An impulsively controlled predator–pest model with disease in the pest

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A B S T R A C T

In this paper, we consider an integrated pest management model with disease in the pest and a stage structure for its natural predator, which is subject to impulsive and periodic controls. A nonlinear incidence rate expressed in an abstract form, is used to describe the propagation of the disease, which is spread through the periodic release of infective pests, the functional response of the mature predator also being given in an abstract, unspecified form. Sufficient conditions for the local and global stability of the susceptible pest-eradication periodic solution are found by means of Floquet theory and comparison methods, the permanence of the system also being discussed. These stability conditions are shown to be biologically significant, being reformulated as balance conditions for the susceptible pest class.

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1. Introduction

The traditional approach to pest control relied on the seasonal use of chemical pesticides, the response to pest resurgence and pest outbreaks being an increase in the amount of pesticides sprayed, or in their toxicity. However, it has been observed that persistent pesticide use increases the incidence of pesticide-resistant pest varieties and inflicts harmful effects on humans through the accumulation of hazardous chemicals in their food chain. Further, pesticide pollution is also a major threat to beneficial insects, which are sometimes more affected by pesticide spraying than target pests.

Integrated pest management (IPM) represents a systemic, holistic approach to reduce pest damage to tolerable levels, without pesticide overuse. IPM strategies are conceived through an understanding of interactions between pests and the environment, relying on precise pest identification and on continuous field observation. Pest control techniques with lower environmental impact are then employed in order to minimize the damage caused to non-target organisms. Such techniques include mechanical methods (erecting pest barriers or using pest traps), biological methods (breeding natural predators of the pest, using biological insecticides such as Bacillus thuringiensis or entomopathogenic fungi, disrupting the reproductive processes of the pest by releasing sterile pest individuals or spreading a disease in the pest population on the grounds that infective pests are usually less damaging to the environment). Some examples of successful uses of biocontrol agents include the use of the predatory arthropod Orius sauteri against the pest Thrips palmi Karny to protect eggplant crops in greenhouses (Nagai and Yano [1]) and the use of the predatory mites Phytoseiulus persimilis and Neoseiulus californicus against the red spider mite Tranychusurticae Koch in field-grown strawberries (PortandScopes [2]). Baculoviruses have been found useful to control the diamondback moth Plutella xylostella in cabbage farms (Grzywacz et al. [3], Sarfraz, Keddie and Dosdall [4]), after it had been noticed that the diamondback moth became resistant to chemical pesticides in many areas (Roush [5]).
Pesticides may still be used in an IPM strategy as a last resort, since they are, in many cases, the quickest way to contain a pest outbreak, but the aim is to minimize their use, focusing on non-chemical controls instead, which are often more cost-effective and self-sustainable.

Zero-tolerance policies may not be necessary for every pest, a key concept in this direction being the economic injury level (EIL), which is defined in Stern et al. [6] as the lowest pest density which causes economic damage. In this regard, the ultimate purpose of an IPM strategy is often not to drive the pest population to extinction, as this may not be cost-effective or may damage the ecosystem, but to stabilize the pest population under EIL. A related threshold parameter is the economic threshold (ET), usually defined as the lowest pest density at which control measures should be taken so that EIL is not exceeded. Consequently, ET has a lower value than EIL (Tang and Cheke [7]).

The inherent discontinuity of human activities and the steep variation in the size of the pest population which occurs immediately after successful control measures are implemented (releasing natural enemies of the pest, spraying pesticides, releasing infective pest individuals) may be described mathematically through the use of impulsive controls. These controls may be employed according to a time-based strategy (time-dependent controls) or as soon as the size of the pest population reaches ET (state-dependent controls).

The life cycle of many species has been found to consist in at least two stages, immature and mature, with significant morphological and behavioral differences between them. To account for these differences, stage-structured models have been proposed and analyzed in recent years. See Aiello and Freedman [8] for a single species model with a stage structure and time delay, Wang [9] or Xiao and Chen [10] for a predator–prey model with a stage structure for the predator which is treated via the theory of competitive systems, Kuang [11] or Arditi and Michalski [12] for general consistency criteria to be satisfied by models describing stage-structured interactions, Liu, Chen and Agarwal [13] for a survey on the dynamics of stage-structured population models with an emphasis on modelling issues.

Recently, many papers have been devoted to the analysis of mathematical models describing IPM strategies. See, for instance, Tang and Cheke [14], Tang et al. [15] for predator–pest models which are impulsively controlled by means of state-dependent controls, Liu, Zhi, Chen [16], Liu, Chen, Zhang [17], Su et al. [18] for predator–pest models which are impulsively controlled by means of time-dependent controls, Georgescu and Moroşanu [19] for an SI model which is subject to impulsive biological and chemical controls, Xiao and van den Bosch [20] for an SI model with additional food resource. See also Tan and Chen [7] for another SI model with additional food resource, Du et al. [21] for a predator–pest model with a stage structure for the pest, Zhang, Chen and Nieto [22] for a single species epidemic model with stage structure, these models being subject to time-dependent impulsive controls. However, very few papers have been devoted to models which combine the release of natural predators of the pest, and of infective pest individuals. See Shi and Chen [23] for a 3-dimensional predator–pest model with disease in the pest which features a bilinear rate of incidence and time-dependent impulsive controls. The approach to biological control which we employ in our paper is to release both infective pest individuals and natural predators of the given pest, in periodic pulses of a constant amount.

As far as disease transmission is concerned, bilinear and standard incidence rates have often been used in epidemic models. See, for instance, Li, Smith and Wang [24], Ebert, Lipsitch and Mangin [25], Tudor [26], Rost and Wu [27] for models with bilinear incidence rates and Hethcote et al. [28], Arino et al. [29], De Leenheer and Smith [30], Gourley, Kuang and Nagy [31] for models with standard incidence rates. Since the number of contacts between infectives and susceptibles may saturate at higher densities of infective individuals due to crowding, and since multiple contacts may be required in certain situations for disease transmission, these incidence rates may not necessarily describe accurately the spread of the disease, an incidence rate of type $g(I)S$ with $g(I) = \frac{KI}{1+\alpha I}$ being proposed in Capasso and Serio [32]. A more general incidence rate of this type used in literature is the one for which $g(I) = \frac{K\alpha I}{1+\alpha I}$ (Ruan and Wang [33], Liu, Levin and Iwasa [34]), where $I$ measures the infection force of the disease and $\frac{1}{1+\alpha I}$ measures the inhibitory effect caused by behavioral changes. Note that if $g(I)$ is decreasing when $I$ is large, this may be interpreted as the fact that susceptibles tend to reduce their social contacts if the perceived number of infectives increases over a psychologically significant value. Very general incidence rates which are not linear in $S$ are also used in Derrick and van den Driessche [35] $(g(S, I, N) = I\Phi(S, I, N)$, where $N = I + S$), Korobeinikov and Maini [36] $(g(S, I) = h_1(I)h_2(S))$, Moghadas and Alexander [37] $(g(S, I) = \beta(1 + f(I; v))IS)$.

The interaction between pests and their natural predators fits the general framework of a predator–prey interaction. In this regard, an abstract predation term of type $g(I)S$ with $g(I)$ being proposed by Yodzis [38]. Here, $F(N, P)$ is the functional response of the predator, which represents the number of prey individuals consumed by a single predator per unit area and unit time. Functional responses which depend only on the size of the prey population $F(N, P) = h(N)$ have been termed as prey-dependent in Arditi and Ginzburg [39], while functional responses which depend also on the size of the predator population $P$ have been termed as predator-dependent, or in the particular case in which $F(N, P) = h_1(I)/N$, as ratio-dependent. Particular examples of prey-dependent functional responses are $h(N) = bN$ (Holling type I, Holling [40]), $h(N) = \frac{\alpha N}{1+bN}$ (Holling type II, Holling [40]), $h(N) = \frac{\alpha N}{1+bN^2}$ (Holling type III, Holling [40]), $h(N) = k(1-e^{-\alpha N})$ (Ivlev [41]).

Differential models relying on the use of impulsively perturbed ordinary differential equations, provide an adequate framework for many evolutionary processes. In this regard, a biological resource management model of predator–prey type with continuous harvesting on predator and impulsive stocking on prey, has been considered in Jiao et al. [42] from the viewpoint of finding a reasonable harvesting strategy. The model discussed in [42] also features a stage structure for the
The existence of periodic solutions for ratio-dependent predator–prey models has been investigated by Wang, Shen and Nieto in [43] and by Ding, Lu and Liu in [44] by means of coincidence degree theory, the latter including a delay term due to the negative feedback of the prey population. Since environmental parameters are naturally subject to fluctuation in time, the periodicity of the functional coefficients which appear in the models employed in [43,44] represent a way to incorporate the periodicity of the environment. Also, the existence of periodic solutions for a single–species Lotka–Volterra model with multiple delays and impulsive perturbations, has been investigated by Yan, Zhao and Nieto in [45], where it has been observed that, under appropriate linear periodic impulsive perturbations, the perturbed systems retain the original periodicity and global attractivity of the unperturbed system. Similar results have been obtained by Meng, Chen and Li [46] for an impulsively perturbed nonautonomous predator–prey model with multiple delays. A discrete host–parasitoid model which involves parasitoid intergenerational survival rates and periodic impulsive perturbations, extending the classical Nicholson and Bailey model, has been analyzed in Tang, Xiao and Cheke [47] and it is found that, under appropriate conditions, there are stable periodic solutions such that the maximal size of the host population does not exceed a pre-specified threshold level. The optimal control of a three-dimensional food chain via biological and chemical controls of a discontinuous nature, has been studied in Apreutesei [48] using the Pontrjagin maximality principle, the purpose being to maximize the total population size at the end of a given interval [0, T]. A similar analysis has been performed in Apreutesei and Dumitriu [49] for a Lotka–Volterra model which is subject to a biological control consisting of the discontinuous (but not impulsive) release of a top predator.

A theoretical study of superficial bladder cancer growth and of its treatment via pulsed immunotherapy with Bacillus Calmette-Guérin has been studied in Buninovich-Mendratzitsky, Byrne and Stone [50], with the purpose of finding appropriate BCG instillation doses and rates of pulsing for successful treatment. The dynamics of a SIR model with pulsed vaccination (the repeated application of vaccine over a defined age range) has been studied by Gao et al. in [51], where it has been proved that the infection-free periodic solution is globally attractive, provided that the vaccination rate is large enough. A related stability analysis has also been performed by Xiang, Li and Song in [52] for a nondelayed SIR model and by Jiao, Meng and Chen in [53] for a stage-structured SI model.

The purpose of this paper is to construct an integrated pest management model which relies on the simultaneous periodic release of infective pest individuals and of natural predators, in a constant amount. An abstract incidence rate of type $g(I)S$ is employed to model the spread of the disease which is propagated through the release of infective individuals, the predation of the susceptibles being also modeled by an abstract prey-dependent functional response $h(S)$, under a few biologically feasible assumptions upon $g$ and $h$. A related predator–pest model of IPM has been discussed by Zhang, Georgescu and Chen in [54]. The model discussed in [54] features a predator-dependent functional response in a particular form, as opposed to our prey-dependent functional response, and two distinct sets of impulsive controls corresponding to periodic pesticide spraying and periodic predators release, respectively. Comparatively, our model adds a stage structure for the predator, a feature which does not appear in [54], and accounts for the use of a biocontrol agent, consisting of the periodic release of infective pests, which is again not employed in [54], while also discussing stability and permanence problems which are related to those treated in [54].

The rest of this paper is organized as follows: in Section 2, the main biological assumption on which the model relies are formulated and the resulting impulsively perturbed differential model is introduced. In Section 3, the biological well-posedness of the model is established and certain comparison and boundedness results for impulsive differential equations are introduced, together with basic elements of Floquet stability. In Section 4, sufficient conditions for the local and global stability of the susceptible pest-eradication periodic solution are established, their biological significance also being discussed. In Section 5, the permanence of our system is analyzed. Further comments on the biological relevance of our results are stated in Section 6, together with a few concluding remarks.

2. The model

In the following, we shall denote by $S$ the size of the susceptible pest population, by $I$ the size of the infective pest population, by $P_I$ the size of the immature predator population and by $P_M$ the size of the mature predator population. To derive our mathematical model, the following assumptions are made.

(A1) The pests are either susceptible or infective. The disease is transmitted from infective pests to susceptible pests and does not propagate to predators. Also, the disease is not vertically transmitted from infective pests to their offspring.

(A2) In the absence of infection, the intrinsic growth rate of the susceptible pest population is described by the logistic function with intrinsic birth rate $r$ and carrying capacity $K$.

(A3) The infective pests neither recover nor reproduce. They also do not damage crops or otherwise contribute to the total size of the environment–supported pest population.

(A4) The incidence rate of the infection is nonlinear in $I$ and given by $g(I)S$, where $g$ is an abstract function satisfying certain assumptions outlined below.

(A5) The life cycle of the predator consists in two stages, immature and mature, only the mature predators having the ability to hunt for prey and reproduce.

(A6) The functional response of the mature predator is described by the abstract function $h$ satisfying certain assumptions outlined below.
(A7) Predators feed on susceptible pests only, and do not prey upon infective pests.

(A8) Predators and infective pests are released in an impulsive and periodic fashion, simultaneously and in the same amount each time.

On the basis of the above assumptions, we formulate the following impulsively perturbed model with disease in the pest and a stage structure for the predator, which describes the dynamics of the predator–pest interaction under consideration, as follows.

\[
\begin{align*}
S'(t) &= rS(t) \left(1 - \frac{S(t)}{K}\right) - g(I(t))S(t) - P_M(t)h(S(t)), & t \neq nT; \\
I'(t) &= g(I(t))S(t) - d_1I(t), & t \neq nT; \\
P_1'(t) &= cP_1(t)h(S(t)) - (m + d_1)P_1(t), & t \neq nT; \\
P_M'(t) &= mP_1(t) - d_MP_M(t), & t \neq nT; \\
\Delta S(t) &= 0, & t = nT; \\
\Delta I(t) &= \delta_1, & t = nT; \\
\Delta P_1(t) &= \delta_1, & t = nT; \\
\Delta P_M(t) &= \delta_M, & t = nT.
\end{align*}
\]  

(1)

Here, \(d_1, d_1, d_M > 0\) are the death rates of the infective pest population and of the immature and mature predator population, respectively, \(\frac{1}{T}\) is the mean length of the immature stage and \(c > 0\) is a proportionality constant relating the reproductive rate of the mature predators to the amount of prey consumed. The constants \(\delta_1, \delta_1, \delta_M > 0\) represent the amount of infective pests, immature and mature predators, respectively, which are released each time and \(T > 0\) is the release periodicity. Also, \(\Delta \varphi(t) = \varphi(t + h) - \varphi(t)\) for \(\varphi \in \{S, I, P, P_M\}, n \in \mathbb{N}^+,\) and \(g, h \in \mathcal{H}\), where

\[\mathcal{H} = \left\{f : \mathbb{R} \to \mathbb{R}; f(0) = 0, f'(x) > 0 \text{ and } f''(x) \leq 0 \text{ for all } x > 0 \right\}.\]

In these settings, the success of the IPM strategy will mostly be expressed in terms of stability properties for the susceptible pest-eradication periodic solution. The global stability of the susceptible pest-eradication periodic solution means that the IPM strategy is successful, irrespective of the initial sizes of the pest and predator populations, while its local stability means that the IPM strategy is successful under favorable circumstances. Strictly speaking, the IPM strategy is considered successful when the size of the susceptible predator population stabilizes under the EIL, but this persistence level cannot be determined using our approach.

It is to be noted that our model can also describe some situations apparently not covered by (1). Specifically, if the infective pests do contribute towards the carrying capacity of the environment by damaging crops or by other means, then the first equation in our model may be substituted by

\[S'(t) = rS(t) \left(1 - \frac{S(t)}{K} - \frac{\alpha I(t)}{K}\right) - g(I(t))S(t) - P_M(t)h(S(t)),\]

\(\alpha\) being a constant which characterizes the fact that susceptible and infective pests have different capabilities to damage the environment. This equation may be restated as

\[S'(t) = rS(t) \left(1 - \frac{S(t)}{K} - \frac{\alpha I(t)}{K}\right) - g(I(t)) + \frac{\alpha \rho(I)}{K} S(t) - P_M(t)h(S(t)),\]

which again fits our framework, for \(\tilde{g}(l) = g(l) + \alpha \rho(l)\), although this time the limiting size of the infective pest population is also of concern. Specifically, the global stability of the susceptible pest-eradication periodic solution would not suffice, another requirement being that the average endemicity level be lower than a certain value which is determined knowing the value of the EIL.

3. Preliminaries

In this section we shall introduce a few definitions and notations, together with some basic notions regarding Floquet theory of impulsive differential equations and comparison estimations. A few properties of the periodic solution of a certain impulsively perturbed differential equation will be determined, these findings being used in the next section to establish the existence of the susceptible pest-eradication periodic solution of (1).

Let \(\mathcal{V}_0\) be the set of functions \(V : \mathbb{R}_+ \times \mathbb{R}_+^4 \to \mathbb{R}_+\) which are locally Lipschitz in the second variable, continuous on \((nT, (n + 1)T] \times \mathbb{R}_+^4\) and for which the limits \(\lim_{t \to (nT+, x)} V(t, y) = V(nT+, x)\) exist and are finite for \(x \in \mathbb{R}_+^4\) and \(n \in \mathbb{N^+}\).

For \(V \in \mathcal{V}_0\), we define the upper right Dini derivative of \(V\) with respect to the system (1) at \((t, x) \in (nT, (n + 1)T] \times \mathbb{R}_+^4\) by

\[D^+ V(t, x) = \limsup_{h \to 0} \frac{1}{h} \left[ V(t + h, x + hf(t, x)) - V(t, x) \right].\]
We now indicate a comparison result for solutions of impulsive differential inequalities which allows us to estimate the values of the solutions of (1). We suppose that \( a : \mathbb{R}_+ \times \mathbb{R}_+ \rightarrow \mathbb{R} \) satisfies the following hypotheses.

\((H)\) \( a \) is continuous on \( (nT, (n + 1)T] \times \mathbb{R}_+ \) and the limits \( \lim_{t \rightarrow (nT+, 0)} a(t, y) = a(nT+, x) \) exist and are finite for \( x \in \mathbb{R}_+ \) and \( n \in \mathbb{N}^* \).

**Lemma 3.1** ([55]). Let \( V \in \mathcal{V}_0 \) and assume that

\[
\begin{align*}
D^+ V(t, x(t)) &\leq a(t, V(t, x(t))), & t \neq nT; \\
V(t, x(t+)) &\leq \psi^1_n(V(t, x(t))), & t = nT,
\end{align*}
\]

where \( a : \mathbb{R}_+ \times \mathbb{R}_+ \rightarrow \mathbb{R} \) satisfies \((H)\) and \( \psi^1_n : \mathbb{R}_+ \rightarrow \mathbb{R}_+ \) are nondecreasing for all \( n \in \mathbb{N}^* \). Let \( r(t) \) be the maximal solution of the impulsive Cauchy problem

\[
\begin{align*}
u'(t) &= a(t, u(t)), & t \neq nT; \\
u(t+) &= \psi^1_n(u(t)), & t = nT; \\
u(0+) &= u_0
\end{align*}
\]

defined on \([0, \infty)\). Then \( V(0+, x_0) \leq u_0 \) implies that \( V(t, x(t)) \leq r(t) \) for all \( t \geq 0 \), where \( x(t) \) is an arbitrary solution of (2).

Note that, under appropriate regularity conditions, the Cauchy problem (3) has a unique solution and, in that case, the comparison function \( r \) becomes the unique solution of (3). We now indicate a result which provides boundedness estimations for the solution of a system of differential inequalities.

**Lemma 3.2** ([55]). Let the function \( u \in PC^{1}(\mathbb{R}_+, \mathbb{R}) \) satisfy the inequalities

\[
\begin{align*}
u'(t) &\geq p(t)u(t) + f(t), & t \neq \tau_k, & t > 0; \\
u(\tau_k+) &\geq d_ku(\tau_k) + h_k, & k \geq 0; \\
u(0+) &\geq u_0,
\end{align*}
\]

where \( p, f \in PC(\mathbb{R}_+, \mathbb{R}), d_k \geq 0, h_k \) and \( u_0 \) are constants and \( (\tau_k)_{k \geq 0} \) is a strictly increasing sequence of positive real numbers. Then, for \( t > 0 \),

\[
u(t) \geq u_0 \left( \prod_{0 < \tau_k < t} d_k \right) e^{\int_0^t p(s)ds} + \int_0^t \left( \prod_{0 < \tau_j < s} d_j \right) e^{\int_0^s p(r)dr} f(s)ds + \sum_{0 < \tau_k < t} \left( \prod_{\tau_{k'} < \tau_k} d_j \right) e^{\int_{\tau_{k'}}^{\tau_k} p(r)dr} h_k.
\]

In the above, \( PC(\mathbb{R}_+, \mathbb{R}) \) \( (PC^{1}(\mathbb{R}_+, \mathbb{R})) \) denotes the class of real piecewise continuous \( (\text{real piecewise continuously differentiable}) \) functions defined on \( \mathbb{R}_+ \). For other results on impulsive differential equations, see Bainov and Simeonov [55].

First, it is easy to see that (1) has a unique solution for every initial set of data. Using **Lemma 3.2**, it is now possible to prove that all solutions of (1), starting with strictly positive initial data, remain strictly positive and bounded on their whole domains of existence.

**Lemma 3.3.** The set \( (\mathbb{R}_+^*)^4 \) is an invariant region for the system (1).

**Proof.** Let us consider \( X = (S, I, P_j, P_M) : [0, T_0) \rightarrow \mathbb{R}^4 \) a solution of (1) defined on its maximal interval of existence and starting with strictly positive \( S(0), I(0), P_j(0), P_M(0) \). It follows that

\[
\begin{align*}
S'(t) &\geq S(t) \left( 1 - \frac{S(t)}{K} \right) - g(I(t)) - P_M(t)h'(0), & t \neq nT; \\
I'(t) &\geq -d_1I(t), & t \neq nT; \\
P_j'(t) &\geq -(m + d_j)P_j(t), & t \neq nT; \\
P_M'(t) &\geq -d_M P_M(t), & t \neq nT
\end{align*}
\]

as long as \( X \) remains positive component-wise, since \( h \in \mathcal{H} \), which implies that \( h(x) \leq h'(0)x \) for all \( x \geq 0 \). By integrating the above inequalities and accounting for the effect of impulsive perturbations which occur at \( t = nT \), one obtains that

\[
\begin{align*}
S(t) &\geq S(0) e^{\int_0^t p_1(s)ds} \\
I(t+) &\geq I(0)e^{-d_1(1 + \delta_1)\frac{t}{4}} \\
P_j(t+) &\geq P_j(0)e^{-\left(m + d_j\right)(1 + \delta_j)\frac{t}{4}} \\
P_M(t+) &\geq P_M(0)e^{-d_M(1 + \delta_M)\frac{t}{4}}
\end{align*}
\]

where

\[
p_1(t) = r \left( 1 - \frac{S(t)}{K} \right) - g(I(t)) - P_M(t)h'(0),
\]

on the interval on which \( X \) stays positive component-wise, so \( X \) is actually strictly positive on \([0, T_0)\). \( \square \)
Using the above positivity result, we may now show that all solutions of (1) are bounded and actually defined on $\mathbb{R}_+$. 

**Lemma 3.4.** All solutions of (1) starting in $(\mathbb{R}_+^4)^4$ are bounded and defined on $\mathbb{R}_+$. 

**Proof.** Let us consider a solution $(S(\cdot), I(\cdot), P_j(\cdot), P_M(\cdot))$ of (1) starting with initial data $(S(0), I(0), P_j(0), P_M(0)) \in (\mathbb{R}_+^4)^4$ and define $u_1 : \mathbb{R}_+ \to \mathbb{R}_+$ by 

$$u_1(t) = cS(t) + cl(t) + P_j(t) + P_M(t).$$

One then sees that 

$$u'_1(t) = rcS(t) \left(1 - \frac{S(t)}{K}\right) - cdI(t) - djP_j(t) - dMP_M(t), \quad t \neq nT.$$ 

Let us denote $D = \min(cd_1, dj, d_M)$. It follows that 

$$u'_1(t) + Du_1(t) \leq (rc + D)S(t) - \frac{rc}{K}S^2(t), \quad t \neq nT.$$ 

(5) 

Since the right-hand side of (5) is bounded from above by $C = \frac{K(rc + D)^2}{4rc}$, it follows that 

$$u'_1(t) + Du_1(t) \leq C, \quad t \neq nT,$$ 

together with 

$$u_1(nT^+) = u_1(nT) + c\delta_i + \delta_j + \delta_M.$$ 

By Lemma 3.2, it follows that 

$$u_1(t) \leq u_1(0+)e^{-Dr} + C\int_0^t e^{-D(t-s)} ds + \sum_{0 < nT < t} (c\delta_i + \delta_j + \delta_M)e^{-D(t-nT)}, \quad t > 0,$$ 

(6) 

which yields 

$$u_1(t) \leq u_1(0+)e^{-Dr} + \frac{C(1 - e^{-Dt})}{D} + (c\delta_i + \delta_j + \delta_M)e^{DT}e^{-DT} - 1, \quad t > 0.$$ 

(7) 

Since the limit for $t \to \infty$ of the right-hand side of (7) is 

$$L = \frac{C}{D} + (c\delta_i + \delta_j + \delta_M)e^{DT}e^{-DT} - 1 < \infty,$$ 

it follows that $u_1$ is bounded on its domain. Consequently, $S, I, P_j, P_M$ are bounded and it follows, by an easy continuability argument, that they are defined on the whole $\mathbb{R}_+$. 

We now introduce a few basic results regarding the Floquet theory for impulsive systems of ordinary differential equations, which will be used in the next section to discuss the local stability of the susceptible pest-eradication periodic solution. Let us consider the impulsive linear system 

$$\begin{cases}
X'(t) = A(t)X(t), & t \neq \tau_k, \ t \in \mathbb{R}; \\
\Delta X = B_kX, & t = \tau_k, \ \tau_k < \tau_{k+1}, \ k \in \mathbb{Z}
\end{cases}$$  

(8) 

under the following hypotheses.

(H1) $A(\cdot) \in PC(\mathbb{R}, M_n(\mathbb{R}))$ and there is $T > 0$ such that $A(t + T) = A(t)$ for all $t \geq 0$.

(H2) $B_k \in M_n(\mathbb{R})$, det$(I_n + B_k) \neq 0$ for $k \in \mathbb{Z}$.

(H3) There is $q \in \mathbb{N}^*$ such that $B_{k+q} = B_k, \tau_{k+q} = \tau_k + T$ for $k \in \mathbb{Z}$.

Let $\Phi(t)$ be a fundamental matrix of $X' = AX$. Then there is a unique nonsingular matrix $M \in M_n(\mathbb{R})$ such that $\Phi(t + T) = \Phi(t)M$ for all $t \in \mathbb{R}$, which is called the monodromy matrix of (8) corresponding to $\Phi$. Actually, all monodromy matrices of (8) are similar and consequently they have the same eigenvalues $\lambda_1, \lambda_2, \ldots, \lambda_n$, which are called the Floquet multipliers of (8). Under these hypotheses, the following stability result holds, where by elementary divisors of a square matrix $A$ we understand the characteristic polynomials of its Jordan blocks. 

**Lemma 3.5 ([55]).** Suppose that conditions (H1)–(H3) hold. Then

1. The system (8) is stable if, and only if, all Floquet multipliers $\lambda_k, 1 \leq k \leq n$ satisfy $|\lambda_k| \leq 1$ and if $|\lambda_k| = 1$, then to $\lambda_k$ there corresponds a simple elementary divisor.

2. The system (8) is asymptotically stable if, and only if, all Floquet multipliers $\lambda_k, 1 \leq k \leq n$ satisfy $|\lambda_k| < 1$.

3. The system (8) is unstable if there is a Floquet multiplier $\lambda_k$ such that $|\lambda_k| > 1$. 


To discuss the dynamics of the system (1) in the absence of susceptible pests, it becomes necessary to consider the properties of the system

\[
\begin{aligned}
    z'(t) &= a(t) - dz(t), & t &\neq nT; \\
    \Delta z(t) &= \delta, & t &= nT; \\
    z(0+) &= z_0,
\end{aligned}
\]

(9)
a being a T-periodic \( PC(\mathbb{R}_+, \mathbb{R}) \) function and \( d \) being a positive real constant. It will be seen that the system consisting in the first two equations in (9) has a T-periodic solution to which all solutions of (9) starting with strictly positive \( z_0 \) tend as \( t \to \infty \). The above-mentioned periodic solution will be labeled in what follows as \( z^*_{a,d,\delta} \).

**Lemma 3.6.** The system consisting of the first two equations in (9) has a T-periodic solution \( z^*_{a,d,\delta} \). With this notation, the following properties are satisfied.

1. \( z^*_{0,d,0} \equiv 0 \).
2. \( \int_0^T z^*_{a,d,\delta}(t) \, dt = \frac{1}{d(1 - e^{-dT})} \left( \delta + \int_0^T a(s) \, ds \right) \).
3. \( \lim_{t \to \infty} \left| z^*_{a,d,\delta}(t) - z_{a,d,\delta}(t) \right| = 0 \) for all solutions \( z_{a,d,\delta} \) of (9) starting with strictly positive initial data.
4. \( \sup_{t > 0} \left| z_{a;1,d,\delta}(t) - z^*_{a;2,d,\delta}(t) \right| \leq \left( 1 + \frac{T}{1 - e^{-dT}} \right) \sup_{t \in [0,T]} |a_1(t) - a_2(t)| \).

**Proof.** First, it is easy to see that

\[
z(t) = e^{-dt} \left( z(0+) + \int_0^t a(s)e^{ds} \, ds \right), \quad t \in (0, T]
\]

for any solution \( z \) of (9). The T-periodicity requirement for \( z^*_{a,d,\delta} \) then reads as

\[
e^{-dt} \left( z^*_{a,d,\delta}(0+) + \int_0^T a(s)e^{ds} \, ds \right) + \delta = z^*_{a,d,\delta}(0+)
\]

(10)
which implies that

\[
z^*_{a,d,\delta}(0+) = \frac{e^{-dt} \int_0^T a(s)e^{ds} \, ds + \delta}{1 - e^{-dT}}.
\]

(11)
Consequently, there is a unique T-periodic solution \( z^*_{a,d,\delta} \) of (9), given by

\[
z^*_{a,d,\delta}(t) = \frac{e^{-dt} \left( z^*_{a,d,\delta}(0+) + \int_0^{t-nT} a(s)e^{ds} \, ds \right)}{1 - e^{-dT}}, \quad t \in (nT, (n + 1)T].
\]

(12)
z^*_{a,d,\delta}(0+) being defined by (11). From (11) and (12), it then easily follows that (1) and (2) hold. Let us now prove (3). To this purpose, let \( z_{a,d,\delta}(t) \) be a solution of (9) with strictly positive initial data. It is seen that \( Z = z_{a,d,\delta} - z^*_{a,d,\delta} \) verifies the system

\[
\begin{aligned}
    Z'(t) &= -dZ(t), & t &\neq nT; \\
    \Delta Z(t) &= 0, & t &= nT; \\
    Z(0+) &= z_{a,d,\delta}(0+) - z^*_{a,d,\delta}(0+)
\end{aligned}
\]

and consequently

\[
Z(t) = e^{-dt}Z(0+), \quad \text{for } t > 0,
\]

from which one easily obtains that (3) holds. To derive (4), let us define

\[
W = z^*_{a;1,d,\delta} - z^*_{a;2,d,\delta}.
\]

It is then seen that \( W \) verifies the system

\[
\begin{aligned}
    W'(t) &= (a_1(t) - a_2(t)) - dW(t), & t &\neq nT; \\
    \Delta W(t) &= 0, & t &= nT; \\
    W(0+) &= \frac{e^{-dT} \int_0^T (a_1(s) - a_2(s))e^{ds} \, ds}{1 - e^{-dT}}
\end{aligned}
\]

and consequently

\[
W(t) = e^{-dt} \left( W(0+) + \int_0^t (a_1(s) - a_2(s))e^{ds} \, ds \right), \quad \text{for } t > 0.
\]
This implies that
\[ |W(t)| \leq |W(0+)| + \sup_{t \in [0,T]} |a_1(t) - a_2(t)| \]
from which (4) easily follows. \( \square \)

4. Local and global stability results

First of all, it is seen that the long-term survival of the infective pest population and of the immature and mature predator populations is assured by the pulsed supply of individuals which occurs for \( t = nT, n \in \mathbb{N}^* \), while the susceptible pest population may be driven to extinction in certain circumstances, since a pulsed supply of susceptibles is not present. Also, at least when the susceptible pest population (the only one which is not impulsively controlled) tends to extinction, it is natural to expect that the solutions of (1) tend to a limiting periodic solution due to the forcing effects of the periodic impulsive perturbations.

Using Lemma 3.6, we are able to study the existence and stability of the susceptible pest-eradication periodic solution \((0, I^*, P^*_j, P^*_M)\). To this purpose, it is seen first that when \( S = 0 \), the system (1) reduces to

\[
\begin{align*}
I'(t) &= -d_I(t), \quad t \neq nT; \\
P^*_j(t) &= -(m + d_j)P^*_j(t), \quad t \neq nT; \\
P^*_M(t) &= mP^*_j(t) - d_MP^*_M(t), \quad t \neq nT; \\
\Delta I(t) &= \delta_I, \quad t = nT; \\
\Delta P^*_j(t) &= \delta_J, \quad t = nT; \\
\Delta P^*_M(t) &= \delta_M, \quad t = nT;
\end{align*}
\]

which describes the dynamics of the system in the absence of the susceptible pest population. It then easily follows through the use of Lemma 3.6 for \( a \equiv 0 \) and \((d, \delta) = (\delta_I, \delta_J), (d, \delta) = (m + d_j, \delta_I)\) respectively, that there are \( I^* \) and \( P^*_j \) which verify the first and the fourth equation of (13) and the second and the fifth equation of (13), respectively, together with the \( T \)-periodicity condition. The existence of \( P^*_M \) which verifies the third and sixth equation of (13) together with the \( T \)-periodicity condition, follows again from Lemma 3.6, for \( a = mP^*_j \) and \((d, \delta) = (d_M, \delta_M)\). That is, with the notations of Lemma 3.6,

\[
I^* = z_{0; d_I, \delta_I}^0, \quad P^*_j = z_{0; m+d_j, \delta_J}^*, \quad P^*_M = z_{nT; d_M, \delta_M}^{nT}.
\]

Furthermore, we may compute explicitly \( I^*, P^*_j \) and \( P^*_M \). It is seen that

\[
\begin{align*}
I^*(t) &= \frac{\delta_I}{1 - e^{-d_I t}} e^{-d_I (t - nT)}, \quad t \in (nT, (n + 1)T]; \\
P^*_j(t) &= \frac{\delta_J}{1 - e^{-(m+d_j)T}} e^{-(m+d_j)(t-nT)}, \quad t \in (nT, (n + 1)T]; \\
P^*_M(t) &= e^{-d_M(t-nT)} \left( P^*_M(0+) + \frac{m\delta_J}{1 - e^{-(m+d_j)T}} A(t - nT) \right), \quad t \in (nT, (n + 1)T],
\end{align*}
\]

where

\[
A(t) = \begin{cases} 
  t, & d_M = m + d_j; \\
  \frac{e^{(d_M-(m+d_j))T} - 1}{d_M - (m + d_j)}, & d_M \neq m + d_j 
\end{cases}
\]

and

\[
P^*_M(0+) = \frac{1}{1 - e^{-d_M T}} \left[ e^{-d_M T} \frac{m\delta_J}{1 - e^{-(m+d_j)T}} A(T) + \delta_M \right].
\]

Having proven the existence of the susceptible pest-eradication periodic solution \((0, I^*, P^*_j, P^*_M)\), we are now ready to study its stability. In what follows, it will be observed that the susceptible pest-eradication periodic solution is locally asymptotically stable, provided that the reproductive potential of the susceptible pest at small densities is less than a certain value.

**Theorem 4.1.** The susceptible pest-eradication periodic solution \((0, I^*, P^*_j, P^*_M)\) is locally asymptotically stable, provided that

\[
rT \leq \int_0^T g(I^*(t)) \, dt + h'(0) \int_0^T P^*_M(t) \, dt
\]

and unstable, provided that the reverse inequality holds.
Proof. To study the stability of the susceptible pest-eradication periodic solution \((0, I^*, P^*_J, P^*_M)\), let us denote

\[
S(t) = x(t), \quad I(t) = y(t) + I^*(t), \quad P_J(t) = z_1(t) + P^*_J(t), \quad P_M(t) = z_2(t) + P^*_M(t),
\]

with \(x, y, z_1, z_2\) being understood as small amplitude perturbations. Substituting (16) into the first four equations of (1), one obtains

\[
\begin{aligned}
\dot{x}(t) &= rx(t) \left(1 - \frac{x(t)}{K}\right) - g(y(t) + I^*(t))x(t) - (z_2(t) + P^*_M(t))h(x(t)), \quad t \neq nT; \\
\dot{y}(t) &= g(y(t) + I^*(t))x(t) - d_I y(t), \quad t \neq nT; \\
\dot{z}_1(t) &= c(z_2(t) + P^*_M(t))h(x(t)) - (m + d_J)z_1(t), \quad t \neq nT; \\
\dot{z}_2(t) &= mz_1(t) - d_M z_2(t), \quad t \neq nT.
\end{aligned}
\]

By linearizing (17) around \((0, 0, 0, 0)\), one obtains

\[
\begin{aligned}
\dot{x}(t) &= rx(t) - g(I^*(t))x(t) - P^*_M(t)h'(0)x(t), \quad t \neq nT; \\
\dot{y}(t) &= g(I^*(t))x(t) - d_I y(t), \quad t \neq nT; \\
\dot{z}_1(t) &= cP^*_M(t)h'(0)x(t) - (m + d_J)z_1(t), \quad t \neq nT; \\
\dot{z}_2(t) &= mz_1(t) - d_M z_2(t), \quad t \neq nT.
\end{aligned}
\]

The corresponding linearization of the jump conditions reads as

\[
\Delta x(t) = \Delta y(t) = \Delta z_1(t) = \Delta z_2(t) = 0, \quad t = nT,
\]

and so a fundamental matrix \(\Phi_t\) of (18) verifies

\[
\frac{d\Phi_t}{dt} = \begin{pmatrix}
1 & 0 & 0 & 0 \\
0 & \frac{r - (g(I^*(t)))}{g(I^*(t))} & 0 & 0 \\
0 & -d_I & 0 & 0 \\
0 & cP^*_M(t)h'(0) & 0 & -(m + d_J)
\end{pmatrix} \Phi_t(t).
\]

Consequently, a fundamental matrix \(\Phi_t\) of (18) is

\[
\Phi_t = \begin{pmatrix}
\Phi_{21}(t) & 0 & 0 & 0 \\
0 & e^{-d_I t} & 0 & 0 \\
0 & 0 & e^{-(m + d_J)T} & 0 \\
0 & 0 & 0 & e^{-d_M t}
\end{pmatrix},
\]

where

\[
\begin{aligned}
\Phi_{21}(t) &= e^{-d_I t} \int_0^t g(I^*(s))e^{d_I s}e^{-\int_0^t \frac{r - (g(I^*(r)))}{g(I^*(r))} \beta^*_M(t) \, dr} \, ds, \\
\Phi_{31}(t) &= e^{-(m + d_J)T} \int_0^t cP^*_M(t)h'(0)e^{\int_0^t \frac{r - (g(I^*(r)))}{g(I^*(r))} \beta^*_M(t) \, dr} e^{(m + d_J)T} \, ds, \\
\Phi_{41}(t) &= e^{-d_M t} \int_0^t me^{d_M s} \beta_{31}(s) \, ds, \\
\Phi_{43}(t) &= e^{-d_M t} \int_0^t me^{d_M s} e^{-(m + d_J)T} \, ds.
\end{aligned}
\]

As seen in Lemma 3.5, one may discuss the local stability of the susceptible pest-eradication periodic solution by analyzing the eigenvalues of the monodromy matrix \(M = \Phi_T(T)\). Note that, as seen in (19), the impulsive controls do not affect the linearized system.

Since the eigenvalues of \(M\) are

\[
\lambda_1 = e^{rT} \left( \int_0^T g(I^*(t)) \, dt \right) \beta^*_M(t) \, dt, \quad \lambda_2 = e^{-d_I T}, \quad \lambda_3 = e^{-(m + d_J)T}, \quad \lambda_4 = e^{-d_M T},
\]

and \(0 < \lambda_2, \lambda_3, \lambda_4 < 1\), it follows that the susceptible pest-eradication periodic solution is locally stable provided that (15) is satisfied, and unstable provided that the reverse inequality holds, which finishes the proof. □

We shall now observe that (15) can actually be rephrased as a balance condition for the susceptible pest class. Namely, let us suppose that \(S\) approaches 0, and consequently that \(I\) approaches \(I^*\), \(P_J\) approaches \(P^*_J\) and \(P_M\) approaches \(P^*_M\). Then \(rT\) approximates the normalized (per-susceptible) number of newborn susceptible pests in a period \(T\), while \(\int_0^T g(I^*(t)) \, dt\)
approximates the normalized loss of susceptible pests in a period due to their movement to the infective class and \( h'(0) \int_0^T P^*_M(t) \, dt \) approximates the normalized loss of susceptible pests in a period due to predation. If (15) is satisfied, then if the susceptible population becomes scarce, the total loss of susceptible pests in a period due to new infections and to predation is larger than the number of newborn susceptible pests in the same amount of time, so the susceptible pests cannot escape extinction. Note that, although at first (15) does not appear to display a formal dependence of the size of the juvenile pest population \( P^*_j \), which neither generates new infections nor predates on susceptible pests, phenomena which are reflected in (15), it is easy to see from (13) that \( P^*_M \) actually does depend upon \( P^*_j \). Also, it is seen that (15) does not display any sort of dependence upon \( c \), which does not appear in the reduced system (13). This is not unexpected, as the local stability of the susceptible pest-eradication periodic solution relates only to the speed at which susceptible pests are removed from the environment (to prey consumption, that is) and does not relate to the rate at which immature predators are born as a result. It will be observed that a condition similar to (15), but describing the reproductive potential of the susceptible pest population at larger population densities as well, will assure the global stability of the susceptible pest-eradication periodic solution.

**Theorem 4.2.** The susceptible pest-eradication periodic solution \((0, I^*, P^*_j, P^*_M)\) is globally asymptotically stable provided that

\[
rT < \int_0^T g(I^*(t)) \, dt + c_h \int_0^T P^*_M(t) \, dt,
\]

where

\[
c_h = \min_{0 \leq u \leq M_5} h'(u),
\]

\( M_5 \) being an ultimate boundedness constant for \( S \).

**Proof.** First, let us recall that

\[
I^* = z^*_0, d_j, I^* = z^*_0, m + d_j, \delta_j, P^*_M = z^*_M, M, \delta_M
\]

Let \( \varepsilon_1 > 0 \) be small enough, so that

\[
z^*_0, d_j, I^*(t) \geq \varepsilon_1, \quad z^*_0, m + d_j, \delta_j(t) \geq \varepsilon_1, \quad z^*_m, m + d_j, \delta_j(t) \geq \varepsilon_1 \quad \text{for all } t > 0
\]

and

\[
\eta = rT - \left( \int_0^T g(z^*_0, d_j, I^*(t) - \varepsilon_1) \, dt + c_h \int_0^T \left( z^*_m, m + d_j, \delta_j(t) - \varepsilon_1 \right) \, dt \right) < 0.
\]

It is seen that \( I'(t) \geq -d_j t \), and so by Lemma 3.1, \( I(t) \geq \tilde{z}_0, d_j, I^*(t) \), where \( \tilde{z}_0, d_j, \delta_j \) is the solution of (9) with the same initial data at \( 0^+ \) as \( I(0^+) \). As any such solution becomes close to \( I^* = z^*_0, d_j, I^* \), there is \( n_1 \in \mathbb{N} \) such that \( I(t) \geq z^*_0, d_j, I^*(t) - \varepsilon_1 \) for \( t \geq n_1 T \). Similarly, there is \( n_2 \geq n_1 \in \mathbb{N} \) such that \( P_j(t) \geq z^*_0, m + d_j, \delta_j(t) - \varepsilon_1 \) for \( t \geq n_2 T \). One then obtain that

\[
P^*_M(t) = mP_j(t) - d_M P_M(t) \geq m(z^*_0, m + d_j, \delta_j(t) - \varepsilon_1) - d_M P_M(t),
\]

so there is \( n_3 \geq n_2 \in \mathbb{N} \) such that

\[
P^*_M(t) \geq z^*_m, m + d_j, \delta_j(t) - \varepsilon_1 \quad \text{for } t \geq n_3 T.
\]

It follows that

\[
S'(t) \leq rS(t) - g(z^*_0, d_j, I^*(t) - \varepsilon_1)S(t) - S(t) c_h \left( z^*_m, m + d_j, \delta_j(t) - \varepsilon_1 \right) \quad \text{for } t \geq n_4 T, \quad n_4 \geq n_3.
\]

By integrating the above inequality on \((n_4 + (l - 1)T, n_4 + lT), l \in \mathbb{N}^*\) it follows that

\[
\ln(S((l + 1)T)) - \ln(S((n_4 + (l - 1)T))) \leq rT - c_h \int_0^T \left( z^*_m, m + d_j, \delta_j(t) - \varepsilon_1 \right) \, dt
\]

and consequently

\[
S((l + 1)T) \leq S(n_4 T) e^{ln_4} \quad \text{as } l \to \infty.
\]

Also,

\[
\frac{S'(t)}{S(t)} = r \left( 1 - \frac{S(t)}{K} \right) - g(I(t)) - \frac{h(S(t))}{S(t)} \leq r
\]
so
\[ S(t) \leq S((n + l - 1)T + e^{(n + l - 1)T}t), \quad t \in ((n + l - 1)T, (n + l)T], \]
which implies that
\[ S(t) \leq S((n + l - 1)T)e^{T}, \quad t \in ((n + l - 1)T, (n + l)T]. \]

Consequently, it follows from (23) and (24) that \( S(t) \to 0 \) as \( t \to \infty \).

We now need prove that \( I(t) - I^*(t) \to 0, P_j(t) - P_j^*(t) \to 0, P_M(t) - \hat{P}_M(t) \to 0 \) as \( t \to \infty \). To this purpose, let
\[ 0 < \varepsilon_2 < \frac{d_j}{g(\varepsilon_2)} \]. Then there is \( n_5 \geq n_4 \in \mathbb{N} \) so that \( 0 < S(t) < \varepsilon_2 \) for \( t \geq n_5T \). One obtains that
\[ I'(t) = g(I(t))S(t) - d_iI(t) \leq -(d_j - \varepsilon_2 g'(0))I(t) \quad \text{for} \ t \geq n_5T, \ t \neq nT. \]

Consequently, there is \( n_6 \geq n_5 \in \mathbb{N} \) so that
\[ z_n^* \leq I(t) \leq z_n^* + \varepsilon_1 \quad \text{for} \ t \geq n_6T. \]

It then follows that
\[ P_j'(t) = cP_M(t)h(S(t)) - (m + d_j)P_j(t) \leq c h(\varepsilon_2)M - (m + d_j)P_j \quad \text{for} \ t \geq n_6T, \ t \neq nT, \]

so there is \( n_7 \geq n_6 \in \mathbb{N} \) so that
\[ z_n^* \leq P_j(t) \leq z_n^* + \varepsilon_1 \quad \text{for} \ t \geq n_7T. \]

One then has
\[ P_M'(t) = mP_j(t) - d_MP_M(t) \leq m \left( \frac{z_n^*}{c} \right) + d_MP_M(t) \]
so there is \( n_8 \geq n_7 \in \mathbb{N} \) such that
\[ z_n^* \leq P_M(t) \leq z_n^* + \varepsilon_1 \quad \text{for} \ t \geq n_8T. \]

Using (25)–(27) and noting that
\[ I^* = z_n^*, \quad P_j^* = z_n^*, \quad P_M^* = z_n^*, \]
the conclusion follows. \( \square \)

First, let us note that a good candidate for an ultimate boundedness constant \( M_5 \) is \( K + \varepsilon, \varepsilon \) being an arbitrary positive value. Also, although (22) has a somewhat theoretical value, its interpretation is also transparent. While (15) stresses the fact that the susceptible pests cannot reproduce fast enough when they are scarce, (22) embeds the fact that the susceptible pests are depleted faster than they are born for larger population sizes as well. Moreover, if \( \delta_i = \delta_m = 0, \) it follows that
\[ P_j^* = z_{0;d_j;0}^* \equiv 0, \quad P_M^* = z_{0;0}^* \equiv 0. \]

Consequently, it follows that \( (0, I^*, 0, 0) \) is globally asymptotically stable provided that
\[ rT < \int_0^T g(I^*(s))ds, \]

which is in line with the results obtained in Georgescu and Moroșanu [19], where impulsive perturbations of a SI model are studied (no predation, that is). A similar validation of our results against the results obtained in [19] can be performed supposing that \( h \equiv 0, \) the case in which the equations for the pest and predator populations decouple, the reduced system which describes the dynamics of the pest population then being a particularization of the model studied in [19]. In this situation, the stability condition (22) also reduces to (28).

It is also to be noted that, while the local stability condition (15) does not depend upon \( c, \) the global stability condition (22) does, since \( c_I \) does depend upon the boundedness constant for \( S, \) which in turn depends upon \( P_M \) (and consequently upon \( P_j \) and \( c \)). This means that while a predator with a low \( c \) may stabilize (1) locally, it does not necessarily stabilize it globally.
5. The permanence of the system

In this section, we shall study the permanence of (1). For this purpose, we introduce the following definition.

**Definition 5.1.** The system (1) is said to be permanent (uniformly persistent) if there are $m, M > 0$ such that for each solution of (1) with strictly positive initial data $S(0), I(0), P_I(0), P_M(0)$, it follows that there is $T_0 > 0$ such that $m \leq S(t), I(t), P_I(t), P_M(t) \leq M$ for all $t \geq T_0$. Here, $T_0$ may depend upon the initial data, but $m$ and $M$ do not.

In biological terms, if (1) is permanent, then pests (both susceptible and infective) and predators (both immature and mature) will coexist, none of them facing extinction or growing indefinitely.

**Theorem 5.1.** The system (1) is permanent, provided that

$$rT > \int_0^T g(I^*(t))dt + h'(0) \int_0^T P_M^*(t)dt.$$ (29)

**Proof.** It has already been observed that, given $\varepsilon_1 > 0$ small enough, one may find $n_{\varepsilon_1} \in \mathbb{N}$ such that

$$I(t) \geq z_{0,t}, \delta \geq \varepsilon_1 > 0$$
$$P_I(t) \geq z_{0,m+\delta}, \delta > \varepsilon_1 > 0$$
$$P_M(t) \geq z_{m+\delta}, \delta > \varepsilon_1 > 0$$

for $t$ large enough, so $I(t), P_I(t), P_M(t) > \frac{\delta}{2}$ for $t$ large enough. Also, we know that $S, I, P_I, P_M$ are bounded, by Lemma 3.4. It now remains to prove that $S(t) \geq \tilde{m}$ for some $\tilde{m} > 0$ and $t$ large enough.

Let $m_1 > 0$ and $\varepsilon_3 > 0$ small enough, so that $m_1 < \frac{d_I}{g(0)}$ and

$$0 < \xi = rT - \left( \int_0^T g(z_{0,d_I} - g(0)m_1, \delta t) + \varepsilon_3 \right) dt + h'(0) \int_0^T \left[ z_m z_m' z_m(m_1, m+d_I, \delta) \right] dt.$$

We shall prove that one cannot have $S(t) \leq m_1$ for all $t > 0$. For this purpose, we give a contrasting argument. Suppose that $S(t) \leq m_1$ for all $t > 0$. One then has

$$I'(t) = g(I(t))S(t) - d_I I(t) \leq -(d_I - g(0)m_1) I(t) \quad \text{for } t \neq nT.$$

Consequently, there is $n_9 \in \mathbb{N}$ such that

$$I(t) \leq z_{0,d_I} - g(0)m_1, \delta t + \varepsilon_3 \quad \text{for } t \geq n_9 T.$$

Also,

$$P_I(t) = cP_M(t)h(S(t)) - (m + d_I)P_I(t) \leq cM(m_1) - (m + d_I)P_I(t) \quad \text{for } t \neq nT.$$

This yields that there is $n_{10} \in \mathbb{N}$ such that

$$P_I(t) \leq z_{cM(m_1, m+\delta), \delta} + \varepsilon_3 \quad \text{for } t \geq n_{10} T.$$

One then has

$$P_M'(m) = mP_I(m) - d_M P_M(m)$$
$$\leq m \left[ z_{cM(m_1, m+\delta), \delta} + \varepsilon_3 \right] - d_M P_M(m), \quad \text{for } t \geq n_{10} T, t \neq nT,$$

so there is $n_{11} \geq n_{10} \in \mathbb{N}$ such that

$$P_M(t) \leq z_{cM(m_1, m+\delta), \delta} + \varepsilon_3 \quad \text{for } t \geq n_{11} T.$$

It then follows that

$$S'(t) = rS(t) \left( 1 - \frac{S(t)}{K} \right) - g(I(t))S(t) - P_M(t)h(S(t))$$
$$\geq S(t) \left[ r - g(z_{0,d_I} - g(0)m_1, \delta t) + \varepsilon_3 + \left( z_{cM(m_1, m+\delta), \delta} + \varepsilon_3 \right) h'(0) \right], \quad \text{for } t \geq n_{11} T.$$
Integrating the above inequality on \((n_{11} + l - 1)T, (n_{11} + l)T\), \(l \in \mathbb{N}^*\), one obtains that
\[
\int \ln(S((n_{11} + l - 1)T)) - \ln(S((n_{11} + l)T)) \geq rT - \left( \int_0^T g(z_0^*, d_{j1} - g'(0)m_{j1}d_j)(t) + \varepsilon_3 \right) dt \\
+ h'(0) \int_0^T \left( e^{s_{m\min(m_{j1}, m+d_{j1} + \varepsilon_3)}} e^{d_{j1} \delta_m(t) + \varepsilon_3} \right) dt
\]
and consequently
\[
S((n_{11} + l)T) \geq S(n_{11}T) e^{\varepsilon_3} \rightarrow \infty \text{ as } l \rightarrow \infty,
\]
which contradicts the boundedness of \(S\). It then follows that there is \(t_1 > 0\) such that \(S(t_1) > m_1\).

If \(S(t) \geq m_1\) for all \(t \geq t_1\), then (1) is persistent and there is nothing left to prove. Otherwise, \(S(t) < m_1\) for some \(t \geq t_1\), and let us denote \(t_2 = \inf\{t > t_1, S(t) < m_1\}\). Then \(S(t) > m_1\) for \(t \in (t_1, t_2)\) and \(S(t_2) = m_1\). By a similar argument, one may construct \((t_n)_{n \geq 2}\) with the following properties

1. \(S(t_n) = m_1\) for all \(n \geq 2\);
2. \(S(t) < m_1\) for \(t \in (t_{2n}, t_{2n+1})\), \(n \geq 1\);
3. \(S(t) > m_1\) for \(t \in (t_{2n-1}, t_{2n})\), \(n \geq 1\),

describing the fact that \(S\) oscillates about \(m_1\).

We now show that \(\bar{T} = \sup\{t_{2k+1} - t_{2k}, k \in \mathbb{N}^*\} < \infty\). Otherwise, there is \((k_j)_{j \geq 1}\) such that \(t_{2k_j+1} - t_{2k_j} > j\); \(t_{2k} \geq n_{11}T\). Consequently, in a way similar to the derivation of (30), one may deduce that
\[
S(t_{2k+1}) \geq S(t_{2k}) e^{(j-2r)T},
\]
which is a contradiction, as \(e^{\varepsilon_3} \rightarrow \infty\) for \(j \rightarrow \infty\) and \(S(t_{2k+1}) = S(t_{2k}) = m_1\). It then follows that
\[
S'(t) = rS(t) \left(1 - \frac{S(t)}{K}\right) - g(l(t))S(t) - P_M(t) h(S(t)) \\
\geq S(t) \left[-\frac{r M}{K} - g(M) - M h'(0)\right].
\]

Let us denote
\[
m_2 = e^{-\left[\frac{r M}{K} + g(M) + M h'(0)\right] T} m_1 \text{ for } t \in (t_{2n}, t_{2n+1}).
\]

It then follows from (31) that \(S(t) \geq m_2\) for \(t \in (t_{2n}, t_{2n+1})\), which implies that \(S(t) \geq m_2\) for all \(t \geq t_1\), \(\square\)

This time, the meaning of (29) (which is the reverse of (15)) is that when the susceptible pests are scarce, the normalized number of newborn susceptible pests exceeds the total normalized loss of susceptible pests due to new infections and due to predation. Consequently, susceptible pests are able to escape extinction.

For 2-dimensional impulsively perturbed systems such as those studied in Georgescu, Zhang and Chen [56] or Georgescu and Zhang [57], it has been observed that the stability of the susceptible pest-eradication periodic solution, which was lost when (15) has been substituted with its reverse inequality, the permanence condition (29), is actually transferred to a newly emerging nontrivial periodic solution which emerges via a supercritical bifurcation. It is expected that the same phenomenon holds for a larger class of impulsively perturbed models currently in use, including certain higher-dimensional models.

6. Biological interpretations of the stability results and concluding remarks

Comparing (22) and (15), one notes that a predator which is voracious enough at small prey densities \((h'(0)\) large enough\) can always stabilize the susceptible pest-eradication periodic solution locally, but in order to be able to stabilize it globally, the predator should be voracious at large prey densities as well, when saturation occurs.

Also, from Lemma 3.6 it is seen that
\[
\int_0^T P_M(t) dt = \frac{1}{d_M \left(1 - e^{-d_{j1}T}\right)} \left( \delta_M + \int_0^T m P_M^*(t) dt \right) \\
\quad = \frac{1}{d_M \left(1 - e^{-d_{j1}T}\right)} \left( \delta_M + \frac{m}{(m + d_j) \left(1 - e^{-\left(m+d_{j1}\right)T}\right)} \delta_j \right),
\]

(32)
so (22) can be rephrased as

\[ rT < \int_0^T g(I^*(t))\,dt + c_h \frac{1}{d_M} \left( \frac{\delta_I}{(1 - e^{-d_M T})} \right) \left( \frac{m}{(m + d_j) \left( 1 - e^{-(m + d_j) T} \right)} \delta_j \right), \tag{33} \]

where \( c_h = \min_{0 \leq u \leq K} h'(u) \).

From (33), it can be seen that, by releasing enough juvenile or mature predators each time \((\delta_I, \delta_M, \delta_J, \delta_M)\) large enough one may always stabilize (1) irrespective of the nonlinear force of infection \(g\) or of the functional response of the predator \(h\). However, the use of this measure alone may be regarded as a “brute force” approach and may not necessary be cost-effective. Also, (33) implies that if the release periodicity \(T\) is small enough, the susceptible pest-eradication periodic solution again becomes globally asymptotically stable.

From Lemma 3.6, it is seen that

\[ \int_0^T g(I^*(t))\,dt \geq \tilde{c}_g \int_0^T I^*(t)\,dt = \tilde{c}_g \frac{\delta_I}{d_I \left( 1 - e^{-d_I T} \right)}, \]

where

\[ \tilde{c}_g = \min_{0 \leq u \leq K} g'(u), \]

so (1) can also be stabilized by releasing enough infective pest individuals as well, or by releasing pest individuals which are infected with a disease which spreads quickly (with large \(\tilde{c}_g\)). Further, it can be observed that (15) and (22) feature a continuous dependence upon \(\delta_I, \delta_M, \delta_J, \delta_M\), establishing the fact that small perturbations or irregularities in the application of controls will not impair the validity of the IPM strategy.

As an example to illustrate the utility of our results, it is easy to see that a system which fits into our framework is

\[
\begin{align*}
S'(t) &= rS(t) - \frac{S(t)}{K} - \beta I(t)S(t) - P_M(t) - aS(t) \frac{aS(t)}{1 + bS(t)}, \quad t \neq nT; \\
I'(t) &= b\beta I(t)S(t) - d_i I(t), \quad t \neq nT; \\
P_j'(t) &= cP_j(t) - aS(t) \frac{aS(t)}{1 + bS(t)} - (m + d_j) P_j(t), \quad t \neq nT; \\
P_M'(t) &= mP_j(t) - d_M P_M(t), \quad t \neq nT; \\
\Delta S(t) &= 0, \quad t = nT; \\
\Delta I(t) &= \delta_I, \quad t = nT; \\
\Delta P_j(t) &= \delta_j, \quad t = nT; \\
\Delta P_M(t) &= \delta_M, \quad t = nT,
\end{align*}
\tag{34}
\]

for which \(g(I) = \beta I, \beta > 0\), leading to the standard incidence rate \(g(I)S = \beta IS\) and \(h(S) = \frac{aS}{1 + bS}, a, b > 0\), that is, \(h\) describes a Holling type II functional response of the mature predator \(P_M\). Since

\[
I^* = z_{0;d_i;\delta_I;\delta_M}, \quad P_j^* = z_{0;m+d_j;\delta_j;\delta_M}^{*}, \quad P_M^* = z_{mP_j^*;d_M;\delta_M}^{*},
\]

it follows that

\[ \int_0^T g(I^*(t))\,dt = \beta \frac{\delta_I}{d_I \left( 1 - e^{-d_I T} \right)}. \]

Also, it has already been observed that

\[ \int_0^T P_M^*(t)\,dt = \frac{1}{d_M} \left( \frac{\delta_M + m}{(m + d_j) \left( 1 - e^{-(m + d_j) T} \right)} \delta_j \right). \]

We note that \(h'(x) = \frac{a}{1 + bx^2}, h''(x) = -\frac{2ab}{(1 + bx^2)^2} < 0\) for \(x > 0\) and, as previously mentioned, a candidate for an ultimate boundedness constant \(M_5\) is \(K + \varepsilon, \varepsilon\) being an arbitrary positive value. It is then seen from Theorems 4.1, 4.2 and 5.1 that the following result holds.

**Theorem 6.1.** The following statements hold.

(1) If

\[ rT \leq \beta \frac{\delta_I}{d_I \left( 1 - e^{-d_I T} \right)} + a \frac{1}{d_M} \left( \frac{\delta_M + m}{(m + d_j) \left( 1 - e^{-(m + d_j) T} \right)} \delta_j \right), \tag{35} \]

then the susceptible pest-eradication periodic solution \((0, I^*, P_j^*, P_M^*)\) is locally asymptotically stable.
Fig. 1. $g(I) = \beta I$ and $h(S) = \frac{am}{1 + bK}$. For $r = 5$, $d_i = 0.5$, $d_j = 0.4$, $d_M = 0.3$, $m = 2$, $c = 1$, $T = 4$, $\delta_i = 0.3$, $\delta_j = 0.2$, $\delta_M = 0.5$, $K = 10$, $a = 2$, $b = 3$, $\beta = 0.5$, the susceptible pest-eradication periodic solution is unstable and (1) is persistent, with a smaller persistency constant.

(2) If

$$rT > \frac{\delta_i}{d_i \left(1 - e^{-\delta_i T}\right)} + a \frac{1}{d_M \left(1 - e^{-\delta_M T}\right)} \left(\delta_M + \frac{m}{m + d_j} \left(1 - e^{-(m+d_j)T}\right) \delta_j\right),$$

then the susceptible pest-eradication periodic solution $(0, I^*, P^*_{J}, P^*_{M})$ is unstable and (1) is permanent.

(3) If

$$rT < \frac{\delta_i}{d_i \left(1 - e^{-\delta_i T}\right)} + \frac{a}{(1 + bK)^2} \frac{1}{d_M \left(1 - e^{-\delta_M T}\right)} \left(\delta_M + \frac{m}{m + d_j} \left(1 - e^{-(m+d_j)T}\right) \delta_j\right),$$

then the susceptible pest-eradication periodic solution $(0, I^*, P^*_{J}, P^*_{M})$ is globally asymptotically stable.

To illustrate our mathematical findings and facilitate their interpretation, we proceed to investigate further by using numerical simulations. For this purpose, we again particularize $g(I) = \beta I$ and $h(S) = \frac{am}{1 + bK}$. For $r = 5$, $d_i = 0.5$, $d_j = 0.4$, $d_M = 0.3$, $m = 2$, $c = 1$, $T = 4$, $\delta_i = 0.3$, $\delta_j = 0.2$, $\delta_M = 0.5$, $K = 10$, $a = 2$, $b = 3$, $\beta = 0.5$, it is seen that (36) holds and it consequently follows from Theorem 6.1 that the susceptible pest-eradication periodic solution $(0, I^*, P^*_{J}, P^*_{M})$ is unstable.

The behavior of the trajectory starting with $S(0) = 6, I(0) = 2, P_j(0) = 1, P_M(0) = 1$ is depicted in Fig. 1, where the time series for $S, I, P_J, P_M$ are provided, together with three dimensional representations $t$ vs $S$ vs $I$ and $t$ vs $P_J$ vs $P_M$, respectively; the value of the persistency constant appears to be small. If the value of $\alpha$ is changed to 1 and the other parameters are kept
constant (i.e. the predator is assumed to be less voracious), then (36) holds again, and consequently (1) is persistent, with a larger persistency constant. Also, the trajectory seems to tend to a nontrivial periodic solution. See Fig. 2 for details.

If the value of $r$ is changed to 1 this time, and the other parameters are kept constant (i.e. the intrinsic growth rate of the susceptible pest population is smaller), then (35) is satisfied and the susceptible pest-eradication periodic solution $(0, I^*, P_J^*, P_M^*)$ becomes locally asymptotically stable. The behavior of the trajectory starting with $S(0) = 6, I(0) = 2, P_J(0) = 1, P_M(0) = 1$ for the new value of $r$ is depicted in Fig. 3.

In this paper, an impulsively perturbed differential model describing an integrated pest management strategy is proposed and analyzed. To limit the growth of the pest population, natural predators are released in an impulsive and periodic fashion, in a constant amount each time. The life cycle of the predators is assumed to consist of two stages, immature and mature, only the mature predators being able to hunt for prey and reproduce. The functional response of the mature predator is given in an abstract, unspecified form, its reproductive rate being assumed to be proportional to the amount of prey consumed.

Also, infective pests are released simultaneously with the predators, with the purpose of spreading disease in the pest population, on the grounds that infective pests have a drastically lower damaging potential. A nonlinear incidence rate is used to describe disease transmission, while it is assumed that the disease does not spread to predators. It is found that the IPM strategy can always succeed, provided that enough resources are allotted (the amount of infective prey $\delta_I$ and of the immature and mature predators $\delta_J$ and $\delta_M$ released each time are large), the controls are used often enough ($T$ is small), the predators are voracious enough (the derivative of $h$ is large) or the disease spreads quickly (the derivative of $g$ is large). Any of these requirements alone assures the success of the IPM strategy, even though a combination of them is usually required in concrete situations. If the susceptibles can reproduce fast enough when they are scarce, it is shown that all pest and predator populations persist in the long term, with population sizes stabilizing above a strictly positive value.
Finally, several directions for further study include investigating whether or not a nontrivial periodic solution emerges when the following threshold equality holds

\[ rT = \int_0^T g(I^*(t)) \, dt + h'(0) \int_0^T P_M^*(t) \, dt, \quad (38) \]

as it is the case for several related 2-dimensional models, and investigating controllability results for models with more infectious stages.

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**References**


