On nonlinear pest/vector control via the Sterile Insect Technique: impact of residual fertility

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Abstract

We consider a minimalist model for the Sterile Insect Technique (SIT), assuming that residual fertility can occur in the sterile male population. Taking into account that we are able to get regular measurements from the biological system along the control duration, such as the size of the wild insect population, we study different control strategies that involve either continuous or periodic impulsive releases. We show that a combination of open-loop control with constant large releases and closed-loop nonlinear control, i.e. when releases are adjusted according to the wild population size estimates, leads to the best strategy in terms both of number of releases and total quantity of sterile males to be released.

Last but not least, we show that SIT can be successful only if the residual fertility is less than a threshold value that depends on the wild population biological parameters. However, even for small values, the residual fertility induces the use of such large releases that SIT alone is not always reasonable from a practical point of view and thus requires to be combined with other control tools. We provide applications against a mosquito species, *Aedes albopictus*, and a fruit fly, *Bactrocera dorsalis*, and discuss the possibility of using SIT when residual fertility among the sterile males, can occur.

Key words: pest control, vector control, sterile insect technique, residual fertility, closed-loop nonlinear control, control failure, impulsive periodic release

1 Introduction

The Sterile Insect Technique (SIT) is a biological control technique with the advantage of targeting the pest that needs to be controlled. The concept of SIT was conceived in the 30s and 40s by three key researchers in the USSR, Tanzania and the United States (see e.g. [8] for further details about the history of SIT). The principle of SIT is very simple: it consists of releasing males that have been sterilized using ionizing radiation; these males will mate with wild females that will not produce viable offspring. However, while conceptually "simple", SIT can be rather difficult to apply in the field as many feasibility steps need to be checked first.

Since the initial field experiments, much progress has been done under the guidance of the IAEA (International Atomic Energy Agency), who is leading or involved in most of the SIT programs around the world. Around thirty SIT "feasibility" programs are currently taking place for mosquitoes. Against agricultural pests, like fruit flies, SIT programs are more advanced, such that in some places (like Spain and Mexico) effective SIT control is being practiced. Research efforts continue in order to improve SIT efficiency and also combination of SIT with other control tools (like Male Annihilation Technique).

We believe that modeling can be an additional and efficient tool within ongoing programs in order to prevent SIT failure, improve field protocols, or test assumptions that could be difficult to verify in real conditions.

In almost all SIT models that have been studied in the last decades, the main assumption is that sterile males are 100% sterile. However, in real applications this is not always true, and partial sterility has been investigated by entomologists as a possible approach to control both pests and vector populations. On one hand, the main drawback with full sterility is that sterile males can lose their fitness, reducing their competitiveness against wild males, such that very large, massive releases are necessary to compensate this weakness, without any warranty of success. On the other hand, releasing partially sterile males can be problematic as it is important to know for which level of residual fertility these releases fail to control a wild population. This might depend on several factors, like the value of the basic offspring number $\mathcal{N}$, a threshold related to the insect population dynamics. The largest $\mathcal{N}$, the more complicated it is to efficiently control the corresponding wild population.

We build an SIT model, taking into account that the sterility induced by irradiation is not necessarily 100%, but can be a bit lower, such that we have a residual fertility ϵ. In general, the irradiation process is made to reach 100% of sterility, for a given dose of irradiation (for instance, 35-40 Gy to sterilize *Aedes albopictus* pupae [13]). However, for some reasons (technical matters as lower dose of irradiation, environmental conditions, or others), full sterility cannot be reached. So it is important to study the impact of partial sterility on the control process. High irradiation doses might affect the competitiveness index, $\gamma \in [0, 1]$, of sterile males compared to wild males, such that we can wonder whether a lower dose, inducing residual fertility, but keeping γ at 1, could be an interesting strategy.

Our work stands within the framework of two SIT feasibility projects that are taking place in La Réunion: one against *Aedes albopictus*, the TIS 2B project, funded by the French Ministry of Health and the European Regional Development Fund (ERDF); the other against a very damaging fruit fly, *Bactrocera dorsalis*, that first appeared in La Réunion three years ago. Latter project, named GEMDOTIS, is funded by the French government, through the EcoPhyto Call. La Réunion is a French tropical island, located in the Indian Ocean, East from Madagascar and South-West from Mauritius island.

The SIT project against *Aedes albopictus* started in 2010 after a huge epidemic of Chikungunya impacted La Réunion in 2005 and in 2006. Dengue fever is also another vector-borne disease that occurs from time to time in that area with more or less virulence (the last huge dengue epidemics in La Réunion occurred in 1977). The dengue vector is *Aedes albopictus* as well. Thus, the regional council and the French authorities decided to foster the development of biological control methods, like SIT. So far, after many years of laboratory and semi-field studies, the SIT project has started to implement local SIT releases to study the behavior and the impact of sterile males in small places. The goal for the next years is to develop release strategies for large and focused areas, and social acceptance of the program by the local people. We believe that modeling can help to choose between different strategies

and also to point out difficulties that could either drive the SIT control to failure or explain failure in field trials.

GEMDOTIS project against *Bactrocera dorsalis* started in 2019. The fruit fly *Bactrocera dorsalis* has been known for a long time (see [20] for an overview on *Bactrocera* species), but only appeared in La Réunion in 2017. Since then it has invaded all crops thanks to its large range of host, that is approximately 560. However, it has some "favorite" hosts, like guava, and mango in particular. This is the reason why, since the arrival of this fly species, the mango production has collapsed. All biological control tools that were developed with success to lower the impact of other fruit flies, like *Ceratitis rosa*, *Ceratitis capitata*, and *Bactrocera zonata*, are completely inefficient against *Bactrocera dorsalis*. SIT is successfully used in Spain and in South Africa against *Ceratitis capitata*. The objective now is to study its feasibility against *Bactrocera dorsalis*, in the context of a tropical island.

In this work we consider the cases of *Aedes albopictus* and *Bactrocera dorsalis* to illustrate our theoretical results.

The outline of the paper is as follows. In Section 2 we briefly recall the population model developed and studied in [3], we build the partial SIT model based on continuous releases and provide conditions to control the wild population. In Section 3 we extend the previous results to impulsive periodic releases, deriving a long term control strategy. In Section 4 we consider feedback from the models to build a closed-loop control both for continuous and periodic releases; we study different cases. Section 5 is devoted to numerical simulations that illustrate our theoretical results. Finally, in section 6, we summarize the main results of this paper and provide future ways to improve or extend this work.

2 The continuous SIT model with residual fertility

We consider the sex-structured model developed in [3], with male M, and female F insects, and M_S, the sterile males. First we assume a continuous release Λ of sterile males. Following [2], we assume residual fertility, i.e. there is a fraction ϵ of sterile males that remain fertile. This gives the dynamics

$$\begin{cases} \dfrac{dM}{dt} = r\rho \dfrac{F(M+\epsilon\gamma M_s)}{M+\gamma M_s} e^{-\beta(M+F)} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho \dfrac{F(M+\epsilon\gamma M_s)}{M+\gamma M_s} e^{-\beta(M+F)} - \mu_F F, \\ \dfrac{dM_S}{dt} = \Lambda - \mu_S M_S. \end{cases} \tag{1}$$

The description of the parameters is given in Table 1 below.

In general, sterilized insects have larger mortality so that

$$\mu_S \geq \mu_M. \tag{2}$$

The residual fertility ϵ, of the sterile males is assumed to satisfy $0 \leq \epsilon < 1$: when $\epsilon = 0$, this means 0 fertility, i.e. full sterility. Similarly we have $0 < \gamma \leq 1$: when $\gamma = 1$, a sterile male is as competitive as a wild male. In general $\gamma < 1$.

Parameter	Description	Unit
r	sex ratio	–
ρ	mean number of viable (that reach the adult stage) eggs by female per day	day^{-1}
μ_M, μ_F	male and female death rates, resp.	day^{-1}
μ_S	sterile male death rate	day^{-1}
β	characteristic of the competition effect per individual	–
γ	competitiveness index of sterile male mosquitoes	–
ϵ	proportion of sterile males that are fertile	–

Table 1: Description of the parameters

Without SIT, i.e. with $M_S \equiv 0$, model (1) becomes

$$\begin{cases} \dfrac{dM}{dt} = r\rho F e^{-\beta(M+F)} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho F e^{-\beta(M+F)} - \mu_F F, \end{cases} \tag{3}$$

and has been studied in [3].

Let us consider the *basic offspring numbers* for the female and male populations,

$$\mathcal{N}_F \coloneqq \frac{(1-r)\,\rho}{\mu_F}, \qquad \mathcal{N}_M \coloneqq \frac{r\rho}{\mu_M}, \tag{4}$$

respectively. Then, the positive equilibrium of (3) is (M_w^*, F_w^*) where

$$M_w^* \coloneqq \frac{\mathcal{N}_M}{\mathcal{N}_F + \mathcal{N}_M}\frac{1}{\beta}\ln \mathcal{N}_F, \qquad F_w^* \coloneqq \frac{\mathcal{N}_F}{\mathcal{N}_F + \mathcal{N}_M}\frac{1}{\beta}\ln \mathcal{N}_F. \tag{5}$$

We recall the following results (see [3, Theorem 1]):

Proposition 1 *The following assertions hold.*

- *If $\mathcal{N}_F \leq 1$, system (3) converges to the trivial equilibrium $\mathbf{0} = (\mathbf{0}, \mathbf{0})$, for any non-negative initial condition.*
- *If $\mathcal{N}_F > 1$, system (3) converges to the unique positive endemic equilibrium (M_w^*, F_w^*) given in* (5)*, for any non-negative initial condition.*

For a matter of viability of the mosquito population in the absence of SIT, it is imposed that

$$\mathcal{N}_F > 1 \quad \text{and} \quad \mathcal{N}_M > 1.$$

Let us first assume that the release Λ of sterile males is constant, such that, at the steady state, the number of sterile insects is

$$M_S^* \coloneqq \frac{\Lambda}{\mu_S}. \tag{6}$$

From a practical point of view, the value M_S^* in (6) can be reached, for instance, with massive constant releases of 2Λ during $t = \dfrac{\ln 2}{\mu_S}$ days. Fixing the size of the sterile population to the value M_S^* given in (6), leads to the following system:

$$\begin{cases} \dfrac{dM}{dt} = r\rho \dfrac{F(M+\epsilon\gamma M_S^*)}{M+\gamma M_S^*} e^{-\beta(M+F)} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho \dfrac{F(M+\epsilon\gamma M_S^*)}{M+\gamma M_S^*} e^{-\beta(M+F)} - \mu_F F. \end{cases} \tag{7}$$

Existence and uniqueness for system (7) follow from standard results.

2.1 Existence of a positive equilibrium of model (7)

Obviously $\mathbf{0} = (0,0)$ is a trivial equilibrium of system (7). In order to find the positive equilibria, let us assume $M > 0$ and $F > 0$, and solve

$$\begin{cases} r\rho \dfrac{F(M+\epsilon\gamma M_S^*)}{M+\gamma M_S^*} e^{-\beta(M+F)} = \mu_M M, \\ (1-r)\rho \dfrac{(M+\epsilon\gamma M_S^*)}{M+\gamma M_S^*} e^{-\beta(M+F)} = \mu_F. \end{cases}$$

We get

$$\frac{(M^*+\epsilon\gamma M_S^*)}{M^*+\gamma M_S^*} e^{-\beta(M^*+F^*)} = \frac{1}{\mathcal{N}_F} \quad \text{and} \quad \frac{F(M^*+\epsilon\gamma M_S^*)}{M^*+\gamma M_S^*} e^{-\beta(M^*+F^*)} = \frac{M^*}{\mathcal{N}_M}, \tag{8}$$

where $\mathcal{N}_F$ and $\mathcal{N}_M$ were introduced in (4), so that it holds

$$\frac{F^*}{M^*} = \frac{\mathcal{N}_F}{\mathcal{N}_M}. \tag{9}$$

Replacing F^* by the latter relation in the first equation in (8) leads to

$$\frac{(M^*+\epsilon\gamma M_S^*)}{M^*+\gamma M_S^*} e^{-\beta\left(1+\frac{\mathcal{N}_F}{\mathcal{N}_M}\right)M^*} = \frac{1}{\mathcal{N}_F},$$

which is equivalent to

$$\mathcal{N}_F\, e^{-\beta\left(1+\frac{\mathcal{N}_F}{\mathcal{N}_M}\right)M^*} = \frac{M^*+\gamma M_S^*}{M^*+\gamma\epsilon M_S^*} = 1 + (1-\epsilon)\frac{\gamma M_S^*}{M^*+\gamma\epsilon M_S^*}.$$

So we aim at finding the roots of the function f given by

$$f(x) := 1 + (1-\epsilon)\frac{a}{x+\epsilon a} - \mathcal{N}_F e^{-cx}, \tag{10}$$

where

$$a := \gamma M_S^*, \qquad c := \beta\left(1+\frac{\mathcal{N}_F}{\mathcal{N}_M}\right).$$

To show the existence of a positive root of f we have to study the variation of f. The case $\epsilon = 0$ has been investigated in [3]. Assume now $\epsilon > 0$. We can use a similar reasoning to the one in [3].

Let us first observe that

$$f(0) = \frac{1}{\epsilon} - \mathcal{N}_F.$$

Thus, we have three cases: $f(0) = 0$, $f(0) < 0$ and $f(0) > 0$:

- When $\epsilon = \mathcal{N}_F^{-1}$, then $f(0) = 0$, then there is only one non negative root, and it is 0.
- When $\mathcal{N}_F^{-1} < \epsilon < 1$, then $f(0) < 0$. In that case, whatever the value of a, f admits only one positive zero. Assuming a, i.e. γM_S^*, very large, we have $f(x) \approx \dfrac{1}{\epsilon} - \mathcal{N}_F e^{-cx}$ which admits a positive root close to $\dfrac{\ln(\epsilon\mathcal{N}_F)}{c}$. It easily follows that $\dfrac{\ln(\epsilon\mathcal{N}_F)}{c}$ is a lower bound for M^*, this is $M^* \geq \dfrac{\ln(\epsilon\mathcal{N}_F)}{c}$. This means that if partial sterility is larger than $\mathcal{N}_F^{-1}$ then, whatever the size of the releases, the wild male population will always be greater than the positive root M^* of f, which leads to a failure of SIT control. Of course, when $\epsilon = 1$, we recover the value M_w^* of the wild equilibrium, as expected.
- When $\epsilon < \mathcal{N}_F^{-1}$, then $f(0) > 0$. Hence, f is first decreasing and then increasing such that we may have none, one or two zeros. In fact, the number of roots might depend on a: for small values of a, two zeros, and for large value of a, no zeros. There exists a^{crit} such that we have only one double root x^{crit} that satisfies

$$f(x^{\text{crit}}) = f'(x^{\text{crit}}) = 0.$$

That is

$$1 + (1-\epsilon)\frac{a^{\text{crit}}}{x^{\text{crit}} + \epsilon a^{\text{crit}}} = \mathcal{N}_F e^{-cx^{\text{crit}}},$$

then

$$(1-\epsilon)a^{\text{crit}}\left(\frac{1}{x^{\text{crit}} + \epsilon a^{\text{crit}}}\right)^2 = \mathcal{N}_F c e^{-cx^{\text{crit}}}.$$

Thus, putting together the two latter equalities leads to

$$(1-\epsilon)a^{\text{crit}}\left(\frac{1}{x^{\text{crit}} + \epsilon a^{\text{crit}}}\right)^2 = c\left(1 + (1-\epsilon)\frac{a^{\text{crit}}}{x^{\text{crit}} + \epsilon a^{\text{crit}}}\right),$$

which has a unique positive root x^{crit} given by

$$x^{\text{crit}} = \frac{2}{c\left(1 + \sqrt{1 + \dfrac{4}{a^{\text{crit}} c\,(1-\epsilon)}}\right)} - \epsilon a^{\text{crit}}.$$

Then, replacing x^{crit} in (2.1), and setting $\Phi_\epsilon \coloneqq (1-\epsilon)\dfrac{a^{\text{crit}} c}{2}$, implies that Φ_ϵ is a positive solution of

$$1 + \Phi_\epsilon\left(1 + \sqrt{1 + \frac{2}{\Phi_\epsilon}}\right) = \mathcal{N}_F e^{-2\frac{\epsilon}{1-\epsilon}\Phi_\epsilon} e^{-\frac{2}{1 + \sqrt{1 + \frac{2}{\Phi_\epsilon}}}}. \tag{11}$$

Summarizing, we get the result below.

Proposition 2 *The following assertions hold.*

(i) If $\epsilon < \mathcal{N}_F^{-1}$, then there exists $\Lambda_{\text{crit}}^\epsilon > 0$ such that system (7) admits

– *two positive distinct equilibria if* $0 < \Lambda < \Lambda^{\epsilon}_{\text{crit}}$,

– *one positive equilibrium if* $\Lambda = \Lambda^{\epsilon}_{\text{crit}}$,

– *no positive equilibria if* $\Lambda > \Lambda^{\epsilon}_{\text{crit}}$.

The value of $\Lambda^{\epsilon}_{\text{crit}}$ *is defined by*

$$\Lambda^{\epsilon}_{\text{crit}} \coloneqq \frac{2}{1-\epsilon}\frac{\mu_S}{\beta\gamma\left(1+\frac{\mathcal{N}_F}{\mathcal{N}_M}\right)}\Phi_\epsilon,$$

where Φ_ϵ *is the unique positive solution to the transcendental equation (11).*

(ii) Assume that $\mathcal{N}_F^{-1} < \epsilon < 1$*, then, for any* $\Lambda > 0$*, i.e. for any* $M_S^* > 0$*, the system (7) admits one positive equilibrium, bounded from below (component-wise) by the point*

$$(M_\ell^*, F_\ell^*) \coloneqq \frac{\ln(\epsilon\mathcal{N}_F)}{\beta(\mathcal{N}_F+\mathcal{N}_M)}\big(\mathcal{N}_M, \mathcal{N}_F\big). \tag{12}$$

Clearly, if partial sterility is too large, then SIT will fail: even very large releases will only have a limited effect on the wild population. In other words it will be impossible to approach **0**. Indeed, according to (ii) in Proposition 2, when the residual fertility is larger than $\mathcal{N}_F^{-1}$, the system will always converge to a positive equilibrium that is bounded below by the point defined in (12), even when the size of the releases is very large. This lower bound (12) can be big depending on the parameters' values. For instance, if we consider $\epsilon = 0.05$ and the values given in Table 2, we derive $(M_\ell^*, F_\ell^*) = (1408, 1220)$, that is a large value.

2.2 Asymptotic analysis of the equilibria

We assume that $\Lambda > \Lambda^{\epsilon}_{\text{crit}}$ such that system (7) possesses only the trivial equilibrium (as established in Proposition 2). We compute the Jacobian related to system (7), that gives $J(M, F)$ equals to

$$\begin{pmatrix} r\rho F\Delta\left(\dfrac{(1-\epsilon)\gamma M_S^*}{M+\gamma M_S^*} - \beta(M+\epsilon\gamma M_S^*)\right) - \mu_M & r\rho(M+\epsilon\gamma M_S^*)(1-\beta F)\Delta \\ (1-r)\rho F\Delta\left(\dfrac{(1-\epsilon)\gamma M_S^*}{M+\gamma M_S^*} - \beta(M+\epsilon\gamma M_S^*)\right) & (1-r)\rho(M+\epsilon\gamma M_S^*)(1-\beta F)\Delta - \mu_F \end{pmatrix},$$

where $\Delta \coloneqq \dfrac{e^{-\beta(M+F)}}{M+\gamma M_S^*}$. Computing J at **0** gives

$$J(0,0) = \begin{pmatrix} -\mu_M & r\rho\epsilon \\ 0 & (1-r)\rho\epsilon - \mu_F \end{pmatrix}.$$

Thus, if $\epsilon < \mathcal{N}_F^{-1}$, then **0** is Locally Asymptotically Stable (LAS). Otherwise it is unstable. The condition $\epsilon < \mathcal{N}_F^{-1}$ is also necessary in order to guarantee that the population can be controlled and become as small as desired in a finite "short" time.

To show that **0** is Globally Asymptotically Stable (GAS), like in [3], we use the Dulac criterion with the following Dulac function:

$$\psi(M, F) \coloneqq \frac{M+\gamma M_S^*}{F(M+\epsilon\gamma M_S^*)}.$$

We compute

$$\frac{\partial}{\partial M}\left(r\rho e^{-\beta(M+F)} - \mu_M \frac{M+\gamma M_S^*}{F(M+\epsilon\gamma M_S^*)}M\right) = \\ - r\rho\beta e^{-\beta(M+F)} - \frac{\mu_M}{F}\left(\frac{\epsilon\gamma M_S^*(2M+\gamma M_S^*)+M^2}{(M+\epsilon\gamma M_S^*)}\right) < 0,$$

and

$$\frac{\partial}{\partial F}\left((1-r)\rho e^{-\beta(M+F)} - \mu_F \frac{M+\gamma M_S^*}{(M+\epsilon\gamma M_S^*)}\right) = -(1-r)\beta\rho e^{-\beta(M+F)} < 0.$$

Thus, according to Poincaré-Bendixson Theorem, since $\mathbf{0}$ is the only LAS equilibrium when $\epsilon < \mathcal{N}_F^{-1}$ and the system has no closed orbits, we deduce that $\mathbf{0}$ is also GAS.

Assume now that $0 < \Lambda < \Lambda_{\text{crit}}^{\epsilon}$. Then, according to Proposition 2, there exists two positive equilibria, that we call $\mathbf{E_1^*}$ and $\mathbf{E_2^*}$, with $\mathbf{E_1^*}$ unstable and $\mathbf{E_2^*}$ LAS. Like for the case $\epsilon = 0$ (see [3]), we have bistability, and the basin of attraction of $\mathbf{0}$ contains the interval

$$[\mathbf{0}, \mathbf{E_1}^*[:= \{(M,F) \in \mathbb{R}_+^2 : 0 \le M < M_1^*, 0 \le F < F_1^*\},$$

and the basin of attraction of $\mathbf{E_2}^*$ contains the interval

$$]\mathbf{E_1^*}, +\infty[:= \{(M,F) \in \mathbb{R}_+^2 : M_1^* < M, F_1^* < F\}.$$

3 Impulsive periodic releases

To achieve practicable strategies, we consider impulsive periodic releases, since the releases in the field are done instantaneously and periodically. Hence M_S follows the dynamics

$$\begin{cases} \dfrac{dM_S}{dt} = -\mu_S M_S, \quad \text{for } t \neq n\tau, \\ M_S(t_c + n\tau^+) = M_S(t_c + n\tau) + \tau\Lambda, \qquad n \in \mathbb{N}, \end{cases} \tag{13}$$

where t_c is the time at which the control starts. We assume that releases are done every $\tau > 0$ days, such that the number of sterile males asymptotically approaches the function $M_{s,\text{per}}$ given by (see [3]):

$$M_{s,\text{per}}(t) := \frac{\tau\Lambda}{1-e^{-\mu_S\tau}} e^{-\mu_S\left(t - \left\lfloor \frac{t}{\tau} \right\rfloor \tau\right)}.$$

Thus we derive the following system with periodic coefficients

$$\begin{cases} \dfrac{dM}{dt} = r\rho \dfrac{F(M+\epsilon\gamma M_{s,\text{per}})}{M+\gamma M_{s,\text{per}}} e^{-\beta(M+F)} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho \dfrac{F(M+\epsilon\gamma M_{s,\text{per}})}{M+\gamma M_{s,\text{per}}} e^{-\beta(M+F)} - \mu_F F. \end{cases} \tag{14}$$

The pest/vector free equilibrium is still an equilibrium of system (14). Like in the previous part, the objective is to find conditions under which the equilibrium $\mathbf{0}$ is GAS for system (14). From (14), we have

$$\frac{dF}{dt} = \left((1-r)\rho \frac{M+\epsilon\gamma M_{s,\text{per}}}{M+\gamma M_{s,\text{per}}} e^{-\beta(M+F)} - \mu_F\right) F. \tag{15}$$

Thus,

$$\frac{M+\epsilon\gamma M_{s,\mathrm{per}}}{M+\gamma M_{s,\mathrm{per}}}e^{-\beta(M+F)}=\left(\frac{(1-\epsilon)M}{M+\gamma M_{s,\mathrm{per}}}+\epsilon\right)e^{-\beta(M+F)}\leq\frac{(1-\epsilon)\alpha}{\gamma M_{s,\mathrm{per}}}+\epsilon,$$

where $\alpha \coloneqq \max\{xe^{-\beta x} : x \geq 0\} = \dfrac{1}{e\beta}$ (see [3]). Then, integrating (15) between $n\tau$ and $t \geq n\tau$, we derive

$$F(t)\leq F(n\tau)\exp\int_{n\tau}^{t}\left((1-r)\rho\left(\frac{(1-\epsilon)\alpha}{\gamma M_{s,\mathrm{per}}}+\epsilon\right)-\mu_F\right)ds.$$

Taking $t=(n+1)\tau$, we deduce that

$$F((n+1)\tau)\leq F(n\tau)\exp\left((1-r)\rho\tau\left[\frac{(1-\epsilon)\alpha}{\gamma}\left\langle\frac{1}{M_{s,\mathrm{per}}}\right\rangle-\left(\mathcal{N}_F^{-1}-\epsilon\right)\right]\right),$$

where $\left\langle\dfrac{1}{M_{s,\mathrm{per}}}\right\rangle \coloneqq \dfrac{1}{\tau}\displaystyle\int_0^\tau \frac{1}{M_{s,\mathrm{per}}(t)}dt$. Therefore, since $\epsilon < \mathcal{N}_F^{-1}$, the sequence $\{F(n\tau)\}_{n\in\mathbb{N}}$ decreases towards 0, if

$$\frac{(1-\epsilon)\alpha}{\gamma}\left\langle\frac{1}{M_{s,\mathrm{per}}}\right\rangle-\left(\mathcal{N}_F^{-1}-\epsilon\right)<0,$$

that is

$$\frac{2\left(\cosh\left(\mu_S\tau\right)-1\right)}{\mu_S\tau^2\Lambda}<\frac{\gamma}{(1-\epsilon)\alpha}\left(\mathcal{N}_F^{-1}-\epsilon\right), \tag{16}$$

since $\left\langle\dfrac{1}{M_{s,\mathrm{per}}}\right\rangle = \dfrac{2\left(\cosh\left(\mu_S\tau\right)-1\right)}{\mu_S\tau^2\Lambda}$ (see [3]). Inequality (16) holds if

$$\Lambda>\Lambda_{\mathrm{crit}}^{\mathrm{per}} \coloneqq \frac{2\left(\cosh\left(\mu_S\tau\right)-1\right)}{\mu_S\tau^2}\frac{(1-\epsilon)\mathcal{N}_F}{\gamma\left(1-\epsilon\mathcal{N}_F\right)e\beta} \tag{17}$$

This is sufficient to ensure that F converges towards 0, which induces the same behavior for M. Thus, condition (17) implies that $\mathbf{0}$ is also GAS. We derive the following result.

Theorem 1 *For any given $\tau > 0$, assuming that Λ is chosen such that (17) is verified, every solution of system (14) converges to $\mathbf{0}$.*

Thus, using Theorem 1, massive releases, i.e. $\tau\Lambda = k \times \tau\left(\lfloor\Lambda_{\mathrm{crit}}^{\mathrm{per}}\rfloor + 1\right)$ with $k \geq 1$, guarantee that the system will be driven close to zero in finite time. However, once the control stops, the system will recover and the population will reach its initial (positive) equilibrium. Also, for real applications, massive releases are not sustainable and can only be conducted for a limited time. Once the system is close to zero, small releases would be preferable in order to maintain the wild population at a low level (which can be determined evaluating the epidemiological risk and/or an economical threshold value). We follow the same strategy developed in [1, 19].

3.1 Long term control strategy for periodic releases

System (14) can be bounded from above by the following system

$$\begin{cases} \dfrac{dM}{dt} = r\rho \dfrac{F(M+\epsilon\gamma M_{s,l})}{M+\gamma M_{s,l}} e^{-\beta F} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho \dfrac{F(M+\epsilon\gamma M_{s,l})}{M+\gamma M_{s,l}} e^{-\beta F} - \mu_F F, \end{cases} \tag{18}$$

where $M_{s,l} > 0$ is a lower bound of $M_{s,\mathrm{per}}(t)$ given by:

$$M_{s,l} \coloneqq \frac{\tau\Lambda}{1-e^{-\mu_S \tau}} e^{-\mu_S \tau}.$$

In fact it is easy to check that system (18) is a monotone cooperative system within the subset $\mathcal{S} \coloneqq \left\{ (F,M) \in \mathbb{R}_+^2 : F < \dfrac{1}{\beta} \right\}$. Hence, once the solution of the periodic system (14), after several "massive" releases, enters $\mathcal{S}$, we can use the fact that, for a given (small) release $M_{S,\mathrm{obj}}$, the equilibria $\mathbf{0}$, $\mathbf{E_1}$, and $\mathbf{E_2}$, of system (18), are ordered, i.e. $\mathbf{0} < \mathbf{E_1} < \mathbf{E_2}$. In particular, the box $[\mathbf{0}, \mathbf{E_1}[$ is included in the basin of attraction of $\mathbf{0}$.

This last result allows us to deduce a long term control strategy that can be split in two phases: a first initial finite phase with massive releases (where $\mathbf{0}$ is GAS) to enter $[\mathbf{0}, \mathbf{E_1}[$; followed by a second infinite phase, where control is insured by small releases.

The first phase is finite in time, meaning that there exists a time $t^* > 0$, such that for all $t > t^*$, $(M(t), F(t)) \in [\mathbf{0}, \mathbf{E_1}[$. The existence and an upper bound of t^* can be estimated using the same approach employed in [17].

Practically, for a given small release $M_{S,\mathrm{obj}}$, we have to estimate $\mathbf{E_1}$. This is done by finding the zeros of the function f in (10) with

$$x = F, \qquad a = \gamma M_{s,l}, \qquad c = \beta\left(1 + \frac{\mathcal{N}_M}{\mathcal{N}_F}\right).$$

It suffices to estimate the smallest positive root of f to derive F_1, then (analogously to system (7)), we have $M_1 = \dfrac{\mathcal{N}_M}{\mathcal{N}_F} F_1$.

4 Closed-loop control approach

In the previous control approach, for the continuous and periodic cases, we did not consider information on the system along the control duration: the size of the releases was only related to the initial value of the population, at the wild equilibrium. In general, several tools exist that may provide information on the wild population size along the year and during the control, such that it is of interest to take into account this information in order to adapt the size of the releases. This is what is done when using a closed-loop control approach.

Here we let $\kappa\colon [0,+\infty) \to \mathbb{R}_0^+$ be a function such that $M_S(t) = \boldsymbol{\kappa}(t) M(t)$. Then (7) becomes

$$\begin{cases} \dfrac{dM}{dt} = r\rho F \dfrac{(1+\epsilon\gamma\boldsymbol{\kappa})}{1+\gamma\boldsymbol{\kappa}} e^{-\beta(M+F)} - \mu_M M, \\ \dfrac{dF}{dt} = (1-r)\rho \left(\dfrac{1+\epsilon\gamma\boldsymbol{\kappa}}{1+\gamma\boldsymbol{\kappa}} e^{-\beta(M+F)} - \mathcal{N}_F^{-1} \right) F. \end{cases} \tag{19}$$

Let us impose that there exists $\theta > 0$ such that

$$\frac{1+\epsilon\gamma\boldsymbol{\kappa}}{1+\gamma\boldsymbol{\kappa}}e^{-\beta(M+F)} - \mathcal{N}_F^{-1} \leq -\theta, \tag{20}$$

which is equivalent to choosing $\boldsymbol{\kappa}$ such that

$$\boldsymbol{\kappa}(t) \geq \frac{1}{\gamma}\frac{e^{-\beta(M(t)+F(t))} - (\mathcal{N}_F^{-1}-\theta)}{(\mathcal{N}_F^{-1}-\theta) - \epsilon e^{-\beta(M(t)+F(t))}}. \tag{21}$$

Note also that (20) only makes sense if $\theta \leq \mathcal{N}_F^{-1}$. In order to always have positive and finite values in the r.h.s. term of (21), the following condition is needed

$$\epsilon + \theta < \mathcal{N}_F^{-1}.$$

Then, from (19) and (20), we deduce that $\dfrac{dF}{dt} \leq -(1-r)\rho\theta F(t)$ which implies

$$F(t) \leq F(0)e^{-(1-r)\rho\theta t}. \tag{22}$$

This yields that F converges exponentially to 0 when t goes to $+\infty$. Then we deduce that

$$\frac{dM}{dt}(t) \leq r\rho F(0)e^{-(1-r)\rho\theta t}\left(\mathcal{N}_F^{-1}-\theta\right) - \mu_M M.$$

Applying Gronwall's Lemma to latter inequality leads to

$$M(t) \leq M(0)e^{-\mu_M t} + F(0)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M - (1-r)\rho\theta}\left(e^{-(1-r)\rho\theta t} - e^{-\mu_M t}\right), \tag{23}$$

so that M also converges exponentially to 0 when t goes to $+\infty$.

From the previous computations, we deduce the following result.

Proposition 3 (Continuous nonlinear feedback release) *For a given nonnegative $\epsilon < \mathcal{N}_F^{-1}$, let θ be a positive real number such that*

$$0 < \theta + \epsilon < \mathcal{N}_F^{-1}. \tag{24}$$

If M_S is chosen such that

$$M_S(t) \geq \kappa\big(M(t)+F(t)\big)M(t),$$

where

$$\kappa(x) := \frac{1}{\gamma}\frac{e^{-\beta x} - (\mathcal{N}_F^{-1}-\theta)}{(\mathcal{N}_F^{-1}-\theta) - \epsilon e^{-\beta x}}, \tag{25}$$

then every solution of (7) converges exponentially to $\mathbf{0}$.

Remark 1 *The function κ in (25) gives a* nonlinear feedback law *for M_S. The continuous linear feedback control result obtained in [3] can be recovered by considering an upper bound for $\boldsymbol{\kappa}(t)$, obtained when $M+F=0$, and setting $k := \mathcal{N}_F^{-1} - \theta$, that gives*

$$\boldsymbol{\kappa}(t) = \kappa(0) = \frac{1}{\gamma}\frac{1-k}{k-\epsilon} \tag{26}$$

Remark 2 (On the choice of $\boldsymbol{\theta}$) *Note that, in view of (24), one has that $\theta = r_1\mathcal{N}_F^{-1}$ and $\epsilon = r_2\mathcal{N}_F^{-1}$, with $r_1 \in (0,1), r_2 \in [0,1)$ and $r_1 + r_2 < 1$. Simple calculations lead to the following alternative expression for the feedback law κ :*

$$\kappa(x) = \frac{1}{\gamma}\frac{e^{-\beta x}(\mathcal{N}_F^{-1} - r_2)}{1 - r_1 - r_2 e^{-\beta x}} - 1.$$

So that, for a fixed value of ϵ (i.e. of r_2), the gain κ increases w.r.t. to θ (i.e. w.r.t. r_1). The same happens to the speed of convergence of F to 0, that is proportional to θ.
In particular, when ϵ is close to $\mathcal{N}_F^{-1}$, i.e. r_2 close to 1, then θ is close to 0 so that the convergence of F to 0 is slow but, at the same time, the size of the gain κ is small.

4.1 Impulsive releases - synchronized measurements and releases

In the sequel we state sufficient conditions to reach elimination, i.e. convergence towards $\mathbf{0}$, when field measurements and releases are synchronized.

Theorem 2 (Sufficient condition for stabilization by impulsive feedback control) *For a given non negative ϵ and a positive θ such that $\theta + \epsilon < \min\left(1, \frac{\mu_S}{\mu_F}\right)\mathcal{N}_F^{-1}$, assume that for any $n \in \mathbb{N}$*

$$\tau\Lambda_n \;\geq \left| K_{\epsilon,n}\begin{pmatrix} M(n\tau) \\ F(n\tau)\end{pmatrix} - M_S(n\tau)\right|_+,$$

with

$$K_{\epsilon,n} := \frac{1}{\gamma}\frac{e^{-\beta(M_{n+1}+F_{n+1})} - \left(\mathcal{N}_F^{-1} - \theta\right)}{\left(\mathcal{N}_F^{-1} - \theta\right) - \epsilon e^{-\beta(M_{n+1}+F_{n+1})}} e^{(\mu_S - \mu_M)\tau}\left(1, \frac{r\rho(\mathcal{N}_F^{-1} - \theta)}{\mu_M + (1-r)\rho\theta}\left(e^{(\mu_M - (1-r)\rho\theta)\tau} - 1\right)\right),$$

where

$$M_{n+1} \coloneqq M(n\tau)e^{-\mu_M\tau} \qquad \text{and} \qquad F_{n+1} \coloneqq F(n\tau)e^{-\mu_F\tau}.$$

Then, every solution of system (7) converges exponentially towards $\mathbf{0}$, with a convergence rate bounded from below by a value independent of the initial condition.

If, moreover

$$\tau\Lambda_n \leq K_{\epsilon,n}\begin{pmatrix} M(n\tau) \\ F(n\tau)\end{pmatrix},$$

then the series of impulses $\sum_{n=0}^{+\infty}\Lambda_n$ converges.

Proof. See Appendix A, page 23.

Remark 3 *We recover the impulsive linear feedback control of [3] when we replace $K_{\epsilon,n}$ in previous theorem with*

$$\frac{1}{\gamma}\frac{1 - \left(\mathcal{N}_F^{-1} - \theta\right)}{\left(\mathcal{N}_F^{-1} - \theta\right) - \epsilon} e^{(\mu_S - \mu_M)\tau}\left(1, \frac{r\rho}{\mu_M + (1-r)\rho\theta}\left(\frac{1}{\mathcal{N}_F} - \theta\right)\left(e^{(\mu_M - (1-r)\rho\theta)\tau} - 1\right)\right).$$

Theorem 2 above provides a release rate of sterile males that guarantees elimination. Note also that, if the population estimate experiment (using, for instance Mark-Release-Recapture, MRR) provides an estimate of the sterile insects in addition to the wild population, then, the release rate can be chosen very precisely, not necessarily large, and, sometimes, if the sterile population is still large enough, i.e. $K_{\epsilon,n}\begin{pmatrix} M(n\tau) \\ F(n\tau) \end{pmatrix} < M_S(n\tau)$, then release is not necessary.

4.2 Sparse measurements

It is reasonable to expect that measurements of the size of the wild female and male populations are not done very frequently. Having this in mind, we assume in this part that measurements are done every $p\tau$ days, with $p \in \mathbb{N}^*$.

We derive the following result.

Theorem 3 (Stabilization by impulsive control with sparse measurements) *Let $p \in \mathbb{N}^*$, $\epsilon \geq 0$ and $\theta > 0$ such that $\theta + \epsilon < \min\left(1, \frac{\mu_S}{\mu_F}\right)\mathcal{N}_F^{-1}$. Assume that, for any $n \in \mathbb{N}$, $m = 0, 1, \ldots, p-1$,*

$$\tau\Lambda_{np+m} \geq -M_S(np\tau)e^{-m\mu_S\tau} - \tau\sum_{i=0}^{m-1}\Lambda_{np+i}e^{-(m-i)\mu_S\tau} + K_{p,\epsilon}\begin{pmatrix} M(np\tau) \\ F(np\tau) \end{pmatrix},$$

with

$$K_{p,\epsilon} \coloneqq \frac{e^{\mu_S\tau}}{\gamma}\frac{e^{-\beta(M_{\min}^{np+m}+F_{\min}^{np+m})} - \left(\mathcal{N}_F^{-1} - \theta\right)}{\left(\mathcal{N}_F^{-1} - \theta\right) - \epsilon e^{-\beta(M_{\min}^{np+m}+F_{\min}^{np+m})}}V_{p,\epsilon}^T,$$

where

$$M_{\min}^{np+m} := M(np\tau)e^{-m\mu_M\tau}, \qquad F_{\min}^{np+m} := F(np\tau)e^{-m\mu_F\tau}, \tag{27}$$

and

$$V_{p,\epsilon} \coloneqq \begin{pmatrix} e^{-\mu_M m\tau}M(np\tau) \\ \frac{r\rho\left(\mathcal{N}_F^{-1} - \theta\right)}{\mu_M - (1-r)\rho\theta}\left(e^{-(1-r)\rho\theta m\tau} - e^{-\mu_M m\tau}\right)F(np\tau) \end{pmatrix}$$

Then, every solution of system (7) converges exponentially towards $\mathbf{0}$, with a convergence speed bounded from below by a value independent of the initial condition.

If moreover

$$\tau\Lambda_{np+m} \leq K_{p,\epsilon}\begin{pmatrix} M(np\tau) \\ F(np\tau) \end{pmatrix},$$

then the series of impulses $\sum_{n=0}^{+\infty}\Lambda_n$ converges.

Proof. See Appendix B, page 24.

Theorem 3 above generalizes Theorem 2 for the case of sparse measurements. Practically, this is the most useful result because population estimate experiments are, in general, tedious and complicate and, thus, are usually done only from time to time. However, the drawback is that these sparse measurements force us to use more sterile insects than the strategy in Theorem 2. In fact, there is a balance to find between accurate estimates of the releases' sizes and the difficulties (and also the cost) of conducting population estimates in the field. This will depend on the targeted pest/vector species.

4.3 Mixed impulsive control strategies

As done in [3], we can combine the open-loop and the closed-loop (feedback) controls in order to derive strategies that will use fewer sterile males. More precisely, at each release time, we compute the open- and closed-loop controls, and we choose the smaller one. See [3, Section 6] for more details. In the forthcoming simulations we implement these mixed strategies, since they perform better in terms of releases.

5 Numerical simulations

We present several numerical simulations to illustrate our results. In particular, we compare the linear and the nonlinear feedback (closed-loop) control laws, as well as mixed control strategies. For each release strategy we compute the total quantity of sterile insects to be released and the number of necessary releases. These values give an indication of the cost of each intervention. Nevertheless, in order to make a satisfactory analysis of the cost, it would be necessary to make a comparison with other classical control strategies (as the use of adulticide, larvicide, and implementation of mechanical control, for instance). This study, however, is not in the scope of this paper.

5.1 *Aedes Albopictus* parameters

Parameters estimate is based on several publications [5, 7, 13, 10]. In particular, we estimate the characteristic β of the competition effect taking into account the population estimates obtained in [10]: around $6,000$ males during the rainy season and, 600 males during the dry season. According to Table 2, we derive $\mathcal{N}_F \approx 49.95$ and $\mathcal{N}_M \approx 43.29$. The basic offspring

Par.	Value	Description
ρ	0.9*0.74*10=6.66	Number of viable eggs (that reach the adult stage) a female can deposit per day
r	0.5	$r : (1-r)$ expresses the primary sex ratio among offsprings
σ	0.05	Regulates the larvae development into adults under density dependence and larval competition
K	165.21	Carrying capacity in the rainy season
μ_M	1/13	Mean mortality rate of wild adult male mosquitoes
μ_F	1/15	Mean mortality rate of wild adult female mosquitoes
μ_S	1/8.5	Mean mortality rate of sterile adult male mosquitoes
γ	0.91	Competitiveness index of sterile male mosquitoes [11]

Table 2: *Aedes albopictus* parameters values (estimated from [5, 6, 7, 13, 10, 11])

number $\mathcal{N}_F$ is pretty large but realistic in tropical context. According to our previous result, we need to impose $\epsilon < \mathcal{N}_F^{-1}$, then individual fertility in the sterile male population has to be lower than 2%. If not, if for instance $\epsilon \approx 5\%$ then, according to (12), $M_\ell^* = 1,404, F_\ell^* = 1,620$ individuals: this value is reached only for very large releases value, i.e. $\Lambda > 10^{10}$, that are completely unrealistic. Altogether, even with very large releases, the population reduction is only of 76.5% which is not sufficient to reduce the epidemiological risk.

Then, assuming $M^* \approx 6,000$ individuals, we deduce the global competition coefficient $\beta = \dfrac{\sigma}{K} = 3.026 \times 10^{-4}$. Thus, at equilibrium, $E^* = (M^*, F^*)$, the mosquito population verifies $M^* = 6,000$ and $F^* \approx 6,923$ individuals per hectare.

When $\epsilon = 0$, for open-loop periodic impulsive releases carried out every 7 (resp. 14) days, we consider the release value given in (17), page 9, to estimate the minimum of sterile males to release, that is, $\tau\left(\lfloor \Lambda_{\text{crit}}^{\text{per}} \rfloor + 1\right) = 7 \times 8,304 = 58,128$ (resp. $14 \times 9,792 = 137,088$) sterile males per hectare and per week (resp. every two weeks). Note also that for the weekly (every 14 days) release, we approximately release 10 (23) times more sterile males than wild males. In fact, we recover the (minimal) amount of sterile males that is usually recommended by the International Atomic Energy Agency (IAEA).

When $\epsilon = 0.015 > 0$, the open-loop control requires to release at least $\tau\left(\lfloor \Lambda_{\text{crit}}^{\text{per}} \rfloor + 1\right) = 7 \times 32,619 = 228,333$ (resp. $14 \times 38,469 = 538,566$) sterile males per hectare and per week (resp. every two weeks). It is interesting to notice the rise in the release size even with a small residual fertility: we need to release almost 4 times more sterile males. Thus, it is preferable to reduce or avoid the residual fertility all along the experiment if we consider only open-loop control. We will study later the impact on mixed-control strategies.

5.2 *Bactrocera dorsalis* parameters

To estimate the parameters we rely on several publications, like [9, 16, 15, 14, 21]. However, *Bactrocera dorsalis* has a rapid dynamics depending on the type of fruits it develops, such that its basic offspring number can vary from 100 to 500 [9, 15, 14]. From Table 3, we get that $\mathcal{N}_M \approx 251.42$ and $\mathcal{N}_F \approx 232.06$.

Population estimate for *Bactrocera dorsalis* are much more difficult to find in the literature than for mosquitoes. However, in [18] the male population was estimated between $3,300$ and $18,000$. Like for mosquitoes, seasonal variation can also occur. Thus, assuming the male population around $6,000$ individuals per hectare, we can deduce β and then, setting $\sigma = 0.05$, estimate K. We get $\beta \approx 4.7210 \times 10^{-4}$ and $K = 106$.

Parameter	Value	Description
ρ	6.0	Number of viable eggs (that reach the adult stage) a female can deposit per day
r	0.485	$r : (1 - r)$ expresses the primary sex ratio in offspring
σ	0.05	Regulates the larvae development into adults under density dependence and larval competition
K	106	Carrying capacity
μ_M	1/86.4	Mean mortality rate of wild adult male fruit flies
μ_F	1/75.1	Mean mortality rate of wild adult female fruit flies
μ_S	1/86.4	Mean mortality rate of sterile adult fruit flies
γ	0.6	Competitiveness index of sterile male fruit flies

Table 3: *Bactrocera dorsalis* parameters values (estimated from [9]) on Mango; see also [21] for the SIT parameters

The minimal Gamma irradiation dose such that the lifespan of irradiated/treated flies is almost similar to untreated flies is 100 Gy [21]. Also, the 100 Gy treatment seems to be sufficient to induce 100% sterility (see [21, Table 2 page 4]).

Despite the fact that the lifespan of the sterile males is large, weekly massive releases are recommended in real experiments. This is mainly due to the fact that the dynamics of *B. dorsalis* is strong. Thus, for a weekly open-loop release strategy, the number of sterile males to release could be, for instance, $2 \times \tau \left(\lfloor \Lambda_{\text{crit}}^{\text{per}} \rfloor + 1 \right) = 2 \times 7 \times 3,494 = 48,916$. Compared to the mosquito case, and since the basic offspring number is very large, the critical value seems to be low. This is thanks to the lifespan of the sterile male being large, regardless of the bad competitive index.

5.3 Simulations with full sterility, i.e. $\epsilon = 0$

We apply the long term control strategy (introduced in Subsection 3.1) which consists in setting a desired long term release size $M_{S,\text{obj}}$, then computing the corresponding value of the threshold $\mathbf{E_1}$ and performing releases in two stages. A first stage with massive releases (either open- or closed-loop/feedback control, or a combination of both) in order to enter the box $[\mathbf{0}, \mathbf{E_1}[$ and a second *long term* stage of releases of constant size $M_{S,\text{obj}}$.

We first start with 100% sterility and compare the results obtained with linear and nonlinear feedback controls. We consider only periodic releases with two different periods: $\tau = 7$ and $\tau = 14$, and we assume to get estimates of the wild population every $p\tau$ days, for $p = 1$ or $p = 4$. We can consider several choices for $\theta > 0$ as long as $\theta + \epsilon < \min\left(1, \frac{\mu_S}{\mu_F}\right) \mathcal{N}_F^{-1}$, where we take $\epsilon = 0$ in this subsection. We consider two values for θ: $0.99\mathcal{N}_F^{-1}$ and $0.2\mathcal{N}_F^{-1}$. We now provide the time needed to enter the box $[\mathbf{0}, \mathbf{E_1}[$, for each period τ, each p and each choice of θ. While the case $\theta = 0.99\mathcal{N}_F^{-1}$ guarantees a faster convergence of F to zero, this does not necessarily imply the best outcome in terms of released insects necessary to enter the box $[\mathbf{0}, \mathbf{E_1}[$.

5.3.1 The *Aedes albopictus* case

We choose $M_{S,\text{obj}} = 100$, leading to $\mathbf{E_1} = (1.45, 1.67)$ when $\tau = 7$, and $\mathbf{E_1} = (0.44, 0.51)$ when $\tau = 14$. We could choose a larger value for $M_{S,\text{obj}}$, but this is to show the release "effort" that is necessary even to control a very small population.

In Tables 4, 5, 6 and 7 we show some results for a 400-day mixed control. Clearly, the choice of θ has a direct influence on the cumulative number of sterile males and the number of massive releases. When $p > 1$, the best results are obtained with the nonlinear mixed control, simply because the first releases are smaller (compare (a) and (b) or (c) and (d) in Fig. 1, page 18). Overall, and taking into account that sparse measurements occur every 4 weeks, the 7-day release strategy seems to be the most appropriate one: the lowest number of insects to release combined with only 21 "massive" mixed releases (see Fig. 1) to reach the box $[\mathbf{0}, \mathbf{E_1}[$. Note that the nonlinear mixed control needs 33% less sterile males than the linear mixed control.

As expected by Remark 2, page 12, the parameter θ has an impact on the duration of the SIT treatment. However, when $p = 4$ and $\theta = 0.2\mathcal{N}_F^{-1}$, the duration is almost the same for both the linear and nonlinear mixed controls, with a certain gain on the number of sterile males to release with nonlinear mixed control.

Fig. 1 provides a typical output of a mixed-control strategy, mixing open- and closed-loop controls. We show the results for the linear and nonlinear mixed controls : the difference between both approaches occurs in the beginning, where in the linear control a large amount of sterile insects (related to the open-loop control) is released. Figs. 1 (b) and (d) show the

times of releases: as seen, the releases do not occur every τ days, but only if the size of the sterile males is not sufficient to continue to drive the wild population to extinction. This may depend on the periodicity of the releases but also on the vital parameters related to the sterile males. However, with a high mortality rate, $\mu_S = 1/8.5$, even 14-day periodic releases can work, but this requires to release a larger number of sterile males.

p	Period (days)	Cumulative number of released sterile males	Number of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	9.87560×10^5	22
	$\tau = 14$	1.181635×10^6	12
4	$\tau = 7$	1.162560×10^6	20
	$\tau = 14$	1.423067×10^6	11

Table 4: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for **linear** mixed control, when $\theta = 0.99\mathcal{N}_F^{-1}$

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	9.87164×10^5	22
	$\tau = 14$	1.181335×10^6	12
4	$\tau = 7$	1.001690×10^6	18
	$\tau = 14$	1.297330×10^6	11

Table 5: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases and number of releases for **nonlinear** mixed control, when $\theta = 0.99\mathcal{N}_F^{-1}$

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	5.81934×10^5	33
	$\tau = 14$	7.41805×10^5	15
4	$\tau = 7$	9.47466×10^5	19
	$\tau = 14$	1.412932×10^6	13

Table 6: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for **linear** mixed control, when $\theta = 0.2\mathcal{N}_F^{-1}$

As done in [1], we consider that another control methods should be used, like for instance one week of adulticide can be implemented before SIT starts, as recommended by the IAEA. Thus, taking $\tau = 7$ and $p = 4$, we obtain the outcome given in Table 8.

Clearly, comparing Tables 8 and 7, the gain is substantial in terms of sterile males to release (almost 55% less) and also in terms of effective releases (10 less).

5.3.2 The *Bactrocera dorsalis* case

We choose $M_{S,\text{obj}} = 2000$, leading to $\mathbf{E_1} \approx (65.31, 60.28)$ when $\tau = 7$ days, and $\mathbf{E_1} \approx (30.35, 28.01)$ when $\tau = 14$ days. We consider $\theta = 0.3\mathcal{N}_F^{-1} < \min\left(1, \frac{\mu_S}{\mu_F}\right)\mathcal{N}_F^{-1}$, $p = 4$ or

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	5.71501×10^5	34
	$\tau = 14$	7.28663×10^5	15
4	$\tau = 7$	6.91024×10^5	21
	$\tau = 14$	8.98257×10^5	10

Table 7: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for **nonlinear** mixed control, when $\theta = 0.2\mathcal{N}_F^{-1}$

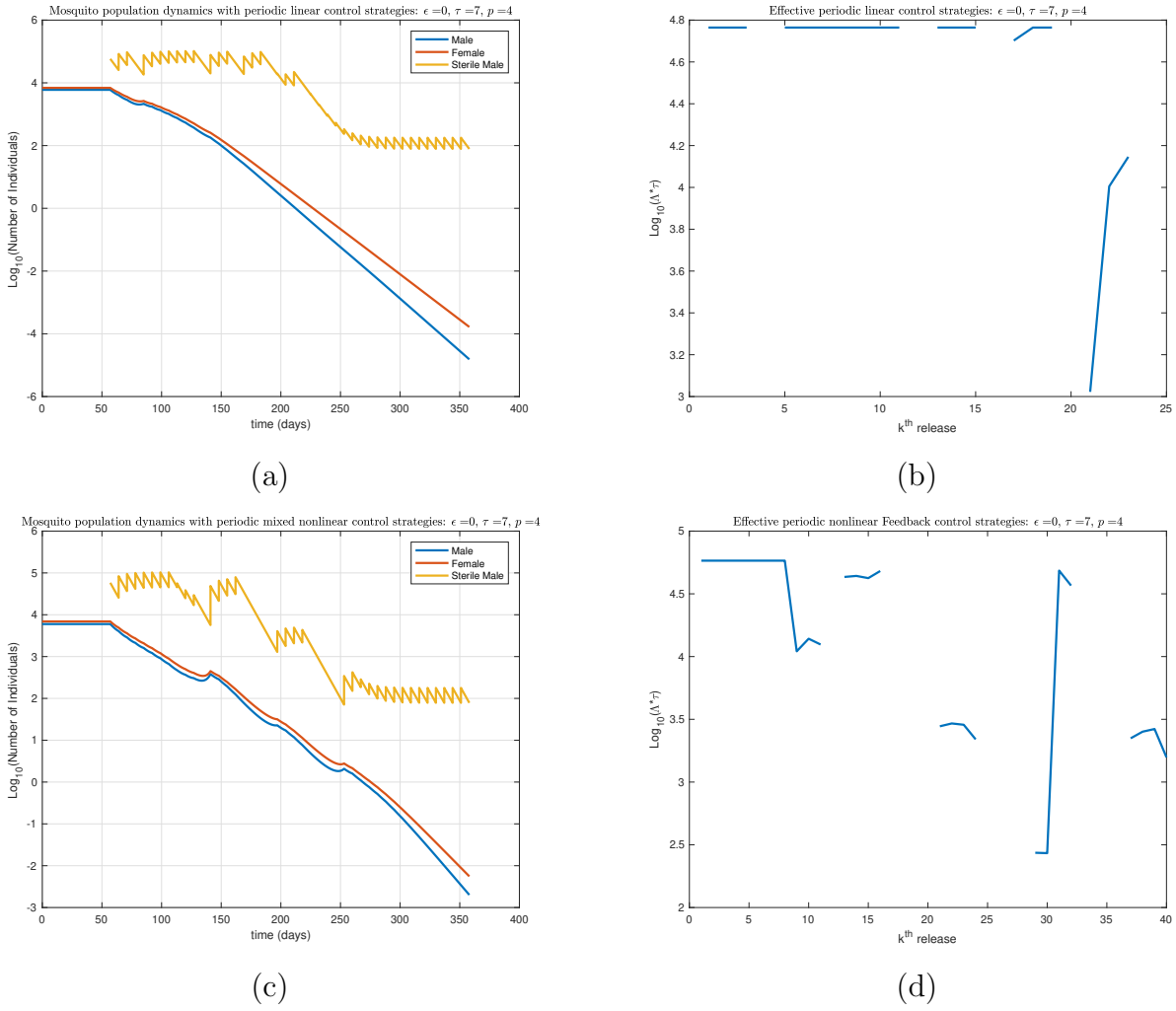

Figure 1: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Mixed periodic impulsive SIT control of system (14) with $\theta = 0.2\mathcal{N}_F^{-1}$, $\tau = 7$ and $p = 4$ - **Linear** control: (a) population dynamics; (b) Field releases timing - **Nonlinear** control: (c) population dynamics; (d) Field releases timing. See Tables 6 and 7, page 17.

$p = 8$. For the open-loop control we assume that we release $2 \times \tau \times \Lambda_{\text{crit}}^{\text{per}}$ sterile males.

The results, given in Tables 9 and 10, are not surprising: in almost all releases, open-loop releases occur. However, in the case $\tau = 14$ and $p = 4$, the mixed-nonlinear control is interesting, with a gain of almost 15% thanks to the mixed-linear control. For a large area,

Mixed-Control	p	Period (days)	Cumulative Number of released sterile males	Nb of effective Releases to reach $[\mathbf{0}, \mathbf{E_1}[$
Linear	4	$\tau = 7$	6.07062×10^5	13
Nonlinear	4	$\tau = 7$	3.12157×10^5	11

Table 8: Full sterility case ($\epsilon = 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for linear or **nonlinear** mixed control, after one week of adulticide, when $\theta = 0.2\mathcal{N}_F^{-1}$

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
4	$\tau = 7$	2.204530×10^6	59
	$\tau = 14$	2.123689×10^6	30
8	$\tau = 7$	2.332524×10^6	47
	$\tau = 14$	2.434298×10^6	25

Table 9: Full sterility case ($\epsilon = 0$) - *Bactrocera dorsalis* - Cumulative number of released sterile males and number of releases for **linear** mixed control, when $\theta = 0.3\mathcal{N}_F^{-1}$

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
4	$\tau = 7$	1.891870×10^6	63
	$\tau = 14$	1.816422×10^6	35
8	$\tau = 7$	1.969639×10^6	48
	$\tau = 14$	2.388379×10^6	25

Table 10: Full sterility case ($\epsilon = 0$) - *Bactrocera dorsalis* - Cumulative number of released sterile males and number of releases for **nonlinear** mixed control, when $\theta = 0.3\mathcal{N}_F^{-1}$

this requires to be able to manufacture billions of sterile males.

These results confirm that control by SIT alone is almost impossible for *Bactrocera dorsalis*. Additional control tools (like female trapping or combination with Methyl-Eugenol [16]) are necessary. Here, as in [1], we consider a one-week treatment with 100% efficiency.

Mixed-Control	p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
Linear	4	$\tau = 14$	1.448964×10^6	23
Nonlinear	4	$\tau = 14$	1.661831×10^6	31

Table 11: Full sterility case ($\epsilon = 0$) - *Bactrocera dorsalis* - Cumulative number of released sterile males and number of releases for **linear** or **nonlinear** mixed control, after one week of adulticide

According to Table 11, there is a clear improvement in the gain of the number of releases and thus in total number of the released sterile males. Also, linear control is better, but in fact this depends on the choice of the open-loop control release.

5.4 The residual fertility case

Since $\mathcal{N}_F$ is very large for *Bactrocera dorsalis*, the residual fertility should be not greater than 0.0043. Assuming that $\epsilon = 0.01$ (i.e. only 1% of residual fertility) then, according to our previous estimate, whatever the size of the releases, the male population can not go down under $\dfrac{\ln(\epsilon\mathcal{N}_F)}{c}$. According to the parameters values, this leads to a minimal population of 928 males per ha and, using relation (9), to a minimal population of 856 females per ha. These lower bounds will not be reached even for very large but still realistic releases.
That is why we will only consider *Aedes albopictus*. We assume a residual fertility of 1.5%, i.e. $\epsilon = 0.015$. We choose $\theta = 0.2\mathcal{N}_F^{-1}$ so that $\theta + \epsilon < \mathcal{N}_F^{-1}$.

In that case, for open-loop periodic impulsive releases carried out every 7 (resp. 14) days, we estimate that we need to release $7 \times 32,619 = 228,333$ ($14 \times 38,469 = 538,566$) sterile males per ha and per week. Compare to the $\epsilon = 0$ case, the amount of insects to release is 4 times larger.

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	5.258893×10^6	74
	$\tau = 14$	6.870483×10^6	42
4	$\tau = 7$	1.2146326×10^7	54
	$\tau = 14$	1.5181271×10^7	32

Table 12: Residual fertility case ($\epsilon > 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for **linear** closed-loop control, when $\theta = 0.2\mathcal{N}_F^{-1}$ and $\epsilon = 0.015$.

p	Period (days)	Cumulative Number of released sterile males	Nb of effective releases to reach $[\mathbf{0}, \mathbf{E_1}[$
1	$\tau = 7$	3.443861×10^6	78
	$\tau = 14$	6.151367×10^6	42
4	$\tau = 7$	4.48830×10^6	58
	$\tau = 14$	6.549222×10^6	42

Table 13: Residual fertility case ($\epsilon > 0$) - *Aedes albopictus* - Cumulative number of released sterile males and number of releases for **nonlinear** closed-loop control, when $\theta = 0.2\mathcal{N}_F^{-1}$ and $\epsilon = 0.015$.

For the residual fertility case, it seems that the best option is the nonlinear control with $\tau = 7$, with a population estimated every $p = 4$ weeks. The gain is almost 50% less releases, even if we have 5 additional releases.

As expected, the induced sterility, even low, increases not only the size of the releases but also the duration of SIT treatment, such that to stay in a realistic time experiment and release sizes, another control methods should be used, for instance one week of adulticide control before SIT starts (see also Fig. 2, page 21). According to Table 14, the gain is significant: around 42% less insects to release than without adulticide treatment. Thus, clearly, without adulticide or equivalent control treatment, releasing sterilized males, even with small residual fertility, is problematic and the risk of failure of the program is high.

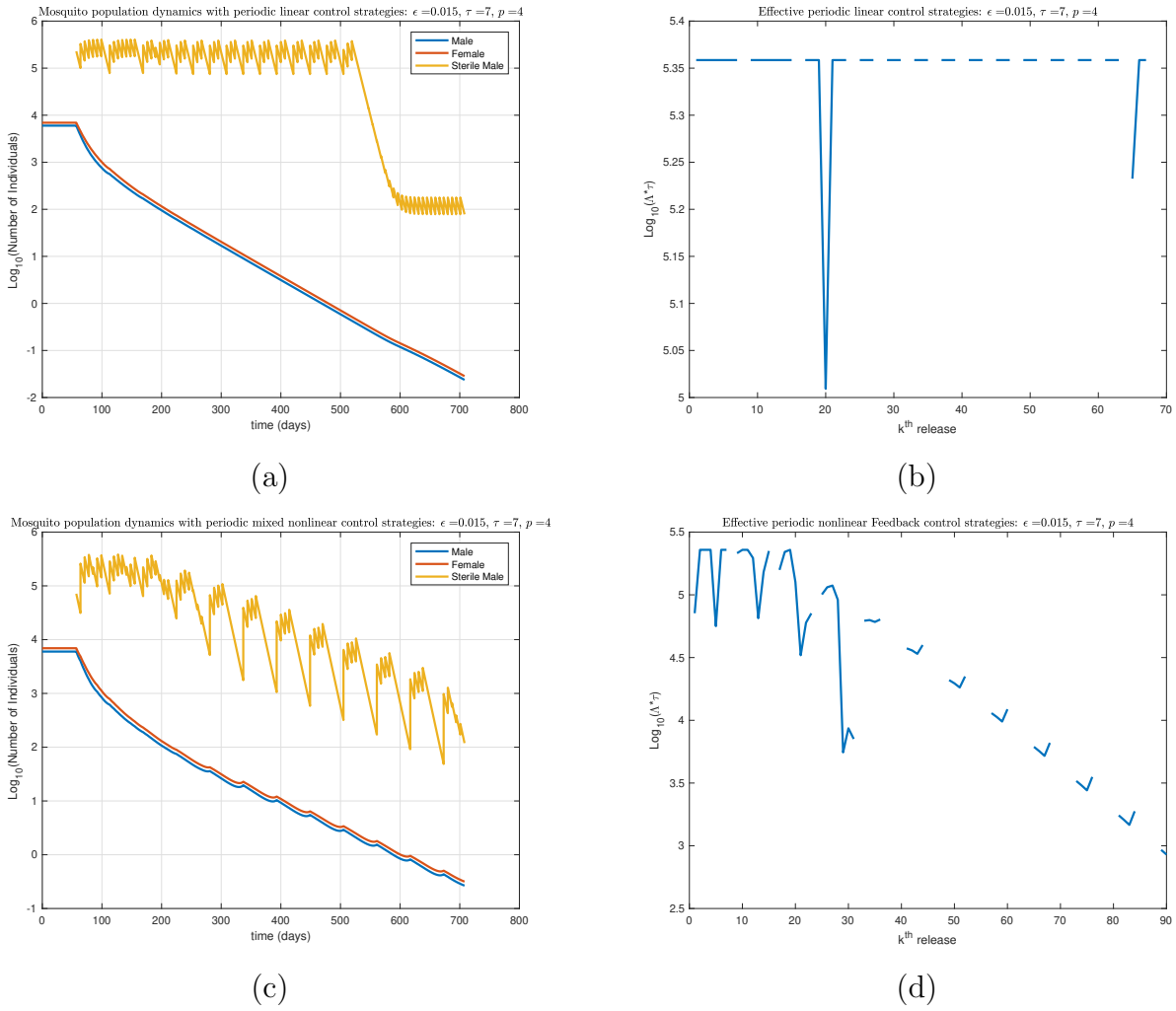

Figure 2: Residual fertility case ($\epsilon > 0$) with adulticide treatment - *Aedes albopictus* - $\epsilon = 0.015$ - Mixed periodic impulsive SIT control of system (14) with $\theta = 0.2\mathcal{N}_F^{-1}$, $\tau = 7$ and $p = 4$ - **Linear** control: (a) population dynamics; (b) Field releases timing - **Nonlinear** control: (c) population dynamics; (d) Field releases timing. See Tables 12 and 13, page 20

Mixed-Control	p	Period (days)	Cumulative Number of released sterile males	Nb of effective Releases to reach $[\mathbf{0}, \mathbf{E_1}[$
Linear	4	$\tau = 7$	1.1012722×10^7	49
Nonlinear	4	$\tau = 7$	2.596800×10^6	47

Table 14: Residual fertility case ($\epsilon > 0$)- *Aedes albopictus* - - Cumulative number of released sterile males and number of releases for **linear** or **nonlinear** mixed control, after one week of adulticide, , when $\theta = 0.2\mathcal{N}_F^{-1}$ and $\epsilon = 0.015$.

Only a combination of controls to reduce the wild population before SIT treatment can be helpful, if the residual fertility is small enough.

6 Conclusion

In this work we have improved the linear feedback control developed in [3] and, in addition, we studied the possibility/risk of releasing partially sterile males, i.e. sterile males with a small, but positive fertility rate ϵ. A control with partial sterility is possible. However, several drawbacks occur: if the fertility is greater than $\mathcal{N}_F^{-1}$, then no control is possible; if the fertility is below $\mathcal{N}_F^{-1}$, then the control needs long time and (very) large releases, with the total number of released sterile males being five times more than the quantity needed in the case of full sterility, i.e. when $\epsilon = 0$ (see Table 5).

Clearly, even if it is showed on a particular model, the condition $\epsilon < \mathcal{N}_F^{-1}$ is needed to guarantee that SIT works under massive releases for almost all SIT models. However, even under that restriction, the size of the releases (or the duration of the control) can be so large that SIT alone becomes unreasonable from a practical point of view. That is why a combination of control tools, including SIT and adulticide, for instance, is needed [16].

Altogether, our results highlight the importance of a very good knowledge of the pest/vector dynamics, i.e. the biological parameters and their sexual behaviors, preferably along the whole year, in order to determine the best period to start the SIT treatment. For pest/vector with a large basic offspring number, when full sterility cannot be achieved, it is clearly recommended to couple SIT with other biological control methods, like mechanical control, (pheromone, food) traps, etc.

Clearly, it seems preferable to release fully sterile males even if there is the cost in terms of fitness. Of course, this requires a sterilization protocol that insures 100% sterility.

The fruit flies case, here *Bactrocera dorsalis*, shows that SIT alone requires huge releases since the dynamics of the pest can be really strong. However, the model we used here does not necessarily reflects the complexity of the fruit flies dynamics, in particular their complex mating behaviors. Thus, precise models and experiments are needed to confirm our results. However, this first insight shows that most probably a combination of control tools would be useful to better control this pest, like for instance, a combination of SIT with a Male Annihilation Technique [12]. The results obtained for *Bactrocera dorsalis* also apply to another fruit fly, *Ceratitis capitata*, that may as well have a very large basic offspring number [4, 14].

Finally, like in [7], where an epidemiological model coupled with an SIT model was studied for the first time within the context of La Réunion, it would be interesting to determine whether, despite the fact that $\epsilon > \mathcal{N}_F^{-1}$, the SIT approach can be helpful to reduce the epidemiological risk, i.e. to stir $\mathcal{R}_0$ below 1 for vector-borne diseases, like chikungunya and dengue fever.

Last but not least, several improvements can be made on the model, like, for instance, taking into account other physiological states. However, it is not sure that the mathematical tractability can be kept: see for instance the complex computations, with an exponential function, done in [17] on a three-state model. The different type of competition (direct and indirect) that might occur within the physiological states could also be better modeled.

Acknowledgments

MSA was supported by the National Council for Scientific and Technological Development (CNPq), by FAPERJ through the *"Jovem Cientista do Nosso Estado"* Program and by the

Getulio Vargas Foundation (FGV, Rio de Janeiro, Brazil) through the *"Projeto de Pesquisa Aplicada"* Program.

YD acknowledges the support of the School of Applied Mathematics of FGV (FGV EMAp) that funded his visit in Rio in 2019. YD is partially supported by the "SIT feasibility project against *Aedes albopictus* in Reunion Island", TIS 2B (2020-2021), jointly funded by the French Ministry of Health and the European Regional Development Fund (ERDF). YD is (partially) supported by the DST/NRF SARChI Chair in Mathematical Models and Methods in Biosciences and Bioengineering at the University of Pretoria (grant 82770). YD is also partially supported by the CeraTIS-Corse project, funded by the call Ecophyto 2019 (project n^o: 19.90.402.001), against *Ceratitis capitata*. This work is done within the framework of the GEMDOTIS project (Ecophyto 2018 funding), that is ongoing in La Réunion. This work was also co-funded by the European Union: Agricultural Fund for Rural Development (EAFRD), by the Conseil Régional de La Réunion, the Conseil Départemental de La Réunion, and by the Centre de Coopération internationale en Recherche Agronomique pour le Développement (CIRAD).

We thank the two reviewers for their useful remarks that helped us improve the manuscript.

7 Appendix A: proof of Theorem 2, page 12

We consider that we release sterile insects with a period of τ. From (22) and (23) we get, for $t \in [n\tau, (n+1)\tau)$,

$$\begin{aligned} F(t) &\le F(n\tau)e^{-(1-r)\rho\theta(t-n\tau)}, \\ M(t) &\le M(n\tau)e^{-\mu_M(t-n\tau)} \\ &\quad + F(n\tau)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M-(1-r)\rho\theta}\left(e^{-(1-r)\rho\theta(t-n\tau)}-e^{-\mu_M(t-n\tau)}\right). \end{aligned}$$

We impose the condition

$$M_S(t) \ge \boldsymbol{\kappa}(t)M(t), \quad t \in [n\tau, (n+1)\tau). \tag{28}$$

This is verified if, for $t \in [n\tau, (n+1)\tau)$,

$$\begin{aligned} M_S(t) \ge \boldsymbol{\kappa}(t)\Big(&M(n\tau)e^{-\mu_M(t-n\tau)} \\ &+F(n\tau)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M-(1-r)\rho\theta}\left(e^{-(1-r)\rho\theta(t-n\tau)}-e^{-\mu_M(t-n\tau)}\right)\Big). \end{aligned}$$

Since κ, introduced in (25), decreases as a function of $M+F$, and M and F remain larger than $M(n\tau)e^{\mu_M(t-n\tau)}$ and $F(n\tau)e^{\mu_F(t-n\tau)}$, respectively, we get that

$$\boldsymbol{\kappa}(t) = \kappa(M(t)+F(t)) \le \kappa\left(M(n\tau)e^{\mu_M(t-n\tau)}+F(n\tau)e^{\mu_F(t-n\tau)}\right) =: \kappa^n_{\max}.$$

Thus, if for $s \in [0,\tau)$, it holds

$$\begin{aligned} M_S(n\tau+s) &= \big(M_S(n\tau)+\tau\Lambda_n\big)e^{-\mu_S s} \ge \\ &\kappa^n_{\max}\Big(M(n\tau)e^{-\mu_M s}+F(n\tau)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M-(1-r)\rho\theta}\left(e^{-(1-r)\rho\theta s}-e^{-\mu_M s}\right)\Big). \end{aligned} \tag{29}$$

then (M, F) converges asymptotically to 0. This last equation is equivalent to

$$\tau\Lambda_n \geq -M_S(n\tau) + \kappa_{\max}^n \Big(M(n\tau) e^{(\mu_S - \mu_M)s} + F(n\tau) \frac{r\rho(\mathcal{N}_F^{-1} - \theta)}{\mu_M - (1-r)\rho\theta} \left(e^{(\mu_S - (1-r)\rho\theta)s} - e^{(\mu_S - \mu_M)s} \right) \Big),$$

Since $\mu_S \geq \mu_M$ and $\theta \leq \mathcal{N}_F^{-1}$, assuming the additional condition $\theta \leq \dfrac{\mu_S}{\mu_F}\mathcal{N}_F^{-1}$, one has that all the coefficients and exponents in the r.h.s. of latter expression are positive, so a stronger inequality is obtained if we take $s = \tau$ for the exponential expressions with positive coefficient and $s = 0$ for the one with negative coefficient. This is, we impose

$$\tau\Lambda_n \geq -M_S(n\tau) + \kappa_{\max}^n e^{(\mu_S - \mu_M)\tau} \Big(M(n\tau) + F(n\tau) \frac{r\rho(\mathcal{N}_F^{-1} - \theta)}{\mu_M - (1-r)\rho\theta} \left(e^{(\mu_M - (1-r)\rho\theta)\tau} - 1 \right) \Big).$$

This ends the proof.

8 Appendix B: proof of Theorem 3, page 13

Like in [3], we need to adapt the proof of previous Theorem 2, given in Appendix 7 above. We have, for $m = 0, \ldots, p-1$, and $s \in [0, \tau)$,

$$\begin{aligned} M_S(s + (np+m)\tau) &= \big(\Lambda_{np+m}\tau + M_S((np+m)\tau) \big) e^{-\mu_S s} \\ &= \Big(\Lambda_{np+m}\tau + \Lambda_{np+m-1}\tau e^{-\mu_S\tau} + \cdots + \Lambda_{np}\tau e^{-m\mu_S\tau} + M_S(np\tau) e^{-m\mu_S\tau} \Big) e^{-\mu_S s}. \end{aligned}$$

We have,

$$\kappa\big((np+m)\tau + s\big) \leq \kappa\big(M_{\min}^{np+m} + F_{\min}^{np+m}\big) =: \kappa_{\max}^{np+m},$$

where $M_{\min}^{np+m}$ and $F_{\min}^{np+m}$ were introduced in (**??**). As done above in (29), we impose

$$\begin{aligned} &\Big(\Lambda_{np+m}\tau + \Lambda_{np+m-1}\tau e^{-\mu_S\tau} + \cdots + \Lambda_{np}\tau e^{-m\mu_S\tau} + M_S(np\tau) e^{-m\mu_S\tau} \Big) e^{-\mu_S s} \\ &\qquad \geq \kappa_{\max}^{np+m} \Big(M(np\tau) e^{-\mu_M(m\tau+s)} \\ &\qquad + F(np\tau) \frac{r\rho(\mathcal{N}_F^{-1} - \theta)}{\mu_M - (1-r)\rho\theta} \left(e^{-(1-r)\rho\theta(m\tau+s)} - e^{-\mu_M(m\tau+s)} \right) \Big). \end{aligned}$$

By multiplying by $e^{\mu_S(m\tau+s)}$ both sides of latter inequality, we get

$$\begin{aligned} &\Lambda_{np+m}\tau e^{\mu_S m\tau} + \Lambda_{np+m-1}\tau e^{\mu_S(m-1)\tau} + \cdots + \Lambda_{np}\tau + M_S(np\tau) \\ &\geq \kappa_{\max}^{np+m} \Big(M(np\tau) e^{(\mu_S - \mu_M)(m\tau+s)} \\ &+ F(np\tau) \frac{r\rho(\mathcal{N}_F^{-1} - \theta)}{\mu_M - (1-r)\rho\theta} \left(e^{(\mu_S - (1-r)\rho\theta)(m\tau+s)} - e^{(\mu_S - \mu_M)(m\tau+s)} \right) \Big). \end{aligned}$$

This inequality gives the strongest condition when $s = \tau$. Thus, we enforce,

$$\begin{aligned}
&\Lambda_{np+m}\tau e^{\mu_S m\tau} + \Lambda_{np+m-1}\tau e^{\mu_S (m-1)\tau} + \cdots + \Lambda_{np}\tau + M_S(np\tau) \\
&\geq \kappa_{\max}^{np+m}\Big(M(np\tau)e^{(\mu_S-\mu_M)(m+1)\tau} \\
&+ F(np\tau)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M-(1-r)\rho\theta}\left(e^{(\mu_S-(1-r)\rho\theta)(m+1)\tau} - e^{(\mu_S-\mu_M)m\tau}\right)\Big).
\end{aligned}$$

We get, for $m = 0, \ldots, p-1$,

$$\begin{aligned}
\Lambda_{np+m}\tau \geq e^{-\mu_S m\tau}\Big[&- \Lambda_{np+m-1}\tau e^{\mu_S (m-1)\tau} - \cdots - \Lambda_{np}\tau - M_S(np\tau) \\
&+ \kappa_{\max}^{np+m}\Big(M(np\tau)e^{(\mu_S-\mu_M)(m+1)\tau} \\
&+ F(np\tau)\frac{r\rho(\mathcal{N}_F^{-1}-\theta)}{\mu_M-(1-r)\rho\theta}\left(e^{(\mu_S-(1-r)\rho\theta)(m+1)\tau)} - e^{(\mu_S-\mu_M)m\tau}\right)\Big)\Big].
\end{aligned}$$

The result follows.

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