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$	1723	1359	966
Mean number of sterile males	12061	9513	6762

Table 10: Weakly releases ($\tau = 7$) - Threshold values to lower the epidemiological risk for DENV, i.e. $\mathcal{R}^{per}_{0,SIT} < 1$, without release of sterile females, $\Lambda_F = 0$

% of mechanical control (days)	0	20%	40%
$\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$	1817	1451	1053
Mean number of sterile insects over one period	12719	10157	7371

Table 11: Weakly releases ($\tau = 7$) - Threshold values to lower the epidemiological risk for DENV, i.e. $\mathcal{R}^{per}_{0,SIT} < 1$, with the release of 5% (from $\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$) of sterile females

In the following simulations, we derive an estimate of $\mathcal{R}_{eff}(t_{DENGV})$, when the Dengue epidemic starts, for weekly periodic impulsive releases, with different level of Mechanical Control, also including (or not) the release of sterile females.

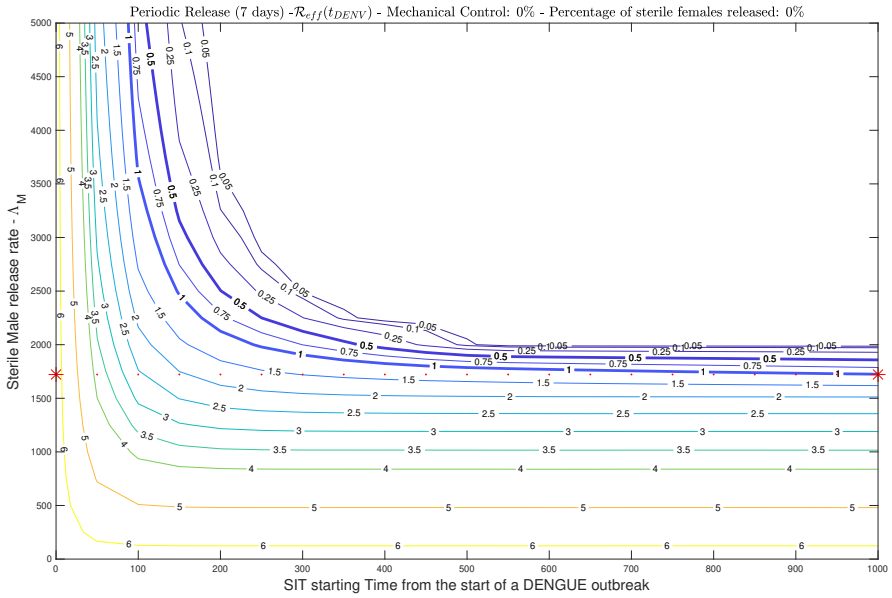

Figure 9: Weekly periodic releases. Estimate of the effective reproduction number thanks to the starting time of the control - Without release of sterile females and without mechanical control

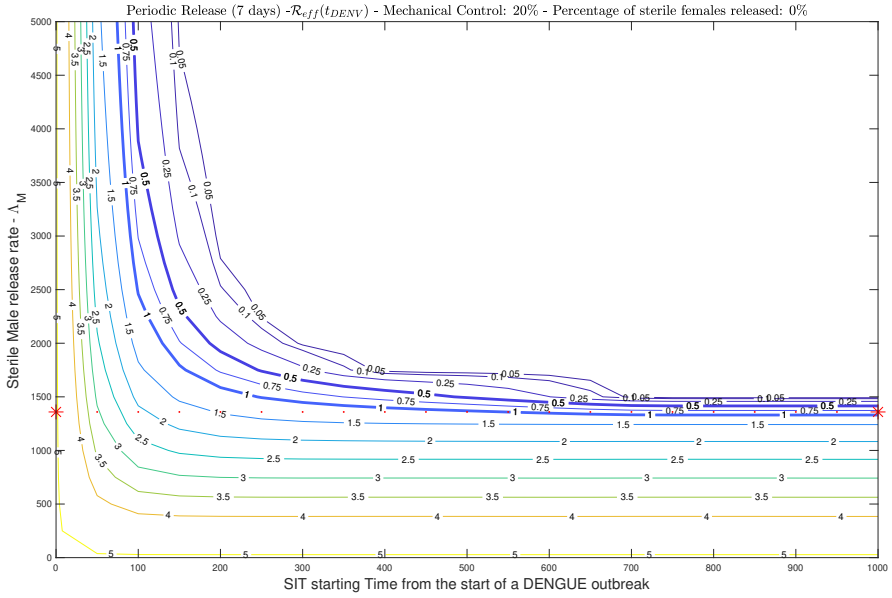

Figure 10: Weekly periodic releases. Impact of mechanical control (20%) on the effective reproduction number - No release of sterile females

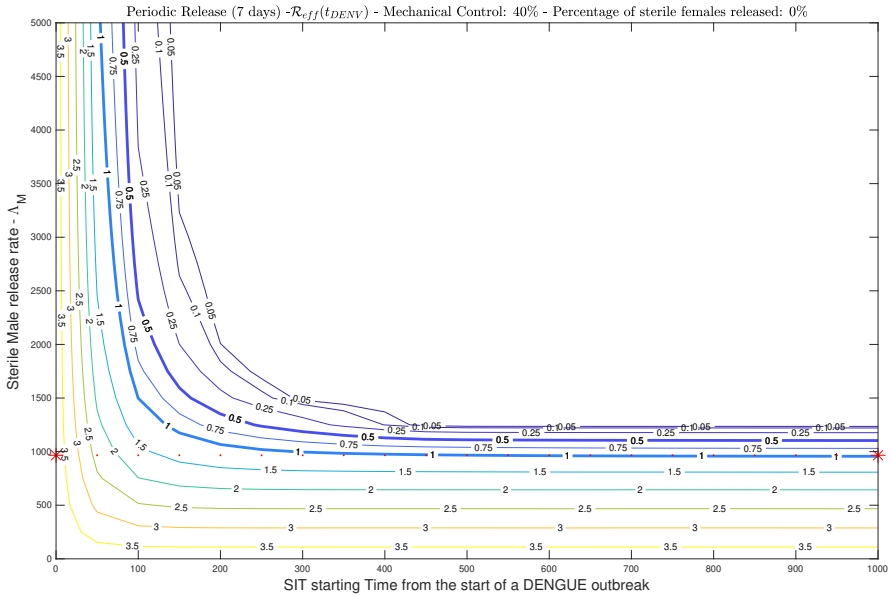

Figure 11: Weekly periodic releases. Impact of mechanical control (40%) on the effective reproduction number - No release of sterile females

Comparing Figs. 9, 10, and 11, we can highlight the great impact of mechanical control (see also Table 10) that allows to reduce the size and even the duration of the SIT control. For instance, in the continuous case, choosing $\Lambda_M = 3000$, it requires 210 days to get $\mathcal{R}_{eff} < 0.5$ without MC, 150 days when $MC = 20\%$, and 110 days when $MC = 40\%$. The red dotted lines indicate the minimal threshold, $\Lambda^*_{\mathcal{R}^{per}_{0,SIT}}$, for the release rate to get $\mathcal{R}^{per}_{0,SIT} < 1$. The readers have to be aware that it can take a long time (and thus a lot of releases) to have $\mathcal{R}^{per}_{0,SIT} < 1$, as showed in Fig. 9. Clearly, large periodic releases, like for continuous releases, are more interesting. However, this might depend on the sterile insects production facilities and also the area to treat. As usual, a balance has to be find.

Let's turn now to the release of sterile females. Like for the continuous case, we consider that, among the sterile insects that are released, 5% are females. As showed in Figs. 12, 13, and 14, we derive the same simulations than before and recover somehow the same results: very large release can induce a negative effect because the amount of sterile females can be so large that it is impossible to decay $\mathcal{R}^{per}_{0,SIT}$ below 0.5, for instance. Following Remark 11: since $(1-\varepsilon)M_{total} = \tau\Lambda_M > M^{crit}_{T,per}$, it is important to notice that as long as $\Lambda_F < \Lambda^{crit}_F \approx 366$, we will have $\mathcal{R}^{per}_{0,SIT} < 1$. Of course, it we want $\mathcal{R}^{per}_{eff} < 0.5$, then we need to check that $\Lambda_F < \Lambda^{crit}_F/2 \approx 183$.

Again, the combination with mechanical control is clearly beneficial and can help lower the time needed to reduce $\mathcal{R}_{eff}$ below 1 or 0.5 and also the size of the releases. For instance, according to Fig. 9, the best release rate would be $\Lambda_M = 3500$ without MC, while for $MC = 40\%$, the release rate $\Lambda_M = 2500$ would decay $\mathcal{R}_{eff}$ below 0.5 in only 150 days.

Globally, if SIT, combined with mechanical control, started early compared to the "start" of an epidemic, the release of sterile females, in a reasonable amount (no more than 5%) does not have an impact on the final efficiency of the SIT control. The only issue would be to consider very massive releases such that a sufficient amount of sterile females would

"artificially" maintain $\mathcal{R}_{0,SIT}^{per}$ above 1. However, if the aim is to reduce the basic reproduction number below 0.5, then our simulations show that the releases rate has to belong between 1500 and 4000 individuals, meaning that every week between 10500 and 28000 sterile males have to be released.

In order to better illustrate our explanations, we provide additional simulations of the time evolution of infected humans with and without SIT control, combined or not with mechanical control (40%). In Figs. 15(a), 15(c), and Fig. 16(a), 16(c), page 40, we show how the dynamic of infected humans can vary according to the size of the releases and the use or not of mechanical control: while SIT control decays the maximum number of infected individuals, it also leads to a spreading of the epidemic which can increase the number of cumulative cases, as showed in Figs. 15(b), 15(d), Fig. 16(b), and 16(d), page 40. Clearly, also, the use of mechanical control allows to reduce significantly the size of the periodic releases needed to avoid an epidemic. Last, the releases of sterile females impact the maximum of the Infected compartment, but in a very limited way. This follows our conclusion that even if irradiated females are released (at a reasonable rate), while a DENV virus is circulating, this will not impact, i.e. favor, the dynamic of the epidemic.

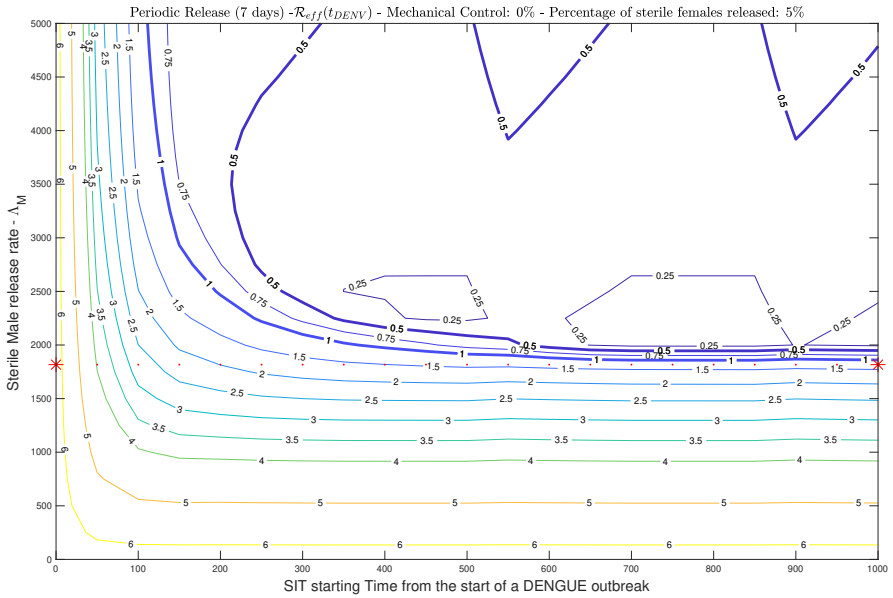

Figure 12: Weekly periodic releases. Estimate of the effective reproduction number thanks to the starting time of the control - With the release of sterile females and without mechanical control

8. Conclusion

In this work we have considered SIT control, based on sterile males releases, coupled with an epidemiological model, taking into account that sterile females can be (accidentally) released too. We showed that a low SIT control is possible but only if the basic reproduction number, $\mathcal{R}_0$, is lower than a certain threshold, $\mathcal{R}_0^*$, that depends on the basic offspring number, $\mathcal{N}$, related to the mosquito species. If not, then only massive releases (above the threshold, Λ_M^{crit} or $M_{T,per}^{crit}$) are useful. This is an important result as it can change the SIT control strategy, locally or along the year since all these threshold parameters can change.

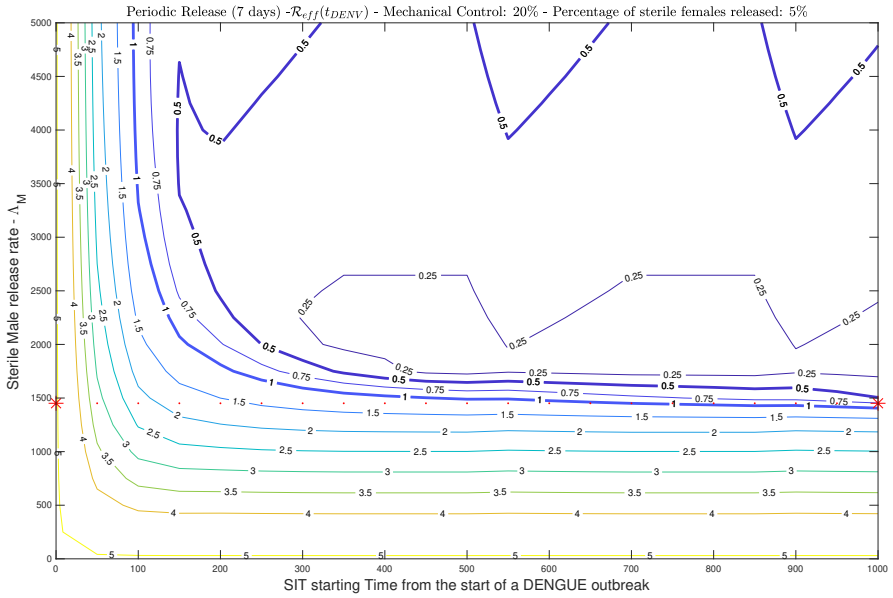

Figure 13: Weekly periodic releases. Estimate of the effective reproduction number thanks to the starting time of the control - With the release of sterile females and 20% of mechanical control

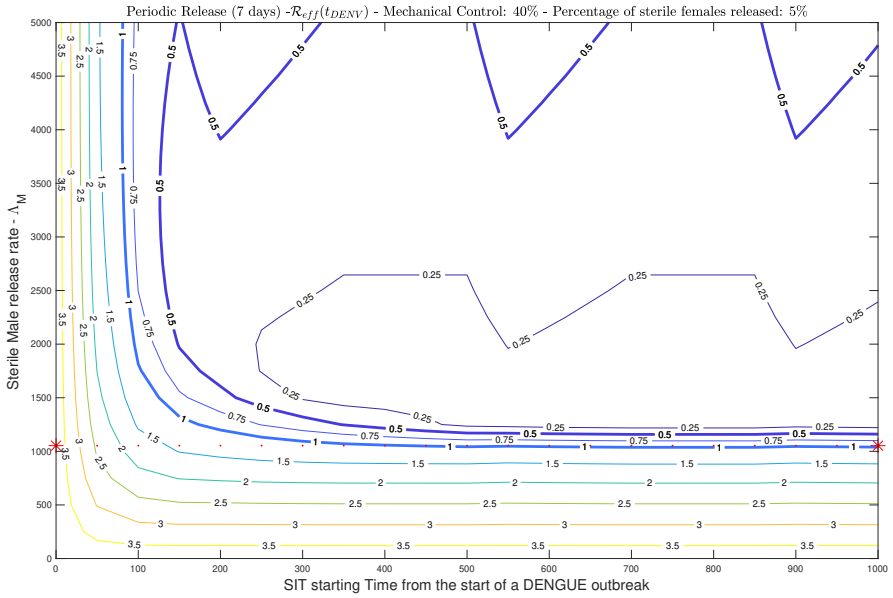

Figure 14: Weekly periodic releases. Estimate of the effective reproduction number thanks to the starting time of the control - With the release of sterile females and 40% of mechanical control

For mosquitoes, sex-separation is already an issue even if new approaches, like Genetic Sex Strain, are in development [17]. Until these new technologies are operational, it is worth, at least for the health authorities and also to reassure local people, to study if the release of sterile females during a SIT campaign can be problematic or not. We showed that as long as

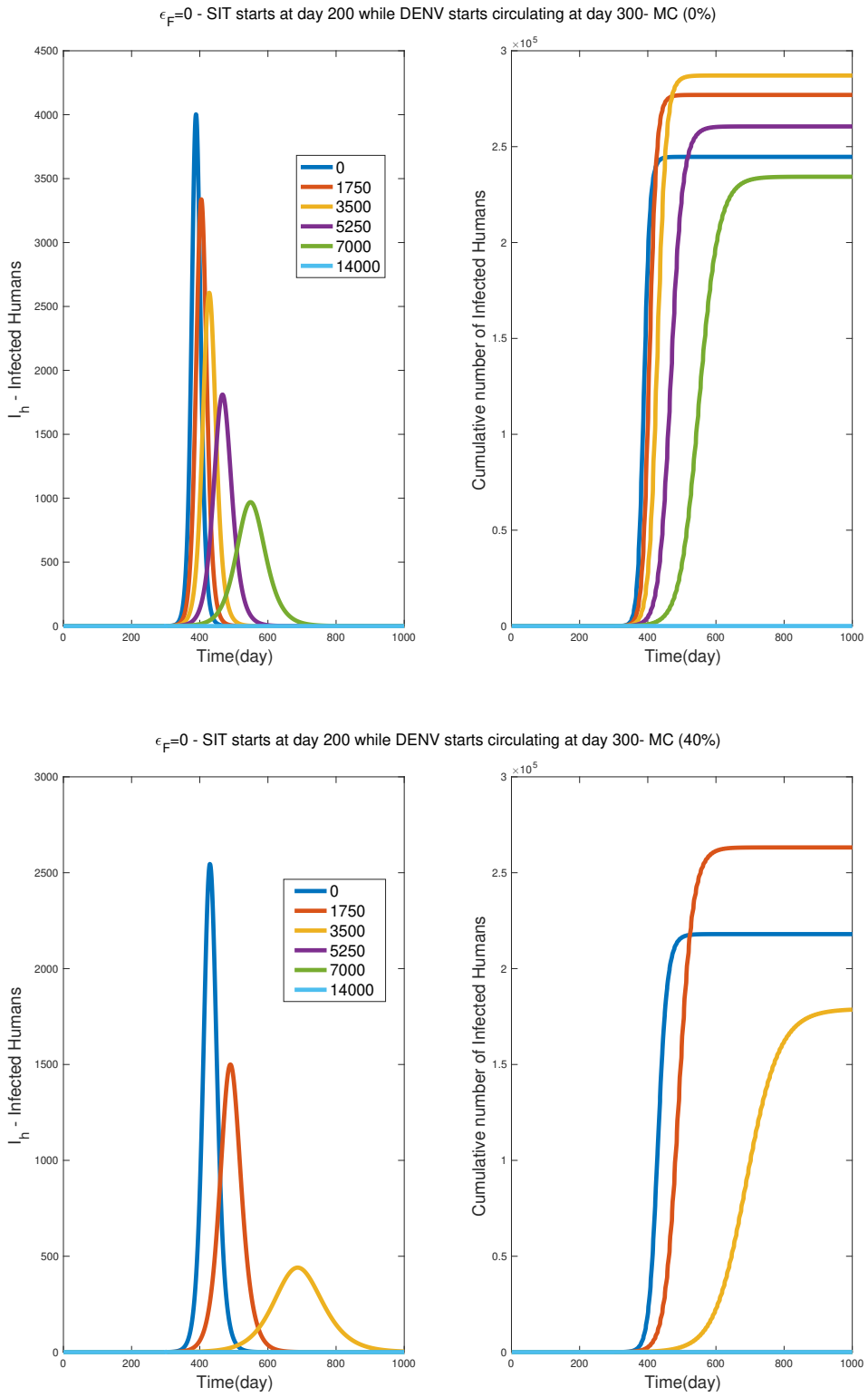

Figure 15: Weakly periodic releases: Time evolution of I_h, the infected humans, and the cumulative number of infected humans when DENV starts circulating at day 300 while SIT starts at time t=200 - without and with 40% of mechanical control - No release of sterile females, $\varepsilon = 0$

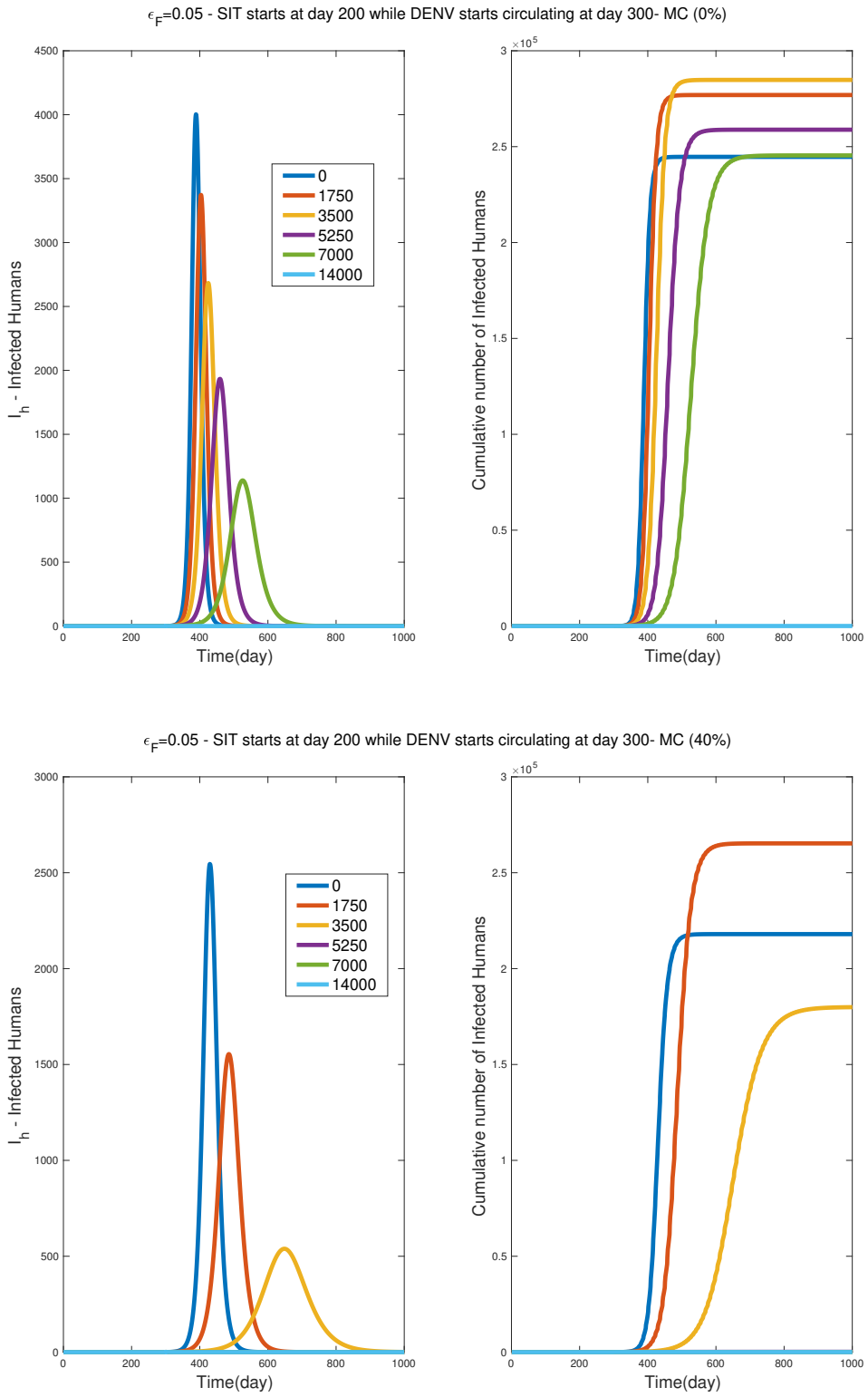

Figure 16: Weakly periodic releases: Time evolution of I_h, the infected humans, and the cumulative number of infected humans when DENV starts circulating at day 300 while SIT starts at time t=200 - 40% of mechanical control - with release of sterile females ($\varepsilon = 0.05$)

the sterile females release rate, Λ_F, is below the threshold Λ_F^{crit}, then SIT control can already be efficient, provided that it starts early enough. We emphasized the fact that if (very) massive releases are considered, the amount of released females has to be small enough, such that $\Lambda_F < \Lambda_F^{crit}$. This result is of primary importance because, when SIT is not efficient, the tendency is to increase the size of the releases: outside an epidemic period, this is not an issue, but it is when a virus is circulating. Thus, the recommendation about the maximal percentage of sterile females to release, given by the IAEA, is not useful because we show that it is the amount of sterile females released that matters.

We illustrated our theoretical findings on Dengue parameters since several dengue viruses are circulating in La Réunion where our SIT project takes place (see [6] for explanations).

Our recommendation, in the absence of virus circulation, is to start SIT control, combined with mechanical control, even when sterile females are released, as early as possible before the risky epidemic season in order to decrease the mosquito population, such that, for instance, $\mathcal{R}_{0,SIT} < 0.5$, and then, continue the SIT control with (very) small releases, as described and illustrated in [2, 5].

Our model can be improved by taking into account that sterile males cannot not necessarily be fully sterilized, such that a certain proportion, ϵ, of residual fertility can occur. This has been studied in [6], where we showed that ϵ needs to be less than $1/\mathcal{N}$ in order to keep the SIT efficient. Most certainly the combination of residual fertility and sterile females releases could be problematic and that is why they have to be studied seriously in order to avoid failures in SIT programs.

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Appendix A. Proof of Lemma 1

1. The right-hand side of system (15)-(16) is continuous and continuously differentiable on $\mathbb{R}^8$. Thus, according to [31, Theorem III.10VI], for any initial condition in $\mathbb{R}^8$, a unique solution exists, at least locally. The vector field defined by the right-hand side of system (15) − (16) is either tangential or directed inwards on the boundary of $\mathcal{D}''$. Hence, $\mathcal{D}''$ is positively invariant by system (15)-(16). Now, we use the notion of invariant regions, see e.g. [28, Chapter 14, pages 198-212], to prove the positive invariance of the set $\Gamma_{\mathcal{N}>1}$. By adding equations of system (15), it is straightforward to deduce that, for all $t \geq 0$, the set $S(t) + I(t) + R(t) = N$ is positively invariant. From system (16), we deduce that:

$$\begin{cases} \dfrac{dA}{dt} & = \phi(F_S + F_E + F_I) - (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ \dfrac{dM}{dt} & = (1-r)\gamma A - \mu_M M, \\ \dfrac{d(F_S + F_E + F_I)}{dt} & \leq r\gamma A - \mu_S(F_S + F_E + F_I). \end{cases} \tag{A.1}$$

Let f be the right-hand side of system (A.1). Assume that $\mathcal{N} > 1$. Hence the positive wild equilibrium $E^* = (A^*, M^*, F^*)'$ is defined. Recall that

$$M^* = \frac{(1-r)\gamma}{\mu_M}A^* \quad \text{and} \quad F^* = \frac{r\gamma}{\mu_S}A^* = \frac{(\gamma + \mu_{A,1} + \mu_{A,2}A^*)A^*}{\phi}$$

. Let us consider

$$G_1 = M - M^*,\ G_2 = F_S + F_E + F_I - F^*,\ G_3 = A - A^*.$$

One has:

$$\begin{aligned} &\nabla G_1 \cdot f|_{M=M^*} = (1-r)\gamma(A - A^*) \leq 0 \text{ in } \Gamma_{\mathcal{N}>1}, \text{ so } M \leq M^*. \\ &\nabla G_2 \cdot f|_{F_S+F_E+F_I=F^*} \leq r\gamma(A - A^*) \leq 0 \quad \text{in} \quad \Gamma_{\mathcal{N}>1}, \quad \text{so} \quad F_S + F_E + F_I \leq F^*. \\ &\nabla G_3 \cdot f|_{A=A^*} = \phi\,(F_S + F_E + F_I) - (\gamma + \mu_{A,1} + \mu_{A,2}A^*)A^* \\ &\qquad\qquad\qquad\qquad = \phi\,(F_S + F_E + F_I - F^*) \\ &\qquad\qquad\qquad\qquad \leq 0 \quad \text{in} \quad \Gamma_{\mathcal{N}>1}, \text{ so } A \leq A^*. \end{aligned}$$

2. Based on he uniform boundedness, we deduce that the solutions of system (15)-(16) exist globally, for all $t \geq 0$. Therefore, system (15)-(16) defines a dynamical system on $\mathcal{D}''$.

This ends the proof of the Lemma.

Appendix B. Proof of Theorem 3, page 11

1. Following [9], we rewrite system (15)-(16) by splitting the uninfected compartments $x = (S, R, A, M, F_S)'$ from the infected compartments $y = (I, F_E, F_I)^T$. That is

$$\begin{cases} \dfrac{dx}{dt} = f(x, y), \\ \dfrac{dy}{dt} = g(x, y), \end{cases}$$

such that $g(x,0)=0$. When $\mathcal{N}>1$, thanks to the results obtained in section 3, it is straightforward to show that the equilibrium $x^*=(N_h,0,A^*,M^*,F_S^*)^T$ is GAS for system $\dfrac{dx}{dt}=f(x,0)$. Let us consider $\dfrac{dy}{dt}=g(x,y)$, that is

$$\begin{cases} \dfrac{dI_h}{dt} &= B\beta_{mh}\dfrac{F_I}{N_h}S_h-(\eta_h+\mu_h)\,I_h,\\ \dfrac{dF_E}{dt} &= B\beta_{hm}\dfrac{I_h}{N_h}F_S-(\mu_E+\nu_m)F_E,\\ \dfrac{dF_I}{dt} &= \nu_m F_E-\mu_I F_I, \end{cases} \tag{B.1}$$

that can be rewritten as

$$\frac{dy}{dt}=\mathcal{F}(x,y)-\mathcal{V}(x,y)=(F-V)y-h(x,y),$$

where

$$\mathcal{F}(x)=\begin{pmatrix} B\beta_{mh}\dfrac{F_I}{N_h}S_h\\ B\beta_{hm}\dfrac{I_h}{N_h}F_S\\ 0 \end{pmatrix},\qquad \mathcal{V}(x)=\begin{pmatrix} (\eta_h+\mu_h)I_h\\ (\nu_m+\mu_S)F_E\\ \mu_I F_I-\nu_m F_E \end{pmatrix}, \tag{B.2}$$

F and V the Jacobian matrices associated with $\mathcal{F}$ and $\mathcal{V}$ at $(x^*,0_{\mathbb{R}^3})'$, that is

$$F=\begin{pmatrix} 0 & 0 & B\beta_{mh}\\ B\beta_{hm}\dfrac{F_S^*}{N_h^*} & 0 & 0\\ 0 & 0 & 0 \end{pmatrix},\qquad V=\begin{pmatrix} \eta_h+\mu_h & 0 & 0\\ 0 & \mu_S+\nu_m & 0\\ 0 & -\nu_m & \mu_I \end{pmatrix}, \tag{B.3}$$

where F_S^* is given by (7), and $h(x,y)=(F-V)y-(\mathcal{F}(x,y)-\mathcal{V}(x,y))$. In fact, we have

$$h(x,y)=\begin{pmatrix} B\beta_{mh}F_I(1-S_h/N_h)\\ B\beta_{hm}\dfrac{I_h}{N_h}(F_S^*-F_S)\\ 0 \end{pmatrix}\geq 0.$$

In addition $F-V$ is a Metzler matrix. When $\mathcal{R}_0^2\leq 1$, DFE is LAS and since assumptions (H1) and (H2) in [9] are verified, DFE is GAS when $\mathcal{R}_0^2\leq 1$.

2. The Jacobian matrix of system (15)-(16) at the DFE is

$$J_{DFE}=\begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & -B\beta_{mh}\\ 0 & 0 & -\eta_h-\mu_h & 0 & 0 & 0 & 0 & B\beta_{mh}\\ 0 & \eta_h & -\mu_h & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & -\gamma-\mu_{A,1}-2\mu_{A,2}A^* & 0 & \phi & \phi & \phi\\ 0 & 0 & 0 & (1-r)\,\gamma & -\mu_M & 0 & 0 & 0\\ 0 & -B\beta_{hm}\dfrac{F_S^*}{N_h} & 0 & r\gamma & 0 & -\mu_S & 0 & 0\\ 0 & B\beta_{hm}\dfrac{F_S^*}{N_h} & 0 & 0 & 0 & 0 & -(\mu_S+\nu_m) & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & \nu_m & -\mu_I \end{pmatrix}$$

Eigenvalues of J_{DFE} are: $-\mu_M$, $-\mu_h$, $-\mu_h$, the solutions of the following equation in λ:

$$\lambda^2 + (\mu_S + \mu_{A,1} + 2\mu_{A,2}A^* + \gamma)\lambda + \mu_S\mu_{A,2}A^* = 0 \tag{B.4}$$

and the eigenvalues of the matrix Z defined by

$$Z = \begin{pmatrix} -(\eta_h + \mu_h) & 0 & B\beta_{mh} \\ B\beta_{hm}F_S^*/N_h & -(\mu_S + \nu_m) & 0 \\ 0 & \nu_m & -\mu_I \end{pmatrix}.$$

Eigenvalues of the matrix Z are solutions of the cubic equation in λ

$$\begin{aligned}\lambda^3 + \lambda^2(\eta_h + \mu_h + \mu_S + \nu_m + \mu_I) + \lambda((\eta_h + \mu_h)(\mu_S + \nu_m) + \mu_I(\eta_h + \mu_h + \mu_S + \nu_m)) \\ +\mu_I(\mu_S + \nu_m)(\eta_h + \mu_h)(1 - \mathcal{R}_0^2) = 0.\end{aligned} \tag{B.5}$$

It is not hard to see that roots of (B.4) have negative real part and that when $\mathcal{R}_0 = 1$, (B.5) admits zero as a root and a negative root. Moreover,

$$\mathcal{R}_0^2 = 1 \iff B = B^* = \sqrt{\frac{\nu_m + \mu_S}{\nu_m}\frac{\mu_I}{\beta_{mh}}\frac{\eta_h + \mu_h}{\beta_{hm}}\frac{N_h}{F_S^*}}.$$

A right eigenvector of J_{NTDFE} corresponding to the zero eigenvalue is $w = (w_i)_{i=1,\dots,8}$ where

$$\begin{aligned}
w_1 &= -\frac{B^*\beta_{mh}}{\mu_h} < 0, \\
w_2 &= \frac{B^*\beta_{mh}}{\eta_h + \mu_h} > 0, \\
w_3 &= \frac{\eta_h}{\mu_h}w_2 > 0, \\
w_4 &= \frac{\phi\left(\mu_S\left(1 + \frac{\mu_I}{\nu_m}\right) - \mu_I\left(1 + \frac{\mu_S}{\nu_m}\right)\right)}{\mu_S\mu_{A,2}A^*}, \\
&= \frac{\phi\,(\mu_S - \mu_I)}{\mu_S\mu_{A,2}A^*} < 0 \quad \text{because} \quad \mu_S < \mu_I, \\
w_5 &= \frac{(1-r)\gamma}{\mu_M}w_4 < 0, \\
w_6 &= \frac{r\gamma\phi\left(1 + \frac{\mu_I}{\nu_m}\right) - (\gamma + \mu_{A,1} + 2\mu_{A,2}A^*)\mu_I\left(1 + \frac{\mu_S}{\nu_m}\right)}{\mu_S\mu_{A,2}A^*}, \\
w_7 &= \frac{\mu_I}{\nu_m} > 0, \\
w_8 &= 1 > 0.
\end{aligned}$$

Since

$$\mu_S < \mu_I,\ \mu_S(\gamma+\mu_{A,1}+2\mu_{A,2}A^*) - r\gamma\phi = \mu_S\mu_{A,2}A^* > 0,\ r\gamma\phi - \mu_I(\gamma+\mu_{A,1}+2\mu_{A,2}A^*) < 0,$$

we have

$$w_6 = \frac{r\gamma\phi - (\gamma + \mu_{A,1} + 2\mu_{A,2}A^*)\mu_I - \mu_S\mu_{A,2}A^*\frac{\mu_I}{\nu_m}}{\mu_S\mu_{A,2}A^*} < 0.$$

A left eigenvector of J_{NTDFE} corresponding to the zero eigenvalue is $v = (v_i)_{i=1,\dots,8}$ where $v_1 = v_3 = v_4 = v_5 = v_6 = 0$ and

$$\begin{aligned} v_2 &= \frac{B^*\beta_{hm}}{\eta_h + \mu_h}\frac{F_S^*}{N_h}, \\ v_7 &= 1, \\ v_8 &= \frac{\nu_m + \mu_S}{\nu_m}. \end{aligned}$$

Following [10], the only non-zero term to be considered are the ones that correspond to v_2, v_7 and v_8. From system (15)-(16), one deduces that

$$a = 2v_2\frac{B^*\beta_{mh}}{N_h}w_1w_8 + 2v_7\frac{B^*\beta_{hm}}{N_h}w_2w_6 < 0$$

and

$$b = v_2w_8\beta_{mh} + v_7w_2\beta_{hm}\frac{F_S^*}{N_h} > 0.$$

Hence, it follows from [10, Theorem 4.1 & Remark 1], that system (15)-(16) undergoes a forward bifurcation or transcritical bifurcation when $\mathcal{R}_0 = 1$.

3. Based on [30, Theorem 2], we deduce that when $\mathcal{R}_0^2 > 1$, then the DFE is unstable.

Appendix C. Proof of Proposition 2, page 12

To find the positive or endemic equilibrium, $EE = (S_h^\sharp, I_h^\sharp, R_h^\sharp, A^\sharp, M^\sharp, F_S^\sharp, F_E^\sharp, F_I^\sharp)^T$, we have to solve the following system

$$\begin{cases} \mu_hN_h - B\beta_{mh}\dfrac{F_I}{N_h}S_h = \mu_hS_h \\ B\beta_{mh}\dfrac{F_I}{N_h}S = (\eta_h + \mu_h)\, I_h, \\ \eta_hI_h = \mu_hR_h, \\ \phi(F_S + F_E + F_I) = (\gamma + \mu_{A,1} + \mu_{A,2}A)A, \\ (1-r)\gamma A = \mu_M M, \\ r\gamma A - B\beta_{hm}\dfrac{I_h}{N_h}F_S = \mu_SF_S, \\ B\beta_{hm}\dfrac{I_h}{N_h}F_S = (\mu_S + \nu_m)F_E, \\ \nu_mF_E = \mu_IF_I. \end{cases} \tag{C.1}$$

Using $(C.1)_6$, $(C.1)_7$ and $(C.1)_8$, we deduce

$$F_E^\sharp = \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h}F_S^\sharp, \tag{C.2}$$

$$F_I^\sharp = \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}F_S^\sharp. \tag{C.3}$$

and

$$\left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)F_S^\sharp = \frac{r\gamma}{\mu_S}A^\sharp. \tag{C.4}$$

Using $(C.1)_4$, we obtain

$$F_S^\sharp + F_E^\sharp + F_I^\sharp = \frac{\gamma + \mu_{A,1} + \mu_{A,2}A^\sharp}{\phi} A^\sharp.$$

Thus

$$\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right) F_S^\sharp = \frac{\gamma + \mu_{A,1} + \mu_{A,2}A^\sharp}{\phi} A^\sharp, \tag{C.5}$$

that is, for $A^\sharp \neq 0$,

$$\phi\frac{r\gamma}{\mu_S}\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right) = \left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)\left(\gamma + \mu_{A,1} + \mu_{A,2}A^\sharp\right),$$

or

$$\mathcal{N}\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right) = \left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)\left(1 + \frac{\mu_{A,2}}{\gamma + \mu_{A,1}}A^\sharp\right), \tag{C.6}$$

from which we deduce

$$A^\sharp = \frac{\gamma + \mu_{A,1}}{\mu_{A,2}}\left(\mathcal{N}\frac{\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right)}{\left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)} - 1\right). \tag{C.7}$$

Note that, when $\mu_I = \mu_S$, the previous relationship reduces to

$$A^\sharp = \frac{\gamma + \mu_{A,1}}{\mu_{A,2}}\left(\mathcal{N} - 1\right).$$

Then, using (C.4), we deduce

$$\left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right) F_S^\sharp = \frac{r\gamma}{\mu_S}\frac{\gamma + \mu_{A,1}}{\mu_{A,2}}\left(\mathcal{N}\frac{\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right)}{\left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)} - 1\right),$$

such that, using (C.3), we derive

$$F_I^\sharp = \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\frac{r\gamma}{\mu_S}\frac{\gamma + \mu_{A,1}}{\mu_{A,2}}\frac{1}{1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}}\left(\mathcal{N}\frac{\left(1 + \frac{B\beta_{hm}}{\mu_S + \nu_m}\frac{I_h^\sharp}{N_h} + \frac{\nu_m}{\nu_m + \mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right)}{\left(1 + \frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)} - 1\right). \tag{C.8}$$

Then, using $(C.1)_2$,

$$B\beta_{mh}F_I^\sharp\left(1 - \left(\frac{\eta_h + \mu_h}{\mu_h}\right)\frac{I_h^\sharp}{N_h}\right) = \left(\eta_h + \mu_h\right)I_h^\sharp,$$

we replace $F_I^\sharp$ in (C.8) to obtain the following equation for $I_h^* \neq 0$

$$\frac{1}{N_h}\frac{\nu_m}{\nu_m+\mu_S}\frac{B\beta_{mh}B\beta_{hm}}{(\eta_h+\mu_h)\,\mu_I}\frac{r\gamma}{\mu_S}\frac{\gamma+\mu_{A,1}}{\mu_{A,2}}\left(\mathcal{N}\frac{\left(1+\frac{B\beta_{hm}}{\mu_S+\nu_m}\frac{I_h^\sharp}{N_h}+\frac{\nu_m}{\nu_m+\mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right)}{\left(1+\frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)}-1\right)\times \left(1-\left(\frac{\eta_h+\mu_h}{\mu_h}\right)\frac{I_h^\sharp}{N_h}\right)=1+\frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h},$$

or equivalently

$$\mathcal{R}_0^2\left(\mathcal{N}\left(1+\frac{B\beta_{hm}}{\mu_S+\nu_m}\frac{I_h^\sharp}{N_h}+\frac{\nu_m}{\nu_m+\mu_S}\frac{B\beta_{hm}}{\mu_I}\frac{I_h^\sharp}{N_h}\right)-\left(1+\frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)\right)\left(1-\left(\frac{\eta_h+\mu_h}{\mu_h}\right)\frac{I_h^\sharp}{N_h}\right)= (\mathcal{N}-1)\left(1+\frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)^2.$$

That is,

$$\mathcal{R}_0^2\left(\mathcal{N}-1+B\beta_{hm}\left(\frac{\mathcal{N}}{\mu_S+\nu_m}+\frac{\mathcal{N}}{\mu_I}\frac{\nu_m}{\nu_m+\mu_S}-\frac{1}{\mu_S}\right)\frac{I_h^\sharp}{N_h}\right)\left(1-\left(\frac{\eta_h+\mu_h}{\mu_h}\right)\frac{I_h^\sharp}{N_h}\right)= (\mathcal{N}-1)\left(1+\frac{B\beta_{hm}}{\mu_S}\frac{I_h^\sharp}{N_h}\right)^2. \tag{C.9}$$

Setting $x=\frac{I_h^\sharp}{N_h}$, we derive

$$ax^2+bx+c=0, \tag{C.10}$$

with

$$a=\mathcal{R}_0^2B\beta_{hm}\left(\frac{\mathcal{N}}{\mu_I}\frac{\nu_m}{\nu_m+\mu_S}+\frac{\mathcal{N}}{\mu_S+\nu_m}-\frac{1}{\mu_S}\right)\left(\frac{\eta_h+\mu_h}{\mu_h}\right)+(\mathcal{N}-1)\left(\frac{B\beta_{hm}}{\mu_S}\right)^2,$$

$$b=\mathcal{R}_0^2\,(\mathcal{N}-1)\left(1+\frac{\eta_h}{\mu_h}\right)+2\,(\mathcal{N}-1)\,\frac{B\beta_{hm}}{\mu_S}-\mathcal{R}_0^2B\beta_{hm}\left(\frac{\mathcal{N}}{\mu_I}\frac{\nu_m}{\nu_m+\mu_S}+\frac{\mathcal{N}}{\mu_S+\nu_m}-\frac{1}{\mu_S}\right),$$

and

$$c=-\,(\mathcal{N}-1)\,(\mathcal{R}_0^2-1)$$

such that, $\mathcal{N}$ being large (larger than μ_I/μ_S), we have $\left(\frac{\mathcal{N}}{\mu_I}\frac{\nu_m}{\nu_m+\mu_S}+\frac{\mathcal{N}}{\mu_S+\nu_m}-\frac{1}{\mu_S}\right)\geq 0$, $A^\sharp>0$ (see (C.7)), $a>0$, and $c<0$ when $\mathcal{R}_0>1$, (C.10) has one positive root

$$x^\sharp=\frac{1}{2a}\left(-b+\sqrt{b^2-4ac}\right)>0.$$

Thus, we deduce $I_h^\sharp$, and, then, all other variables can be deduced. Moreover, the application of [10, Theorem 4.1], to prove item 2 of Theorem 3, page 11, also establishes the local asymptotic stability of the unique endemic or positive equilibrium for system (15)-(16) when $\mathcal{R}_0^2>1$.

Appendix D. Proof of Theorem 7

Eigenvalues of the monodromy matrix of system (18)-(39)-(40) at $TDFS_{per}$ are

$$\begin{array}{rcl}\lambda_1 &=& \lambda_2 = e^{-\mu_h \tau},\\ \lambda_3 &=& \lambda_4 = e^{-\mu_S \tau},\\ \lambda_5 &=& e^{-\mu_M \tau},\ \lambda_6 = e^{-(\mu_S+\nu_m)\tau},\\ \lambda_7 &=& e^{-\mu_I \tau},\ \lambda_8 = e^{-(\gamma+\mu_{A,1})\tau}\end{array}$$

and the eigenvalues of the 3×3 sub-matrix Z defined as:

$$Z = (z_{ij})_{1\leq i,j\leq 3} = \begin{pmatrix} -(\eta_h+\mu_h)\tau & 0 & B\beta_{mh}\tau \\ \dfrac{B\beta_{hm}}{N_h}\displaystyle\int_0^\tau S_S(x)dx & -(\nu_m+\mu_S)\tau & 0 \\ 0 & \nu_m\tau & -\mu_I\tau \end{pmatrix}.$$

Moreover, let us set:

$$\begin{array}{rcl} C_1 &=& -z_{11}-z_{22}-z_{33} > 0,\\ C_2 &=& -z_{11}z_{22}z_{33} - z_{21}z_{32}z_{13},\\ C_3 &=& z_{11}z_{33}+z_{11}z_{22}+z_{22}z_{33} - \dfrac{C_2}{C_1}.\end{array}$$

Hence, according to the Routh-Hurwitz theorem, eigenvalues of the matrix Z have negative real parts if and only if $C_1 > 0$ and $C_2 > 0$ and $C_3 > 0$.

$$\begin{array}{rcl} C_2 &=& -z_{11}z_{22}z_{33} - z_{21}z_{32}z_{13},\\ &=& \tau^3(\eta_h+\mu_h)(\nu_m+\mu_S)\mu_I\left(1 - \dfrac{\nu_m}{\nu_m+\mu_S}\dfrac{B^2\beta_{hm}\beta_{mh}}{(\eta_h+\mu_h)\mu_I N_h}\dfrac{1}{\tau}\displaystyle\int_0^\tau S_S(x)dx\right)\\ &=& \tau^3(\eta_h+\mu_h)(\nu_m+\mu_S)\mu_I\left(1-\mathcal{T}_{0,pulse}\right).\end{array}$$

Hence, $C_2 > 0$ because $\mathcal{T}_{0,pulse} < 1$. Moreover,

$$\begin{array}{rcl} C_3 &=& z_{11}z_{33}+z_{11}z_{22}+z_{22}z_{33} - \dfrac{C_2}{C_1},\\ &=& \dfrac{1}{C_1}(C_1(z_{11}z_{33}+z_{11}z_{22}) - z_{22}z_{33}(z_{22}+z_{33}) + z_{21}z_{32}z_{13}),\\ &>& 0.\end{array}$$

Therefore, if $\mathcal{T}_{0,pulse} < 1$, then $s(Z) < 0$, where $s(\cdot)$ denotes the stability modulus (i.e., the maximum of the real part of eigenvalues). Thus, eigenvalues of the matrix Z have negative real part and the exponential of their real are therefore strictly less than one. Consequently, the trivial periodic disease-free solution is locally asymptotically stable whenever $\mathcal{T}_{0,pulse} < 1$. Similarly, if $\mathcal{T}_{0,pulse} > 1$, then $s(Z) > 0$ and the trivial periodic disease-free solution is unstable. Moreover, solutions $(A, M, F_S, F_E, F_I)^T$ of system (39) satisfy:

$$\left\{\begin{array}{rcl} \dfrac{dA}{dt} &=& \phi(F_S+F_E+F_I) - (\gamma+\mu_{A,1}+\mu_{A,2}A)A,\\ \dfrac{dM}{dt} &=& (1-r)\gamma A - \mu_M M,\\ \dfrac{d(F_S+F_E+F_I)}{dt} &=& \dfrac{M}{M+M_T^{per}}r\gamma A - \mu_S(F_S+F_E) - \mu_I F_I,\\ &\leq& \dfrac{M}{M+M_T^{per}}r\gamma A - \mu_S(F_S+F_E+F_I).\end{array}\right. \tag{D.1}$$

The auxiliary system (D.2) is a non-autonomous monotone non-decreasing system:

$$\left\{\begin{array}{rcl} \dfrac{dA}{dt} & = & \phi(F_S+F_E+F_I)-(\gamma+\mu_{A,1}+\mu_{A,2}A)A, \\ \dfrac{dM}{dt} & = & (1-r)\gamma A-\mu_M M, \\ \dfrac{d(F_S+F_E+F_I)}{dt} & = & \dfrac{M}{M+M_T^{per}}r\gamma A-\mu_S(F_S+F_E+F_I). \end{array}\right. \tag{D.2}$$

Moreover, substituting M_T^{per} by $\underline{M}_T$ in system (D.2) leads to the following constant SIT model

$$\left\{\begin{array}{rcl} \dfrac{dA}{dt} & = & \phi(F_S+F_E+F_I)-(\gamma+\mu_{A,1}+\mu_{A,2}A)A, \\ \dfrac{dM}{dt} & = & (1-r)\gamma A-\mu_M M, \\ \dfrac{d(F_S+F_E+F_I)}{dt} & = & \dfrac{M}{M+\underline{M}_T}r\gamma A-\mu_S(F_S+F_E+F_I) \end{array}\right. \tag{D.3}$$

whose solution X_M is such that $X_M \geq X_{per}$ for all time $t>0$, using a comparison principle with X_{per} being the solution of (D.2). Therefore, we deduce from Theorem 2, page 8 that:

- For $\underline{M}_T > M_{T_1}$ or equivalently, $\tau\Lambda_M > M_{T,per}^{crit}$, $\mathbf{0}_{\mathbb{R}^3}$ is globally asymptotically stable for system (D.3). Hence, for system (D.1), one has $\lim\limits_{t\to+\infty}(A,M,F_S,,F_E,F_I)^T(t)=\mathbf{0}_{\mathbb{R}^5}$. Since $\mathcal{T}_{0,pulse}<1$, the trivial periodic disease-free solution TDFS_{per} is locally asymptotically stable for system (18)-(39)-(40). The LAS property means that there exists a neighborhood $\mathcal{V}$ of TDFS_{per} in $\mathbb{R}_+^{11}$ such that if at a time $t^*\geq 0$,

  $$X_{per}^*=(S(t^*),I(t^*),R(t^*),A(t^*),M(t^*),F_S(t^*),F_E(t^*),F_I(t^*),S_S(t^*),S_E(t^*),S_I(t^*))^T\in\mathcal{V},$$

  then for $t\geq t^*$, one has

  $$\lim_{t\to+\infty}(S(t),I(t),R(t),A(t),M(t),F_S(t),F_E(t),F_I(t),S_S(t),S_E(t),S_I(t))^T=TDFS_{per}. \tag{D.4}$$

  It therefore follows from (D.4) that

  $$X_{per}^*\in\mathcal{V}\implies\lim_{t\to+\infty}(S(t),I(t),R(t))^T=(N_h,0,0)^T.$$

  However, it is straightforward to obtain from system (18) that

  $$\forall t>0,\quad (S+I+R)(t)=N_h$$

  which implies that

  $$\lim_{t\to+\infty}(S+I+R)(t)=N_h. \tag{D.5}$$

  We deduce from (D.5) that for all neighborhood $\mathcal{V}_0$ of N_h in $\mathbb{R}_+$, there exists a time $t^\dagger\geq 0$ such that

  $$(S+I+R)(t^\dagger)\in\mathcal{V}_0\implies\lim_{t\to+\infty}(S+I+R)(t)=N_h. \tag{D.6}$$

  In particular for $\mathcal{V}_0=\mathcal{V}$, (D.6) holds true. By the unicity of the limit, we deduce that

  $$\text{for}\quad (S,I,R)^T(0)\in\mathbb{R}_+^3,\quad \lim_{t\to+\infty}(S(t),I(t),R(t))^T=(N_h,0,0)^T. \tag{D.7}$$

Hence, the limit system of $(S_S, S_E, S_I)^T$ is

$$\left\{\begin{array}{rcl} \dfrac{dS_S}{dt} &=& -\mu_S S_S, \\ \dfrac{dS_E}{dt} &=& -(\nu_m+\mu_S)S_E, \\ \dfrac{dS_I}{dt} &=& \nu_m S_E - \mu_I S_I, \end{array}\right. \tag{D.8}$$

$$\left\{\begin{array}{l} S_S(n\tau_+) = S_S(n\tau) + \tau\Lambda_M \\ S_E(n\tau_+) = S_E(n\tau), \\ S_I(n\tau_+) = S_I(n\tau), \end{array}\right. \tag{D.9}$$

It is therefore straightforward to deduce that

$$\lim_{t\to+\infty}(S_S(t), S_E(t), S_I(t))^T = (S_S^{per}(t), 0, 0)^T. \tag{D.10}$$

- Assume that $\tau\Lambda_M = M_{T,per}^{crit}$, and $(A, M, F_S+F_E+F_I)^T(0) \in [\mathbf{0}_{\mathbb{R}^3}, E_\dagger(\underline{M}_T))$. Therefore, we deduce from Theorem 2, page 8, that $\lim_{t\to+\infty}(A, M, F_S, F_E, F_I)^T(t) = \mathbf{0}_{\mathbb{R}^5}$. Therefore, we proceed in the same way as in (D.4)-(D.10).
- Finally, assume that $0 < \tau\Lambda_M < M_{T,per}^{crit}$, and $(A, M, F_S+F_E+F_I)^T(0) \in [\mathbf{0}_{\mathbb{R}^3}, E_1(\underline{M}_T))$. Therefore, we deduce from Theorem 2, page 8, that $\lim_{t\to+\infty}(A, M, F_S, F_E, F_I)^T(t) = \mathbf{0}_{\mathbb{R}^5}$. Hence, the rest of proof is done as in (D.4)-(D.10).

This ends the proof.

Appendix E. Proof of Theorem 8

For system (51), the following result is valid.

Lemma 4. *Assume that $\mathcal{N} > 1$ and $\overline{\Lambda}_M \in (0, \Lambda_M^{crit}]$. Then, system (51) has at least one positive τ-periodic solution $(A(t), M(t), F_S(t))^T$ with $\overline{E}_{1,3D} \le (A(t), M(t), F_S(t))^T \le E^*$ whenever $\overline{E}_{1,3D} \le (A(0), M(0), F_S(0))^T \le E^*$.*

Proof. Assume that assumptions of Lemma 4 are valid. First, we define a shift operator, which is also known as a Poincaré mapping $\sigma : \mathbb{R}^3 \to \mathbb{R}^3$. For $(t_0, (A_0, M_0, F_{S_0})^T)^T \in \mathbb{R}_+ \times \mathbb{R}^3$,

$$\sigma((A_0, M_0, F_{S_0})^T) = (A(\tau, t_0, (A_0, M_0, F_{S_0})^T), M(\tau, t_0, (A_0, M_0, F_{S_0})^T), F_S(\tau, t_0, (A_0, M_0, F_{S_0})^T))^T,$$

where $(A(\tau, t_0, (A_0, M_0, F_{S_0})^T), M(\tau, t_0, (A_0, M_0, F_{S_0})^T), F_S(\tau, t_0, (A_0, M_0, F_{S_0})^T))^T$ denotes the solution of system (51) through the point $(t_0, (A_0, M_0, F_{S_0})^T)^T$. Proposition 4, page 24, tells us that the set

$$\Omega = \{(A, M, F_S)^T \in \mathbb{R}^3_+ : \overline{E}_1 \le (A, M, F_S)^T \le E^*\}$$

is positively invariant by system (51). That is to say, the operator σ defined above maps Ω into itself: $\sigma(\Omega) \subset \Omega$. Since the solution of (51) is continuous with respect to the initial value, the operator σ is continuous. Moreover, Ω is a bounded, closed, convex set in $\mathbb{R}^3$. Hence, by the Brouwer fixed point theorem, see for example [27, Theorem 2.1.11], σ has at least one fixed point in Ω, i.e., there exists at least one positive τ-periodic solution of (51) in Ω. This ends the proof. □

Let us now consider the auxiliary system (E.1):

$$\begin{cases} \dfrac{dS_S}{dt} = v(t) - \mu_S S_S, \\ S_S(n\tau_+) = S_S(n\tau) + \tau\Lambda_F \end{cases} \tag{E.1}$$

where v is a continuous, τ-periodic, positive and bounded real valued function. The following result is valid:

Lemma 5. *System (E.1) admits a unique positive and periodic solution $S_S(t)$ defined by:*

$$S_S(t) = \left(\frac{\tau\Lambda_F + e^{-\mu_S\tau} \displaystyle\int_{\lfloor t/\tau \rfloor \tau}^{(\lfloor t/\tau \rfloor \tau + 1)\tau} v(x) e^{\mu_S x} dx}{1 - e^{-\mu_S \tau}} + \int_{\lfloor t/\tau \rfloor \tau}^{t} v(x) e^{\mu_S x} dx \right) e^{-\mu_S (t - \lfloor t/\tau \rfloor \tau)}. \tag{E.2}$$

Based on Lemma 4, it is straightforward to deduce Lemma 6 and Lemma 7:

Lemma 6. *Assume that $\mathcal{N} > 1$ and $\overline{\Lambda}_M \in (0, \Lambda_M^{crit}]$. Then, for*

$$\overline{E}_{1,3D} \leq (A(0), M(0), F_S(0))^T \leq E^*,$$

system (49)-(50) has at least one positive τ-periodic solution $(S_h(t), A(t), M(t), F_S(t), S_S(t))^T$ with

$$\overline{E}_{1,3D} \leq (A(t), M(t), F_S(t))^T \leq E^*,$$

$S_h(t) = N_h$ and $S_S(t)$ is defined by (E.2) with

$$v(t) = \frac{M_T^{per}(t)}{M(t) + M_T^{per}(t)} r\gamma A(t).$$

Lemma 7. *Under conditions of Lemma 6, system (18)-(39)-(40) has at least one positive τ-periodic solution $DFS_{per} = (S_h(t), 0_{\mathbb{R}^2}, A(t), M(t), F_S(t), 0_{\mathbb{R}^2}, S_S(t), 0_{\mathbb{R}^2})^T$ with $S_h(t)$, $A(t)$, $M(t)$, $F_S(t)$, $S_S(t)$ given in Lemma 6.*

Theorem 8 therefore follows from Lemma 7.