

Global stability and optimisation of a general impulsive biological control model

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Abstract

An impulsive model of augmentative biological control consisting of a general continuous predator-prey model in ordinary differential equations, *i.e.* a meta-model, augmented by a discrete part describing periodic introductions of predators is considered. The existence of an invariant periodic solution that corresponds to prey eradication is shown and a condition ensuring its global asymptotic stability is given. An optimisation problem related to the preemptive use of augmentative biological control is then considered. It is assumed that the per time unit budget of biological control (*i.e.* the number of predators to be released) is fixed and the best deployment of this budget is sought in terms of release frequency. The cost function to be minimised is the time needed to reduce an unforeseen prey (pest) invasion occurring at a worst time instant under some harmless level. The analysis shows that the optimisation problem admits a countable infinite number of solutions. An argumentation considering the required robustness of the optimisation result with respect to the invasive prey population level and to the model parameters is then conducted. It is shown that the cost function is decreasing in the predator release frequency so that the best deployment of the biocontrol agents is to carry out as frequent introductions as possible.

Key words: Predator-prey dynamics, impulsive model, global stability, optimisation

1. Introduction

Biological control aims to reduce the populations of harmful organisms by utilising their natural enemies, the biological control agents. It can be used to control vectors in vector-borne diseases like malaria or invasive species (may they be animals or plants), but it is mostly dedicated to the control of insect pests, especially at the agricultural cropping system scale [30]. Four main biological control strategies can be identified (see *e.g.* [10, 36] and references therein):

- conservation biological control: biocontrol agents are already present in the system and are favoured through various means (habitat management, cropping practices...).
- classical biological control: exotic biocontrol agents are introduced once in the system and expected to establish and persist indefinitely to provide long term pest control.

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- inoculation biological control: biocontrol agents are introduced (once or repeatedly) and are expected to reproduce to provide pest control for an extended period, but not permanently.
- inundative biological control: biocontrol agents are repeatedly introduced in order to eradicate the pest population. Pest control is expected to be achieved by the released organisms themselves and not by their potential progeny.

As noted by Eilenberg *et al.* [10], it might be difficult to distinguish between repeated inoculation biological control and inundative biological control. Indeed, biocontrol agents used in an inundative program are very likely to reproduce and the distinction between the “inundative” effects and the “inoculative” effects may not be that clear. Hence, “augmentative” biological control is commonly used to denominate as well repeated inoculation biocontrol or inundative biocontrol, though it is not the perfect denomination either [10].

In this contribution we focus on this “augmentative” method, which is fairly well modelled by an impulsive system of ordinary differential equations (a continuous system affected by sudden state modifications at some discrete time moments). The intrinsic dynamics of the two interacting populations (pests and biocontrol agents) are described by the continuous part of the system and the repeated (periodic) biocontrol agents introductions by the discrete one. Early examples of the use of impulsive models in biology are [8, 14] in chemostat modelling. Since then, they have received considerable attention in the area of epidemiology [3, 16, 37], cancer treatment [4, 5, 20], populations dynamics [24, 25, 41, 21, 31] or integrated pest management [23, 32, 34, 22, 42]; we refer to *e.g.* [28] for a recent overview. Though there are numerous studies on bio-inspired impulsive models, to our knowledge only a few of these are concerned with optimisation: see [2, 11, 29, 40, 43]. Here we focus both on a global convergence result for a rather general predator-prey model with periodic impulsive predator introductions and on a problem of optimisation on how to best deploy these introductions in the context of crop protection through biological control. Our results follow and generalise earlier ones that were obtained in a more restricted context [27].

In this paper, we first present the impulsive predator-prey model of augmentative biological control that is considered. The hypotheses assumed on the biological part (continuous system) are qualitative so that they can encompass a large number of classical biological/ecological functions. The existence of an invariant periodic prey-eradication solution is proved and a sufficient condition that ensures its global asymptotic stability is given.

Then, we focus on an optimisation problem in the perspective of preemptive/preventative use of augmentative biological control. This departs from most classical biological control procedures which are of a more “feedback” type: biocontrol agents are released once the pests have been detected only. Such a preventive approach is also important because it appears to achieve more acceptable pest control especially for high valued agricultural crops like *e.g.* orchards, vegetables, mushrooms or ornamentals, that are very sensitive to the slightest pest outbreak (see [13, 38, 12] for theoretical/simulation studies and [9, 19, 18, 1, 17] for real life experiments and a biological perspective). It is assumed that a fixed budget ensuring pest eradication is allowed and that only its deployment (a tradeoff between frequent-small or rare-large releases) may be modified. The cost function to be minimised is the time taken to reduce

an unforeseen pest invasion under some harmless level and the input is the impulsive release frequency.

It is shown that the local optimisation problem has a countable infinite number of solutions and, in a second step using an argumentation based on the needed robustness of our result, it is proved that the locally optimal solution is to choose as frequent (and thus as small) releases as possible. It is also shown that this strategy gives a sub-optimal solution to the global optimisation problem. Moreover, the higher the frequency, the lower the cost function, so that even if one cannot achieve a very high frequency, it is always interesting to choose the highest possible one. A Monte Carlo like numerical simulation illustrates our analytical optimisation result and the article is concluded by some recommendations to biological control practitioners that follow from our analysis.

2. Model Description and Analysis

2.1. A general impulsive biological control model

In this paper, we assume that the tri-trophic system (crop - pests - biocontrol agents) may be well represented by its bi-trophic simplification representing the prey (pest) predator (biocontrol agent) interactions. The underlying assumption is that the prey population remains at moderate levels so that the crop is not limiting for prey growth and thus do not need to be taken into account in the model. We consider the simple case in which no intra specific interactions within the predator population affects the predator prey interaction, though some in the prey population may occur (see Remark 1 below). According to classical predator-prey modelling, we get the following two-dimensional model

$$\begin{cases} \dot{x} = f(x) - g(x)y \\ \dot{y} = h(x)y - my \end{cases} \quad (1)$$

with x denoting the prey density and y the predator density. $f(x)$ denotes the prey's growth rate, $g(x)$, $h(x)$ and m denotes the predators' functional response, numerical response and mortality rate, respectively. Since biological processes are always difficult to model, we only assume general qualitative hypotheses on the functions $f(\cdot)$, $g(\cdot)$ and $h(\cdot)$ (see Hypothesis 1 below). Thus, we consider a family of models, *i.e.* a “meta-model” *sensu* [6, 7], rather than a specific mathematical model. Such an approach guarantees that our analyses apply to a wide range of biological systems, may they be directly related to biological control or not. Indeed, many are the biological systems that fall in the metamodeling (1): examples may for instance be found in immunology, epidemiology or cancer modelling.

Hypotheses 1 (H1): Let $f(\cdot)$, $g(\cdot)$ and $h(\cdot)$ be locally Lipschitz functions on \mathbb{R}^+ such that:

- (i) $f(0) = 0$
- (ii) $g(0) = 0$, $g'(0) > 0$ and $\forall x > 0$, $g(x) > 0$
- (iii) the function $\frac{f(x)}{g(x)}$ is upper bounded (for $x > 0$)
- (iv) $h(0) = 0$ and $\forall x > 0$, $h(x) \geq 0$

Remark 1: A large part of the predator-prey functions encountered in the literature fit these hypotheses: (H1-i) only indicates that no spontaneous generation of pests (prey) is possible; $f(\cdot)$ may be constant, linear, logistic or

model an Allee effect etc...(H1-ii) means that there is no prey consumption as the prey level is zero and that if some prey are present, the predator are able to find and consume them. Moreover, $g(x)$ is supposed to be increasing at the origin. As a consequence $g(x)$ may be a Holling I or II function. $g(x)$ might even be non-monotonic (in case of e.g. prey group defence abilities) like in Holling IV, but in this case from (H1-iii) $f(x)$ must be of type IV too or become negative for high x values (like for logistic growth). Notice however that Holling III like functional response (i.e. those with null derivative at $x = 0$) fall beyond the scope of this study. No hypotheses are made on the numerical response $h(x)$ except (H1-iv) which is verified by most classical numerical responses (but does not account for intra-predatory interference nor Allee effects for the predators). Indeed, as it will be clearer in the following, our argumentation is mainly based on the fact that $g(x)$ is positive for positive x . Note that according to the different types of biocontrol strategies recalled in the introduction, $h(x) = 0$ for all x would correspond to a purely inundative biocontrol, while $h(x) > 0$ for positive x would take into account inoculative effects too.

We now model the augmentative biological control procedure as the T -periodic releases of biological control agents (with $T > 0$). Releases are, by their very nature, discrete phenomena: every time nT ($n \in \mathbb{N}$) some predators are instantly added into the system. Let us suppose that there is a fixed rate of predators μ (i.e. number of predators per unit time) to be released in the system. Notice that such a quantity μ is a direct measure of the costs of the biological control method over some time period. Henceforth it will be referred to as well as the release rate or the biological control budget.

This results in the following: at each time period T , μT predators are added to the predator population y , yielding the discrete process

$$\forall n \in \mathbb{N}, y(nT^+) = y(nT) + \mu T, \quad (2)$$

with nT^+ denoting the instant right after $t = nT$.

Combining (1) and (2) yields the following impulsive biological control model

$$\begin{cases} \dot{x} = f(x) - g(x)y, \\ \dot{y} = h(x)y - my, \\ \forall n \in \mathbb{N}, y(nT^+) = y(nT) + \mu T. \end{cases} \quad (3)$$

Remark 2: It is quite easy to check from (H1) that the proposed model is, as required for biological models in general, a non-negative system, i.e. the non-negative orthant of the state space is positively invariant for system (3). Then for every non-negative initial conditions x_0 and y_0 at initial time t_0 (that only make sense with respect to the considered problem) model (3) produces non-negative trajectories $x(t)$ and $y(t)$ for all times $t \geq t_0$.

Special cases of model (3) with explicitly formulated functions $f(x)$, $g(x)$ and $h(x)$ have been previously studied in [23, 25]. Their results will be discussed after the statement and proof of our global convergence Theorem.

2.2. Global convergence

In this section we show that provided the predator release rate μ is greater than some value, augmentative biological control is able to drive any pest population to zero. This result is summarised into the following Theorem.

Theorem 1. Under Hypotheses 1, model (3) possesses a “pest free” T -periodic solution

$$(x_p(t), y_p(t)) = \left(0, \frac{\mu T e^{-m(t \bmod T)}}{1 - e^{-mT}} \right) \quad (4)$$

which is locally asymptotically stable if and only if

$$\mu > \frac{mf'(0)}{g'(0)}, \quad (5)$$

and globally asymptotically stable if

$$\mu > S \triangleq \sup_{x \geq 0} \frac{mf(x)}{g(x)}. \quad (6)$$

Remark 3: Note that from (H1-iii) S as defined in (6) does exist.

Proof: We first focus on the “pest free” set: $\{(x, y) \in \mathbb{R}_+^2, x = 0\}$, which is clearly invariant by system (3) through (H1-i) and (H1-ii). Within this set, according to (H1-iv), system (3) becomes

$$\begin{cases} \dot{x} = 0 \\ \dot{y} = -my \\ \forall n \in \mathbb{N}, y(nT^+) = y(nT) + \mu T \end{cases} \quad (7)$$

that yields: $y((n+1)T^+) = y(nT^+)e^{-mT} + \mu T$. It is clear that the sequence $(y(nT^+))_{n \in \mathbb{N}}$ has a single and globally stable equilibrium $y^* = \mu T / (1 - e^{-mT})$. Then, the trajectories of system (7) globally converge towards the T -periodic solution (4).

We now concentrate on the stability of the pest free solution (4) for system (3), that is to say we do not restrict ourselves to the pest free set. We change system (3) variables to consider the deviations from the pest free solution that are denoted (\tilde{x}, \tilde{y}) so that:

$$(\tilde{x}(t), \tilde{y}(t)) = (x(t), y(t)) - (x_p(t), y_p(t))$$

that yields:

$$\begin{cases} \dot{\tilde{x}} = f(\tilde{x}) - g(\tilde{x})(\tilde{y} + y_p(t)) \\ \dot{\tilde{y}} = h(\tilde{x})(y_p(t) + \tilde{y}) - m\tilde{y} \end{cases} \quad (8)$$

We first investigate local asymptotic stability (LAS) of the periodic solution $(x_p(t), y_p(t))$. Assuming (\tilde{x}, \tilde{y}) are small, we get from (8) at first order in \tilde{x} and \tilde{y}

$$\begin{cases} \dot{\tilde{x}} = (f'(0) - g'(0)y_p(t))\tilde{x} \\ \dot{\tilde{y}} = h'(0)y_p(t)\tilde{x} - m\tilde{y} \end{cases} \quad (9)$$

From Theorem 1 in [20], $(x_p(t), y_p(t))$ is LAS if and only if

$$e^{-\int_0^T m d\tau} < 1 \quad \text{and} \quad e^{\int_0^T (f'(0) - g'(0)y_p(\tau)) d\tau} < 1.$$

The former condition is trivial and the latter gives

$$f'(0)T < \int_0^T g'(0) \frac{\mu T e^{-m\tau}}{1 - e^{-mT}} d\tau \Leftrightarrow \mu > \frac{mf'(0)}{g'(0)}. \quad (10)$$

so that the LAS of $(x_p(t), y_p(t))$ is proved.

We now focus on the global asymptotic stability (GAS) of the pest free solution. From now on, we assume that system (3) is initiated at (x_0, y_0) at time $t_0 \geq 0$, *i.e.* system (8) is initiated at $(\tilde{x}_0, \tilde{y}_0) = (x_0, y_0 - y_p(t_0))$ at time $t_0 \geq 0$. Let us consider the function

$$G(\tilde{x}) = m \int_{\varkappa}^{\tilde{x}} \frac{1}{g(s)} ds$$

for some $\varkappa > 0$. Since from (H1-ii) $g(x)$ is positive for positive x , $G(\cdot)$ is an increasing function from $\tilde{x} = 0$, where it goes to $-\infty$ since $g(\cdot)$ is locally Lipschitz on \mathbb{R}^+ . In the following we investigate $G(\tilde{x}(t))$ behaviour as time t goes to infinity, we have

$$\begin{aligned} G(\tilde{x}(t)) - G(\tilde{x}_0) &= m \int_{\tilde{x}_0}^{\tilde{x}(t)} \frac{1}{g(s)} ds, \\ &= m \int_{t_0}^t \frac{\dot{\tilde{x}}(\tau)}{g(\tilde{x}(\tau))} d\tau, \\ &= \int_{t_0}^t \left(\frac{mf(\tilde{x}(\tau))}{g(\tilde{x}(\tau))} - m(\tilde{y}(\tau) + y_p(\tau)) \right) d\tau. \end{aligned}$$

Considering system (8) together with (H1-iv) and Remark 2, we get

$$\dot{\tilde{y}} \geq -m\tilde{y},$$

so that, through standard arguments

$$\tilde{y}(t) \geq \min(0, \tilde{y}_0) e^{-m(t-t_0)}. \quad (11)$$

Using (11) and the definition of S in (6), we then have for all $t \geq t_0$

$$\begin{aligned} G(\tilde{x}(t)) - G(\tilde{x}_0) &\leq \int_{t_0}^t \left(S - my_p(\tau) - m \min(0, \tilde{y}_0) e^{-m(\tau-t_0)} \right) d\tau, \\ &= \int_{t_0}^{\left(\left\lfloor \frac{t_0}{T} \right\rfloor + 1\right)T} (S - my_p(\tau)) d\tau + \left(\left\lfloor \frac{t}{T} \right\rfloor - \left\lfloor \frac{t_0}{T} \right\rfloor - 1 \right) \int_0^T (S - my_p(\tau)) d\tau \\ &\quad + \int_{\left\lfloor \frac{t}{T} \right\rfloor T}^t (S - my_p(\tau)) d\tau + \min(0, \tilde{y}_0) \left(e^{-m(t-t_0)} - 1 \right), \end{aligned}$$

since $y_p(t)$ is T -periodic. It is clear that the first, third and fourth term of the right hand side of (12) are upper bounded.

Now, suppose that (6) holds, then in a similar way to (10)

$$\int_0^T (S - my_p(\tau)) d\tau < 0,$$

so that, since $\left\lfloor \frac{t}{T} \right\rfloor$ goes to infinity as t does, the right hand side of (12) goes to $-\infty$. Then, condition (6) implies

$$\lim_{t \rightarrow +\infty} G(\tilde{x}(t)) = -\infty \Leftrightarrow \lim_{t \rightarrow +\infty} \tilde{x}(t) = 0.$$

One can now prove that \tilde{y} converges to zero as well. Indeed, from (11)

$$\tilde{y}_0 \leq 0 \Rightarrow \tilde{y}(t) \geq \tilde{y}_0 e^{-m(t-t_0)},$$

so that, either $\tilde{y}(t)$ converges to zero in infinite time from below, or it reaches the positively invariant region where $\tilde{y} \geq 0$ in finite time. Up to initial time t_0 translation, we then have to consider the positive \tilde{y}_0 only. Since \tilde{x} converges to zero and $h(0) = 0$, it is clear that there exists a time t_m such that

$$\forall t > t_m, h(\tilde{x}(t)) \leq \frac{m}{2} \Rightarrow \forall t > t_m, \dot{\tilde{y}} \leq h(\tilde{x})y_p(t) - \frac{m}{2}\tilde{y}.$$

Since $h(\tilde{x})y_p(t)$ goes to zero as t goes to infinity, so does \tilde{y} . We have shown that if condition (6) holds, $(0,0)$ is globally attractive for (8), *i.e.* $(x_p(t), y_p(t))$ is globally attractive for (3).

It is then easily checked through l'Hospital rule, (H1-i) and (H1-ii) that condition (6) implies that the necessary and sufficient condition (5) for LAS of (x_p, y_p) holds true, so that GAS follows. \square

Some comments should be given on Theorem 1. In general, the local stability condition (5) and the global stability condition (6) are different. However there are some cases where these two conditions are similar. Indeed consider a classical Lotka Volterra (LV) system where $f(x) = \alpha x$ and $g(x) = \beta x$, then condition (6) becomes

$$\mu > \sup_{x \geq 0} \frac{mf(x)}{g(x)} = \frac{m\alpha}{\beta},$$

which is also condition (5) applied to such a system. Then for a LV system (or systems whose prey equation is similar to the LV prey equation, see *e.g.* [42]) a necessary and sufficient condition for GAS of the pest-free solution (4) is $\mu > \frac{m\alpha}{\beta}$. This result was previously proved for the LV model in [23].

Now consider a Rosenzweig-Mac Arthur like model with logistic growth of the prey and a Holling II functional response of the predator

$$f(x) = rx \left(1 - \frac{x}{K}\right) \quad \text{and} \quad g(x) = \frac{ax}{c+x}. \quad (12)$$

Then condition (6) becomes

$$\mu > \begin{cases} \frac{mr(K+c)^2}{4Ka} & \text{for } 0 \leq c \leq K \\ \frac{mrc}{a} & \text{for } c \geq K \end{cases}$$

Since for such a system, condition (5) becomes $\mu > \frac{mrc}{a}$, for a carrying capacity K smaller than the half saturation constant c of the Holling II, the local stability condition is necessary and sufficient to ensure GAS of the pest free solution [33]. However if $K > c$, the GAS condition of the pest free solution is then stronger than the LAS condition. If the LAS condition holds true, but the GAS does not, Theorem 1 does not allow to conclude on the global behaviour of the system. Indeed, a model verifying (12) has been previously studied in [25] and was shown to possibly exhibit co-existing attractors: this is a possibility here and depending on the initial conditions, trajectories of the system may converge either to the pest free set or to another attractor. This conclusion obviously applies to systems for which condition (5) and (6) are different.

Special cases of model (3) were also studied by [25, 23] as the LAS condition of the pest free set (5) was not satisfied. The classical methodology is to prove the existence of a positive periodic solution through a bifurcation Theorem proposed by [20] and then to show the existence of various routes to chaos through numerical simulations (see also [21, 22, 31, 41] for similar approaches on predator-prey systems).

In this paper however, with respect to our application we concentrate on the pest eradication problem only. In the case where condition (5) holds true, but condition (6) does not, the pest free solution is still locally stable but not globally. Such a release rate has the advantage of being smaller than the one required for GAS so that it would be cheaper for the grower to choose it as such: it allows good control of small pests invasions. The price to pay is however the risk of considerable crop damage if the system escapes to an alternative attractor to the pest free set.

It is to be noted that the stability conditions (5) and (6) on the release rate μ are both independent of the release period T . Therefore, once a release rate μ fulfilling a stability condition is chosen, pest eradication could be achieved independently of the choice of the release period T . Indeed, both infrequent and large releases (T and μT large) or frequent and small releases (T and μT small) would ultimately drive the pest population to zero. Though there is no difference in the long term, different values of the release period T might however result in different outcomes for the transient dynamics of the system. Hence in the following, we will take advantage of the adjustable parameter T to address the practically important question: how to “best” (in a sense that will be detailed in what follows) deploy a given biological control budget μ to eradicate pest outbreaks ?

3. Optimal Choice of the Release Period in the Preemptive Case

We first show two rather general results on one-dimensional non autonomous differential equations that are piecewise monotonous. These results will be central to the analysis we present afterwards; however the reader interested by the application only can go directly to section 3.2.

3.1. Preliminary results

Lemma 1. *Consider a one-dimensional non-autonomous differential equation initiated at $x(t_0) = x_0$*

$$\dot{x} = w(t), \quad (13)$$

with $w(t)$ a T -periodic, negative, increasing function which is continuous on $(0, T)$ and such that

$$\int_0^T w(\tau) d\tau = -\omega T. \quad (14)$$

Let $x_f < x_0$ be the fixed terminal value of x reached at $t = t_f(t_0)$ and $k = \left\lfloor \frac{x_0 - x_f}{\omega T} \right\rfloor$, then either:

- (i) $\left(\frac{x_0 - x_f}{\omega T} \right) = k$ and $(t_f(t_0) - t_0) = kT$ for all t_0 .
- (ii) $\left(\frac{x_0 - x_f}{\omega T} \right) > k$ and $(t_f(t_0) - t_0)$ is maximum at and only at the uniquely defined t_0^* such that $t_f(t_0^*) = (k + 1)T$.

Notice that from (14) and since $w(t)$ is increasing in t , $w(\cdot)$ has necessarily a discontinuity with a negative jump at $t = T$.

Proof: Without loss of generality we assume that $t_0 \in [0, T)$. Define $r(t_0)$ such that $t_f(t_0) = t_0 + kT + r(t_0)$. We have from (13)

$$\frac{x_f - x_0}{\omega T} = \frac{1}{\omega T} \int_{t_0}^{t_0+kT} w(\tau) d\tau + \frac{1}{\omega T} \int_{t_0+kT}^{t_0+kT+r(t_0)} w(\tau) d\tau,$$

so that, from (14) and the fact that $w(t)$ is T -periodic

$$k - \frac{x_0 - x_f}{\omega T} = \frac{1}{\omega T} \int_{t_0}^{t_0+r(t_0)} w(\tau) d\tau. \quad (15)$$

From the definition of k , the left hand side of (15) belongs to $(-1, 0]$, so that, since $w(\cdot)$ is negative and verifies (14), we have

$$r(t_0) \in [0, T).$$

Suppose that $\left(\frac{x_0 - x_f}{\omega T}\right) = k$, then the left hand side of (15) cancels and $r(t_0) = 0$, so that $(t_f(t_0) - t_0) = kT$ for all t_0 .

Else, suppose that $\left(\frac{x_0 - x_f}{\omega T}\right) > k$, then, differentiating (15) with respect to t_0 and rearranging the terms we get for $t_0 \neq 0$ and $t_0 + r(t_0) \neq T$

$$\frac{dr}{dt_0} = \frac{w(t_0)}{w(t_0 + r(t_0))} - 1. \quad (16)$$

Since $w(t)$ is piecewise increasing, T -periodic and $r(t_0) \in [0, T)$, we have

$$t_0 + r(t_0) < T \Rightarrow w(t_0 + r(t_0)) > w(t_0), \text{ and } t_0 + r(t_0) > T \Rightarrow w(t_0 + r(t_0)) < w(t_0),$$

which with (16) yields

$$t_0 + r(t_0) < T \Rightarrow \frac{dr}{dt_0} < 0, \text{ and } t_0 + r(t_0) > T \Rightarrow \frac{dr}{dt_0} > 0.$$

Then as $r(t_0)$ is continuous in t_0 , it has necessarily a maximum if and only if $t_0 = t_0^*$ such that $r(t_0^*) = T - t_0^*$. Thus $(t_f(t_0) - t_0)$ is also maximum if and only if $t_0 = t_0^*$ and we have $t_f(t_0^*) = (k+1)T$. The uniqueness of t_0^* is easily obtained from (15). \square

Lemma 1 is illustrated on figure 1. Notice that, although we do not show it here, Lemma 1 is still valid when $w(t)$ is locally increasing at $t = 0^+$ and $t = T^-$ only and non-decreasing elsewhere.

Lemma 2. Consider equation (13) and other notations as in Lemma 1. Then

$$\max_{t_0} (t_f(t_0) - t_0) \geq \frac{x_0 - x_f}{\omega}$$

with the equality holding if and only if $T = \frac{x_0 - x_f}{n\omega}$ for some $n \in \mathbb{N}^*$.

Proof: Suppose that $T = \frac{x_0 - x_f}{n\omega}$ for some $n \in \mathbb{N}^*$. Then

$$n = \frac{x_0 - x_f}{\omega T}$$

and from case (i) of Lemma 1 $(t_f(t_0) - t_0) = nT = \frac{x_0 - x_f}{\omega}$ for all t_0 .

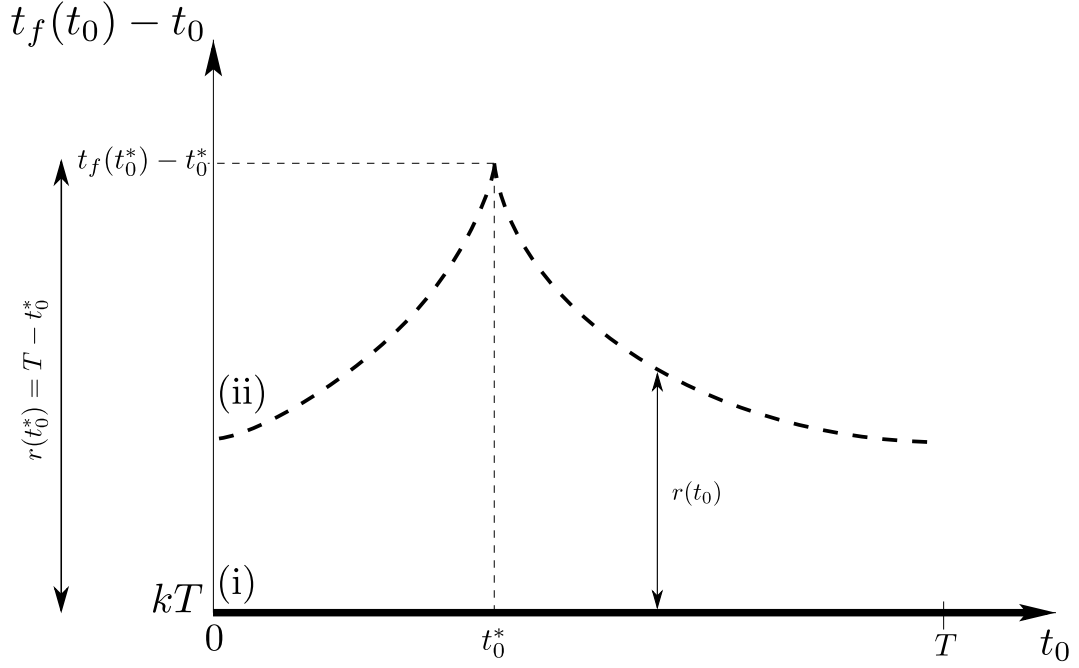


Figure 1: Graphical illustration of Lemma 1 for $w(t)$ increasing in t . Either case (i) holds and $t_f(t_0) - t_0 = kT$ for all t_0 (thick line), or case (ii) holds and $t_f(t_0) - t_0$ is maximum for t_0^* such that $r(t_0^*) = T - t_0^*$ (dashed line). It is shown in Lemma 2 that $r(t_0) > 0$ if case (i) does not hold.

Now suppose that $T \neq \frac{x_0 - x_f}{n\omega}$ for all $n \in \mathbb{N}^*$ and let k and $r(t_0)$ be as in Lemma 1. Then

$$t_f(t_0) - t_0 = kT + r(t_0),$$

with $r(t_0) \in (0, T)$, since $r(t_0) = 0$ leads to the contradiction $\frac{x_0 - x_f}{\omega T} \in \mathbb{N}^*$.

Using this in equation (15), we get at $t_0 = t_0^*$

$$\frac{x_0 - x_f}{\omega} = (t_f(t_0^*) - t_0^*) - r(t_0^*) - \frac{1}{\omega} \int_{t_0^*}^T w(\tau) d\tau,$$

since $t_0^* + r(t_0^*) = T$.

Obviously, as $r(t_0^*) < T$ by definition, $t_0^* > 0$ and as $w(t)$ is increasing on $(0, T)$, we have from (14)

$$\frac{1}{T - t_0^*} \int_{t_0^*}^T w(\tau) d\tau > -\omega,$$

so that

$$\begin{aligned} \frac{x_0 - x_f}{\omega} &< (t_f(t_0^*) - t_0^*) - r(t_0^*) + T - t_0^* \\ &= t_f(t_0^*) - t_0^* \end{aligned}$$

□

We now come back to our main purpose, the identification of the “best” biocontrol agents release policy in model (3).

3.2. Statement of the optimisation problem and optimal choice of the release period

We focus on preemptive/preventative use of biological control agents releases: we suppose that biological control agents are released in anticipation of pests outbreaks, so that natural enemies would be able to fight the pest right at the time of their invasion. Hence in the following, we suppose that preventive releases are being performed and that the system is in the invariant set $\{\tilde{y} \geq 0\}$ as a pest population x_0 invades the crop at some unforeseen moment t_0 .

Our goal is to provide recommendations on how to deploy such a preemptive biological control strategy (*i.e.* how to choose T) to minimise crop damage due to a pest outbreak. To evaluate crop damage induced by the pests, we use the the concept of “Economic Injury Level” (EIL) that has been introduced from the early bases of theoretical biological control [39]. EIL (denoted \bar{x} in the following) is defined as the “lowest (positive) pest population level that will cause economic losses on the crop”. \bar{x} is then a constant parameter of our problem. For a given pest outbreak $x_0 > \bar{x}$, we consider that the damage cost J is an increasing function of the time spent by the pest population above the EIL \bar{x} , *i.e.*

$$J = \int_{t_0}^{t_f} \gamma(\tau) d\tau, \quad (17)$$

with t_f defined such that $x(t_f) = \bar{x}$ and $\gamma(\cdot) > 0$. It is clear that minimising J is equivalent to minimising $\Pi = (t_f - t_0)$ so that we will only concentrate on the latter in the following.

We are left with two more problems. The first one is that, for given x_0 and T , the damage cost J (or Π) strongly depends on the pest outbreak instant t_0 , *i.e.* for a given T the same invading pest level x_0 yields different damage costs depending on the value of t_0 . Without loss of generality, from now on we will consider that $t_0 \in [0, T]$. We seek to provide the safest biological control procedure so that we will concentrate on the damage cost for the worst t_0 , ensuring any other t_0 would result in smaller damage costs. Hence in the following, we seek the time period T that minimises the damage cost for its worst t_0 *i.e.* we seek to minimise $\max_{t_0 \in [0, T]} \Pi$.

The remaining problem is that our analysis cannot be achieved for the general model (3). It is however possible to carry it out for a local approximation of \dot{x} about $x = 0$ as well as for an upper-bound of \dot{x} . Indeed, consider the local approximation of \dot{x} near (x_p, y_p) . From (9), and considering the change of variable $z_1 = \frac{m}{g'(0)} \ln\left(\frac{x}{\bar{x}}\right)$ (which is obviously increasing in x), we get

$$\dot{z}_1 = S_l - my_p(t),$$

where $S_l = \frac{mf'(0)}{g'(0)} \leq S$.

Now we consider the general \dot{x} equation of model (3) under the hypothesis that the system is in the positively invariant set $\{\tilde{y} \geq 0\}$. We get for all $t \geq t_0$

$$\dot{x} \leq f(x) - g(x)y_p(t).$$

Let us make the change of variable $z_2 = m \int_{\bar{x}}^x \frac{1}{g(x)} dx$ (which is increasing in x), we get

$$\dot{z}_2 \leq S - my_p(t),$$

whose right hand side is similar to the \dot{z}_1 equation.

In the rest of the paper we will then focus on the system

$$\dot{z} = \sigma - my_p(t), \quad (18)$$

with z standing for z_1 or z_2 and σ for S_l or the original S . Through equation (18) we will thus study in one step the exact local approximation of our system as well as an upper bound of it, yielding a locally optimal result as well as a globally sup-optimal one.

Since the biological control program must eradicate the pest population, we assume that the stability condition of the pest free solution holds true, we have

Hypothesis 2 (H2): *The biological control budget is chosen such that: $\mu > \sigma$*

We first show that (see Appendix A):

Proposition 1. *There exists a release period \hat{T} such that if $T < \hat{T}$, the pest population is decreasing for $t \geq t_0$.*

Proposition 1 is of practical interest regarding growers concerns: indeed it is not interesting to invest in preventative releases of biological control agents that allow an invading pest population to proliferate, even temporarily, after the moment of invasion. It is much more desirable to choose a procedure that will always make the pest population decreasing after the invasion. Thus in what follows we assume that

Hypothesis 3 (H3): *The release period is chosen such that $T \in (0, \hat{T})$.*

We now seek the moment of invasion t_0 that maximises the damage cost J (i.e. that maximises the damage time Π) for a given invading pest population $x_0 > \bar{x}$ (corresponding to $z_0 > 0$) and a given release period $T \in (0, \hat{T})$. Notice that with the z formalism, we have $\Pi = t_f - t_0$ with $z(t_0) = z_0$ and $z(t_f) = 0$. Since from (H2) and (H3) equation (18) falls within the scope of Lemma 1 with $\omega = \mu - \sigma$. We easily have:

Lemma 3. *Consider equation (18), assume that $z_0 > 0$ and $T \in (0, \hat{T})$ are fixed, and let $k = \left\lfloor \frac{z_0}{(\mu - \sigma)T} \right\rfloor$, then either:*

- (i) $z_0 = (\mu - \sigma)kT$ and $\Pi = kT$ for all t_0
- (ii) $z_0 > (\mu - \sigma)kT$ and $\max_{t_0} \Pi(t_0) = (k + 1)T - t_0^*$ is reached at, and only at, the uniquely defined t_0^* such that $t_f(t_0^*) = (k + 1)T$.

We now investigate which $T \in (0, \hat{T})$ minimises the worst case damage time $\max_{t_0} \Pi$ (i.e. that minimises the worst case damage $\max_{t_0} J$). Applying Lemma 2, we get the following result:

Theorem 2. *Suppose $z_0 > 0$ is fixed. Let*

$$T_1 = \frac{z_0}{\mu - \sigma}. \quad (19)$$

Then there exists $n_0 \in \mathbb{N}^*$ such that $\min_{T \in (0, \hat{T})} \max_{t_0} \Pi(t_0) = T_1$ is reached at, and only at, $T = T_n = \frac{T_1}{n}$ for all integer $n > n_0$.

Proof: Consider T_1 that is positive since μ is assumed to be greater than σ . It is then clear that there exists an integer n_0 such that for all integer $n > n_0$, $T_n < \hat{T}$ so that equation (18) verifies the condition of Lemma 1 for all $n > n_0$. The remaining part of the proof is straightforward using Lemma 2. \square

Before commenting on Theorem 2, we notice that for any $T < \hat{T}$ one can express $\max_{t_0}(\Pi)$ as a function of T_1 , T and t_0^* . Indeed, integrating (18) between t_0^* and $t_f(t_0^*)$ for some $T < \hat{T}$ yields

$$\begin{aligned} z(t_f) = 0 &= z_0 + \sigma \max_{t_0} \Pi - \frac{m\mu T}{1 - e^{-mT}} \left[\int_{t_0^*}^T e^{-m\tau} d\tau + k \int_0^T e^{-m\tau} d\tau \right] \\ &= z_0 + \sigma \max_{t_0} \Pi - (k+1)\mu T + \mu T \frac{1 - e^{-mt_0^*}}{1 - e^{-mT}} \end{aligned} \quad (20)$$

so that, recalling from Lemma 3 that $(k+1)T = t_0^* + \max_{t_0} \Pi$, we get

$$\max_{t_0} \Pi = T_1 + \frac{\mu}{\mu - \sigma} \left(\frac{1 - e^{-mt_0^*}}{1 - e^{-mT}} T - t_0^* \right) \quad (21)$$

Theorem 2 shows that for a given z_0 we have a countable infinite number of release periods, the T_n , that solves our optimisation problem.

However, we have two difficulties with Theorem 2. On the one hand, through equation (18) we studied both the local optimisation problem and the global suboptimal one. To solve these two, we need to choose a release period $T = T_n$, which depends on σ . In the local optimisation case, σ equals $S_l = \frac{mf'(0)}{g'(0)}$ while in the global suboptimal one, σ equals $S = \sup_{x \geq 0} \frac{mf(x)}{g(x)}$. There is no reason why $\frac{S}{S_l}$ would be rational, so that Theorem 2 states that it is not possible to choose a release period T that solves both the local and global optimisation problem; the best that can be done is to choose T to be as close as possible to the solution of both optimisation problems.

On the other hand, it is to be noticed that T_1 , which determines the optimal values of the release period, depends on some “biological” values, namely the invading pest population level x_0 (*i.e.* z_0) and the combination of parameters σ . This is quite a big drawback since biological parameters are usually badly known and the initial invading pest level x_0 is not known at all. Clearly such model-based optimisation approaches should take these uncertainties into account [26].

Hence, in the following, we will show how T should be chosen in order to ensure that the worst case damage time $\max_{t_0} \Pi$ remains close to its minimal value, independently of the value of x_0 and σ . In doing so, we will provide an almost optimal choice of T that solves both the local and global optimisation problem and that is robust to the value of x_0 and to uncertainty of the parameters.

3.3. Robustness Analysis

In the preceding sections, we wanted our choice of T to minimise the time that it takes to reach \bar{x} from x_0 for the worst possible initial time t_0 . In addition to that, we now also want this to be robust with respect to the uncertainty

on the initial condition x_0 and to the model parameters that we will denote p in the rest of the paper (p is simply the pair (σ, m)). Indeed, in the poorly measured conditions of crop culture and with the accompanying scarcely known models, it is fundamental for a control method to be robust with respect to these uncertainties. We then suppose that the “true” x_0 and p belongs to some compact sets:

Hypothesis 4 (H4): *The uncertainties on the initial condition and the model parameters are bounded, with $z_0 \in [\underline{z}_0, \overline{z}_0]$ denoted \mathbf{Z} and p that belongs to a compact set \mathbf{P} .*

We still assume that the invading population is greater than the EIL \bar{x} (i.e. $\underline{z}_0 > 0$) and that the stability condition holds, i.e. $\mu > \max_{p \in \mathbf{P}}(\sigma)$. Our optimisation procedure will be robust provided our choice of T minimises $\max_{t_0} \Pi$ for the worst z_0 and p choice within the sets defined in Hypotheses (H4).

Notice that the minimum (with respect to T) of $\max_{t_0} \Pi$ is equal to T_1 that does depend on both z_0 and p . Thus in our robustness analysis it makes more sense to consider the deviation of $\max_{t_0} \Pi$ from T_1 , rather than the absolute value of $\max_{t_0} \Pi$. Hence, we first focus on the evolution of

$$\max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right]$$

with respect to T . We have the following result:

Theorem 3. *The function*

$$T \mapsto \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] \quad (22)$$

is increasing for T smaller than some positive constant T_L . Moreover

$$\lim_{T \rightarrow 0^+} \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = 0. \quad (23)$$

Proof: From equation (21), we have

$$\begin{aligned} \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] \\ = \max_{p \in \mathbf{P}} \left[\frac{\mu}{\mu - \sigma} \max_{z_0 \in \mathbf{Z}} \left(\frac{1 - e^{-m t_0^*(T, z_0, p)}}{1 - e^{-mT}} T - t_0^*(T, z_0, p) \right) \right]. \end{aligned}$$

We first concentrate on

$$\max_{z_0 \in \mathbf{Z}} \left(\frac{1 - e^{-m t_0^*(T, z_0, p)}}{1 - e^{-mT}} T - t_0^*(T, z_0, p) \right) \quad (24)$$

for fixed T and p . Let us introduce

$$T_l(\overline{z}_0, \underline{z}_0, p) = \min \left(\hat{T}(p), \frac{\overline{z}_0 - \underline{z}_0}{2(\mu - \sigma)} \right),$$

with $\hat{T}(p)$ defined in Proposition 1. In the following, we consider that T belongs to $(0, T_1(\bar{z}_0, \underline{z}_0, p))$. On the one hand, this ensures through Proposition 1 that, independently of the actual parameters, the pest population (*i.e.* z in our new variables) always decreases after the invasion time t_0 . On the other hand, we have

$$\frac{\bar{z}_0 - \underline{z}_0}{T(\mu - \sigma)} > 2,$$

so that there exists $n \in \mathbb{N}$ and $z_{01}, z_{02} \in [\underline{z}_0, \bar{z}_0]$ such that

$$\frac{z_{01}}{T(\mu - \sigma)} = n \text{ and } \frac{z_{02}}{T(\mu - \sigma)} = n + 1.$$

From Theorem 2, T is thus an optimal period for both the initial conditions z_{01} and z_{02} .

For all $z_0 \in (z_{01}, z_{02})$, case (ii) of Lemma 3 holds for $k = n$, since $k = \lfloor \frac{z_0}{T(\mu - \sigma)} \rfloor$. Then, since T is an optimal period for the initial condition z_{01} , we should have by continuity

$$\lim_{z_0 \rightarrow z_{01}^+} \Pi(t_0^*(T, z_0, p)) = (n + 1)T - \lim_{z_0 \rightarrow z_{01}^+} t_0^*(T, z_0, p) = T_1(z_{01}, p),$$

so that

$$\begin{aligned} \lim_{z_0 \rightarrow z_{01}^+} t_0^*(T, z_0, p) &= (n + 1)T - T_1(z_{01}, p), \\ &= (n + 1)T - \frac{z_{01}}{\mu - \sigma}T = T. \end{aligned}$$

A similar argumentation on z_{02} yields

$$\lim_{z_0 \rightarrow z_{02}^-} t_0^*(T, z_0, p) = 0.$$

From (20) and since $\max_{t_0} \Pi = (k + 1)T - t_0^*$, we can conclude that $t_0^*(T, z_0, p)$ is a continuous function of z_0 . Then, as z_0 covers $[z_{01}, z_{02}]$, $t_0^*(T, z_0, p)$ reaches its whole interval of definition $[0, T]$ so that we have:

$$\begin{aligned} \max_{z_0 \in [z_{01}, z_{02}]} \left(\frac{1 - e^{-mt_0^*(T, z_0, p)}}{1 - e^{-mT}} T - t_0^*(T, z_0, p) \right) &= \max_{t_0^* \in [0, T]} \left(\frac{1 - e^{-mt_0^*}}{1 - e^{-mT}} T - t_0^* \right), \\ &= \max_{z_0 \in \mathbb{Z}} \left(\frac{1 - e^{-mt_0^*(T, z_0, p)}}{1 - e^{-mT}} T - t_0^*(T, z_0, p) \right). \end{aligned}$$

Differentiating $\left(\frac{1 - e^{-mt_0^*}}{1 - e^{-mT}} T - t_0^* \right)$ with respect to t_0^* , we show that it reaches its maximum for

$$\hat{t}_0^* = \frac{1}{m} \ln \left(\frac{mT}{1 - e^{-mT}} \right). \quad (25)$$

$\hat{t}_0^* > 0$ since $mT > 1 - e^{-mT}$. In addition, $\hat{t}_0^* < T$ since $e^{mT} > \frac{mT}{1 - e^{-mT}}$, then

$$\begin{aligned} \max_{z_0 \in \mathbb{Z}} \left(\frac{1 - e^{-mt_0^*(T, z_0, p)}}{1 - e^{-mT}} T - t_0^*(T, z_0, p) \right) &= \frac{1 - e^{-m\hat{t}_0^*}}{1 - e^{-mT}} T - \hat{t}_0^*, \\ &= \frac{1}{m} \left[\frac{mT}{1 - e^{-mT}} - 1 - \ln \left(\frac{mT}{1 - e^{-mT}} \right) \right], \\ &\triangleq H(T, p). \end{aligned}$$

$H(T, p)$ is an increasing function in T on $(0, T_l(\underline{z}_0, \overline{z}_0, p))$ since

$$\frac{\partial H(T, p)}{\partial T} = \frac{e^{-mT}(e^{mT} - 1 - mT)}{(1 - e^{-mT})^2} \left[1 - \frac{1 - e^{-mT}}{mT} \right] > 0.$$

Moreover, through l'Hospital rule, we have for all $p \in \mathbf{P}$, $\lim_{T \rightarrow 0^+} H(T, p) = 0$.

Let us introduce:

$$T_L = \min_{p \in \mathbf{P}} T_l(\underline{z}_0, \overline{z}_0, p) > 0.$$

Through the above analysis, it has been shown that for $T \in (0, T_L)$ and $p \in \mathbf{P}$, $H(T, p)$ is an increasing function of T with $\lim_{T \rightarrow 0^+} H(T, p) = 0$.

We now come back to the function defined in (22). We have

$$\max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = \max_{p \in \mathbf{P}} \left(\frac{\mu}{\mu - \sigma} H(T, p) \right).$$

Then, from $H(T, p)$ properties, for T smaller than T_L the worst deviation of $\max_{t_0} \Pi$ from T_1 (according to $(z_0, p) \in \mathbf{Z} \times \mathbf{P}$) is an increasing function of T . Moreover

$$\lim_{T \rightarrow 0^+} \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = 0.$$

□

One can deduce the following Corollary from Theorem 3 (see Appendix B):

Corollary 1. Let $\overline{T} = \min_{p \in \mathbf{P}} \hat{T}(p)$, then

$$\inf_{T \in (0, \overline{T})} \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0 \in [0, T]} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = 0 \quad (26)$$

is achieved for $T = 0^+$.

With the help of Theorem 3, we know that provided T is not too large, the smaller T is chosen, the less will be the worst case (according to parameters and initial condition uncertainty) deviation of $\max_{t_0} \Pi$ from its minimal value T_1 . It has moreover been shown that as T tends to 0, the worst case deviation of $\max_{t_0} \Pi$ from its minimal value goes to 0 as well.

We conclude from this analysis that the robust choice of T , as defined by the criterion (26) consists in choosing $T = 0$. However, it is obviously not possible to achieve such a T in practical applications: it would consist in continuously releasing predators into the crop culture (see also the discussion in Section 3.4). As an alternative, and since the argument of the infimum in (26) is an increasing function of T for $T \in (0, T_L)$, it is then advised to take T as small as possible, *i.e.* to perform as small and as frequent introductions as possible, to achieve the best possible robustness.

A last remark should be made about the need for robustness. In the preceding analysis, we have concentrated on a single model in the form (18). We should remember that this model covers two cases: a local linearisation of

the system (which yields a certain value of S) and a system that globally upper-bounds the actual nonlinear system (which yields another value of S). At the very least, the choice of T that we made should work well in both those cases. We can then conclude that, even in the unrealistic case where the initial conditions and the parameters are very well-known, one should make a choice of T that is sufficiently robust, so that both global and local approximations are covered, *i.e.* one should take T as small as possible.

3.4. As small and as frequent introductions as possible ?

In the preceding, it was supposed that the overall cost of the pest management program was determined by those of the biocontrol agents to be released (μ which is fixed) and of the damage J . Minimising the pest management program cost was then equivalent to minimising J . In practice, additional expenses may also be incurred by higher frequencies of releases, in terms for instance of fixed labor costs related to the time spent doing one biocontrol agents release on the crop. In this case, assuming a fixed spending for each predator release, leads to add a positive, convex and decreasing in the release period T function to the damage J to estimate the overall pest management cost. The overall worst case cost would then be the sum of a positive, convex and decreasing function in T and of a positive, convex and increasing function in T (defined on the basis of (22)). It is then clear that this overall worst case cost would have a minimum with respect to the release period T : below this value, the cost would be decreasing in T and above it would be increasing.

Precisely determining the value of T for which the overall worst case cost is minimum relies however on the perfect knowledge of the function (22), which is in turn strongly dependent on the uncertainties on the parameters and invading pest population. Hence, determining the optimal T is not really possible. However one can argue that for the high valued and sensitive crops that we consider in this paper, the labor costs related to a release remains low with respect to the costs induced by a pest outbreak. In this situation it is most probable that the optimal T would be below any reasonable biocontrol agents release period¹. Therefore there remains fair reasons to prefer frequent and small releases over large and infrequent ones.

4. Numerical Simulation

In order to go beyond the worst case analysis and to illustrate the robustness result stated in Theorem 3 we performed a Monte Carlo like numerical simulation that showed how the damage time Π varied around T_1 . An impulsive predator-prey model of the form (3) was thus considered with constant a priori chosen parameters for the continuous part of the model and μ verifying condition (6). A triplet of the remaining parameters was randomly chosen: first a release period $T \in (0, T_L)$, the pest invasion moment $t_0 \in [0, T)$ and the pest invasion level $x_0 > \bar{x}$. The

¹Indeed, and as in [15] that reported an analysis on the optimisation of the spatial deployment of a fixed amount of biological control agents, “as small as possible” (size and period of releases) should be interpreted loosely: too small releases would actually drive the system beyond the validity domain of the spatially implicit model (3).

value of T_L is easy to compute from (27) since in this case the parameter set \mathbf{P} is reduced to a singleton and we have chosen a large \mathbf{Z} interval. For each triplet, the time to reach \bar{x} , Π , as well as its deviation from T_1 , was then computed by means of numerical integration of the model. The process was repeated until a set of 2.10^5 different simulations was obtained and the results, the deviation of Π from T_1 , were plotted against the time period T . As such, we obtained a representative set of simulations of pest invasions (varying in intensity and moment of invasion) that challenged a crop under strategies of biological control varying in dose and frequency of biocontrol agents releases, as described by equation (2).

The results of this numerical work is presented on Figure 2, each of the dot corresponding to one of the 2.10^5 simulations. What actually corresponds to Theorem 3 is the upper envelope of the data set only (since it is the maximum with respect to parameters of the worst case, according to t_0 , of Π).

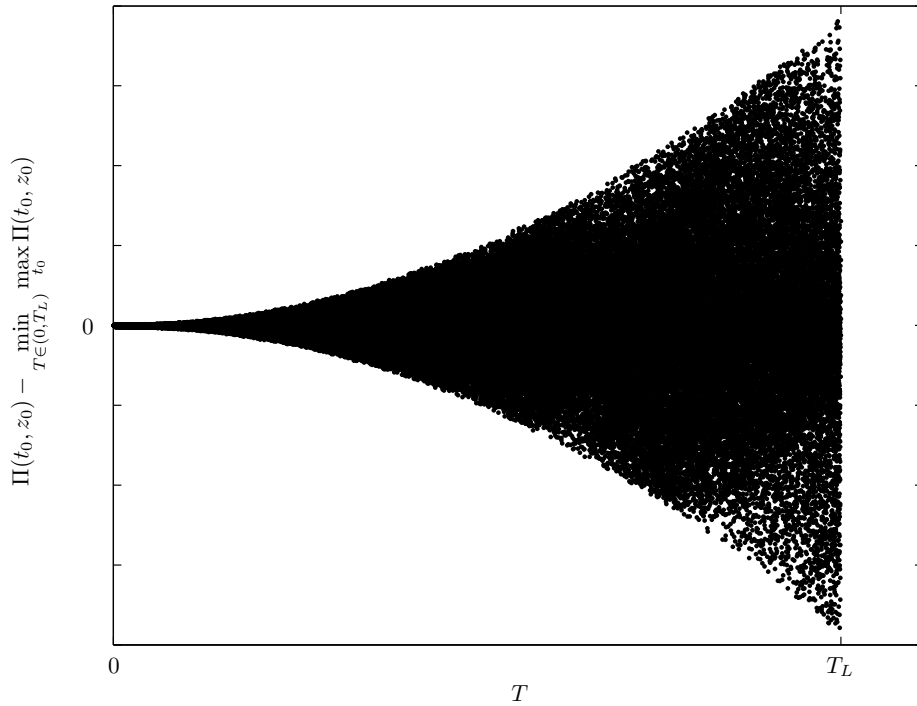


Figure 2: Graphical illustration of Theorem 3. Each dot corresponds to the deviation of the damage time Π from its minimal worst case T_1 for one of the 2.10^5 randomly chosen triplet $T \in (0, T_L)$, $t_0 \in (0, T)$ and $x_0 > \bar{x}$. The larger the release period T , the higher the damage time Π may be.

As it can be noticed the smaller the choice of T , the smaller the positive deviation of Π (thus of the crop damage) from its optimal value T_1 . The price to pay is however that such a choice reduces also the negative deviation of Π from T_1 . Indeed an analysis similar to the one performed in Section 3, but focusing on:

$$\min_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\min_{t_0} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right]$$

would show that this is a decreasing function of T with limit 0 as T tends to 0^+ . Hence, if one feels lucky, a choice of

T small may not be the best one since one can obtain, fortunately, smaller Π (thus smaller crop damage) if the actual t_0 is not the worst one.

5. Conclusion

In this contribution we studied a rather general predator-prey model with periodic impulsive additions of predators (releases) that is relevant for biological control modelling. The impulsive releases of the biological control agents were modelled on the basis of a fixed budget to be spent per time period (release rate). As such it allowed us to compare the different releases period as the same overall amount of predators was used. In a first step, it was shown that, provided the release rate of biocontrol agents was high enough, the biological control program guarantees the eradication of the prey (pest) population, whatever the value of the release periods. Then the efficiency of different release periods (for a given release rate) were compared on the assumption that the biological control program is conducted on a preventive basis. It was shown that the time needed to eradicate an unforeseen pest invasion occurring at a worst moment had a minimum that could be achieved through a countable infinite number of release periods. However, these release period values were strongly dependent on the model parameters and a robustness analysis of this optimisation problem showed that the smaller the release period, the less the time needed to eradicate the pest invasion, for the worst invasion moment and accounting for parameter uncertainty. Hence, as an advice to biological control practitioners, our study indicates that it is less risky to release frequent small amounts of biocontrol agents rather than massive infrequent ones. This result (frequent releases of small doses yield better control) is similar to the ones of [44, 45, 13] that were obtained from simulation models. Such a biocontrol strategy is also advocated by [1] from real life experiments and by [38] on the basis of a simulation model. To our knowledge, this contribution is among the firsts to recommend such a strategy on the basis of a mathematical analysis.

In a broader context, we believe that the techniques used in this work may also be of interest for optimisation problems on impulsive models from other applied fields: optimisation of vaccination strategies in epidemiology [37, 3, 16] or optimisation of chemotherapy in cancer treatments [20, 4, 5]. For instance, a first application may be to re-investigate the work by Pugliese and Gandolfi in immunology [35], that shows, without the explicit use of impulsive modelling techniques, that frequent host re-infections and/or vaccinations are necessary to the persistence of host immunity.

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A. Proof of Proposition 1

Consider equation (18). Provided $\min_t y_p(t) > \frac{\sigma}{m}$, z is decreasing for $t \geq t_0$; then so does the pest population x . Notice that

$$\min_t y_p(t) = \frac{\mu T}{e^{mT} - 1}$$

is a decreasing function of T with

$$\lim_{T \rightarrow 0^+} \min_t y_p(t) = \frac{\mu}{m} > \frac{\sigma}{m},$$

since the stability condition holds.

Then Proposition 1 holds true with \hat{T} solution of

$$\frac{\mu \hat{T}}{e^{m\hat{T}} - 1} = \frac{\sigma}{m}. \quad (27)$$

□

B. Proof of Corollary 1

From Theorem 3:

$$\inf_{T \in (0, T_L)} \max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0 \in [0, T]} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = 0 \quad (28)$$

and is achieved for $T = 0^+$ since the argument of the infimum in (28) is increasing in T and tends to 0 as T does.

To complete the proof of Corollary 1 we have to study

$$\max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0 \in [0, T]} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right]$$

for $T \in [T_L, \bar{T}]$. Notice that from T_L definition, we have:

$$T_L = \min_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left(\min \left(\hat{T}(p), \frac{\bar{z}_0 - z_0}{2(\mu - \sigma)} \right) \right) = \min \left(\bar{T}, \min_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left(\frac{\bar{z}_0 - z_0}{2(\mu - \sigma)} \right) \right),$$

so that the set $[T_L, \bar{T}]$ is non-empty if and only if $\bar{T} > \min_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left(\frac{\bar{z}_0 - z_0}{2(\mu - \sigma)} \right)$. Suppose that this holds and that $T \in [T_L, \bar{T}]$, then we have

$$\max_{z_0 \in \mathbf{Z}} \left(\frac{1 - e^{-m_0^*(T, z_0, p)}}{1 - e^{-mT}} - t_0^*(T, z_0, p) \right) > 0,$$

since otherwise it would be required that $\forall z_0 \in \mathbf{Z}$ there would exist $n \in \mathbb{N}$ such that $T = \frac{z_0}{n(\mu - \sigma)}$ which is obviously not possible. Then, $\forall T \in [T_L, \bar{T}]$

$$\max_{(z_0, p) \in \mathbf{Z} \times \mathbf{P}} \left[\left(\max_{t_0 \in [0, T]} \Pi(t_0, z_0, p) \right) - T_1(z_0, p) \right] = \max_{p \in \mathbf{P}} \left[\frac{\mu}{\mu - \sigma} \max_{z_0 \in \mathbf{Z}} \left(\frac{1 - e^{-m_0^*(T, z_0, p)}}{1 - e^{-mT}} - t_0^*(T, z_0, p) \right) \right],$$

this latter being positive, which concludes the proof. □

References

- [1] M. E. De Courcy Williams. Biological control of thrips on ornamental crops: Interactions between the predatory mite neoseiulus cucumeris (acari : Phytoseiidae) and western flower thrips, frankliniella occidentalis (thysanoptera : Thripidae), on cyclamen. *Biocontrol Science and Technology*, 11(1):41–55, 2001.
- [2] L. Dong, L. Chen, and L. Sun. Optimal harvesting policies for periodic gompertz systems. *Nonlinear Analysis: Real World Applications*, 8(2):572–578, 2007.
- [3] A. D’Onofrio. Stability properties of pulse vaccination strategy in SEIR epidemic model. *Mathematical Biosciences*, 179(1):57–72, 2002.
- [4] A. D’Onofrio and A. Gandolfi. Tumour eradication by antiangiogenic therapy: analysis and extensions of the model by hahnfeldt et al. (1999). *Mathematical Biosciences*, 191(2):159–184, 2004.
- [5] A. D’Onofrio. A general framework for modeling tumor-immune system competition and immunotherapy: Mathematical analysis and biomedical inferences. *Physica D-Nonlinear Phenomena*, 208(3-4):220–235, 2005.
- [6] A. D’Onofrio. Tumorimmune system interaction: Modeling the tumor-stimulated proliferation of effectors and immunotherapy. *Mathematical Models and Methods in Applied Sciences*, 16:1375–1401, 2006.
- [7] A. D’Onofrio. Metamodeling tumorimmune system interaction, tumor evasion and immunotherapy. *Mathematical and Computer Modelling*, 47(5-6):614–637, 2008.
- [8] W. Ebenhoh. Coexistence of an unlimited number of algal species in a model system. *Theoretical Population Biology*, 34:130–144, 1988.
- [9] L. E. Ehler, R. F. Long, M. G. Kinsey, and S. K. Kelley. Potential for augmentative biological control of black bean aphid in california sugarbeet. *Entomophaga*, 42(1-2):241–256, 1997.
- [10] J. Eilenberg, A. Hajek, and C. Lomer. Suggestions for unifying the terminology in biological control. *BioControl*, 46(4):387–400, 2001.
- [11] K. Erdlenbruch, A. Jean-Marie, M. Tidball, and M. Moreaux. Cyclical versus non-cyclical harvesting policies in renewable resource economics. In *15th EAERE Annual Conference, Thessaloniki, Greece*, 2007.
- [12] A. Fenton, R. L. Gwynn, A. Gupta, R. Norman, J. P. Fairbairn, and P. J. Hudson. Optimal application strategies for entomopathogenic nematodes: integrating theoretical and empirical approaches. *Journal of Applied Ecology*, 39(3):481–492, 2002.
- [13] A. Fenton, R. Norman, J. P. Fairbairn, and P. J. Hudson. Evaluating the efficacy of entomopathogenic nematodes for the biological control of crop pests: A nonequilibrium approach. *American Naturalist*, 158(4):408–425, 2001.
- [14] E. Funasaki and M. Kot. Invasion and chaos in a periodically pulsed mass-action chemostat. *Theoretical Population Biology*, 44(2):203–224, 1993.
- [15] F. S. Grevstad. Factors influencing the chance of population establishment: Implications for release strategies in biocontrol. *Ecological Applications*, 9(4):1439–1447, 1999.
- [16] J. Hui and L. S. Chen. Impulsive vaccination of SIR epidemic models with nonlinear incidence rates. *Discrete and Continuous Dynamical Systems- Series B*, 4(3):595–605, 2004.
- [17] J. Hulshof, E. Ketoja, and I. Vanninen. Life history characteristics of frankliniella occidentalis on cucumber leaves with and without supplemental food. *Entomologia Experimentalis Et Applicata*, 108(1):19–32, 2003.
- [18] R. J. Jacobson, D. Chandler, J. Fenlon, and K. M. Russell. Compatibility of beauveria bassiana (balsamo) vuillemin with amblyseius cucumeris oudemans (acarina : Phytoseiidae) to control frankliniella occidentalis pergande (thysanoptera : Thripidae) on cucumber plants. *Biocontrol Science and Technology*, 11(3):391–400, 2001.
- [19] R. J. Jacobson, P. Croft, and J. Fenlon. Suppressing establishment of frankliniella occidentalis pergande (thysanoptera : Thripidae) in cucumber crops by prophylactic release of amblyseius cucumeris oudemans (acarina : Phytoseiidae). *Biocontrol Science and Technology*, 11(1):27–34, 2001.
- [20] A. Lakmeche and O. Arino. Bifurcation of non trivial periodic solutions of impulsive differential equations arising chemotherapeutic treatment. *Dynamics of Continuous Discrete and Impulsive Systems*, 7(2):265–287, 2000.
- [21] B. Liu, L. S. Chen, and Y. J. Zhang. The dynamics of a prey-dependent consumption model concerning impulsive control strategy. *Applied Mathematics and Computation*, 169(1):305–320, 2005.

- [22] B. Liu, Y. J. Zhang, and L. S. Chen. Dynamic complexities of a Holling I predator-prey model concerning periodic biological and chemical control. *Chaos Solitons & Fractals*, 22(1):123–134, 2004.
- [23] B. Liu, Y. J. Zhang, and L. S. Chen. The dynamical behaviors of a Lotka-Volterra predator-prey model concerning integrated pest management. *Nonlinear Analysis - Real World Applications*, 6(2):227–243, 2005.
- [24] S. Q. Liu, L. S. Chen, and R. Agarwal. Recent progress on stage-structured population dynamics. *Mathematical and Computer Modelling*, 36(11-13):1319–1360, 2002.
- [25] X. N. Liu and L. S. Chen. Complex dynamics of holling type ii lotka-volterra predator-prey system with impulsive perturbations on the predator. *Chaos Solitons & Fractals*, 16(2):311–320, 2003.
- [26] C. Loehle. Control theory and the management of ecosystems. *Journal of Applied Ecology*, 43(5):957–966, 2006.
- [27] L. Mailleret and F. Grogard. Optimal release policy for prophylactic biological control. *Positive Systems, Lecture Notes in Control and Information Sciences*, Springer, 341:89–96, 2006.
- [28] L. Mailleret and V. Lemesle. A note on semi-discrete modelling in the life sciences. *Philosophical Transactions of the Royal Society, series A*, In Press, 2009.
- [29] X. Z. Meng, Z. T. Song, and L. S. Chen. A new mathematical model for optimal control strategies of integrated pest management. *Journal of Biological Systems*, 15(2):219–234, 2007.
- [30] W. W. Murdoch, J. Chesson, and P. L. Chesson. Biological control in theory and practice. *The American Naturalist*, 125(3):344–366, 1985.
- [31] K. Negi and S. Gakkhar. Dynamics in a Beddington-DeAngelis prey-predator system with impulsive harvesting. *Ecological Modelling*, 206(3-4):421–430, 2007.
- [32] S. Nundloll, L. Mailleret, and F. Grogard. The effects of partial crop harvest on biological pest control. *Rocky Mountain Journal of Mathematics*, 38(5):1633–1661, 2008.
- [33] S. Nundloll, L. Mailleret, and F. Grogard. Two models of interfering predators in impulsive biological control. *Journal of Biological Dynamics*, In Press, 2009.
- [34] S. Nundloll, L. Mailleret, and F. Grogard. Impulsive biological control with intra-predatory interference: global stability and convergence. *Bulletin of Mathematical Biology*, Submitted.
- [35] A. Pugliese and A. Gandolfi. A simple model of pathogen-immune dynamics including specific and non-specific immunity. *Mathematical Biosciences*, 214(1-2):73–80, 2008.
- [36] G.K. Roderick and M. Navajas. Genes in new environments: Genetics and evolution in biological control. *Nature Reviews Genetics*, 4(11):889–899, 2003.
- [37] B. Shulgin, L. Stone, and Z. Agur. Pulse vaccination strategy in the SIR epidemic model. *Bulletin of Mathematical Biology*, 60(6):1123–1148, 1998.
- [38] D. J. Skirvin, M. E. De Courcy Williams, J. S. Fenlon, and K. D. Sunderland. Modelling the effects of plant species on biocontrol effectiveness in ornamental nursery crops. *Journal of Applied Ecology*, 39(3):469–480, 2002.
- [39] V. M. Stern, R. F. Smith, R. van den Bosch, and K. S. Hagen. The integrated control concept. *Hilgardia*, 29:81–101, 1959.
- [40] S. Tang, R. A. Cheke, and Y. Xiao. Optimal impulsive harvesting on non-autonomous beverton-holt difference equations. *Nonlinear Analysis*, 65(12):2311–2341, 2006.
- [41] S. Y. Tang and L. S. Chen. Multiple attractors in stage-structured population models with birth pulses. *Bulletin of Mathematical Biology*, 65(3):479–495, 2003.
- [42] S. Tang and R. A. Cheke. Models for integrated pest control and their biological implications. *Mathematical Biosciences*, 215:115–125, 2008.
- [43] Y. Xiao, D. Cheng, and H. Qin. Optimal impulsive control in periodic ecosystem. *Systems & Control Letters*, 55(7):558–565, 2006.
- [44] E. Yano. A simulation study of population interaction between the greenhouse whitefly *trialeurodes vaporarum* westwood (homoptera: Aleyrodidae), and the parasitoid *encarsia formosa* gahan (hymenoptera: Aphelinidae). i. description of the model. *Researches on Population Ecology*, 31:73–88, 1989.

- [45] E. Yano. A simulation study of population interaction between the greenhouse whitefly *trialeurodes vaporarium* westwood (homoptera: Aleyrodidae), and the parasitoid *encarsia formosa* gahan (hymenoptera: Aphelinidae). ii. simulation analysis of population dynamics and strategy of biological control. *Researches on Population Ecology*, 31:89–104, 1989.