

**THE IMPULSIVE CONTROL OF A TWO-PATCH
INTEGRATED PEST MANAGEMENT MODEL**PAUL GEORGESCU¹ AND HONG ZHANG

ABSTRACT. We hereby consider a two-patch SI integrated pest management model with dispersal of susceptible pests between patches, which is subject to periodic impulsive biological and chemical controls. The biological control consists in the periodic release of infective pests, in a constant amount, while the chemical control consists in periodic pesticide spraying, which causes the removal of fixed proportions of the infective and susceptible pest populations, respectively. The spread of the disease inflicted by the release of infective pests is characterized by a nonlinear incidence rate of infection expressed in an abstract, unspecified form. A sufficient condition for the local stability of the susceptible pest-eradication periodic solution is obtained through the use of Floquet theory for impulsive and periodic ordinary differential equations, the effect of population dispersal between patches upon the stability of this solution being then investigated for several particular cases.

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1. INTRODUCTION

After it has been noted that the traditional approach to pest control, consisting in the repeated use of pesticides, has led in many situations to pest resistance, which encourages even greater pesticide use, with little useful effects but causing yet more pest resistance and having unwelcomed secondary effects such as the persistence of hazardous residual chemicals in the environment, food contamination and the loss of biodiversity, an integrative approach called integrated pest management started to prevail. Integrated pest management consists in the use of a large array of methods to control pests, with an

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emphasis on minimizing the environmental and health risks and understanding the ecological relationships within the managed ecosystem. This approach may involve the use of biological agents (fungi, parasites or predators of the undesirable pest), chemical agents (selective pesticides and pheromones), together with cultural and physical aspects (pest barriers, crop rotation and tillage) and provides a conceptual framework for the development of effective and sustainable pest control methods. See Koul and Cuperus [11] for a discussion on ecologically-based integrated pest management strategies.

The biological control method consists in the use of natural enemies to regulate pest populations (see Hoffman and Frodsham [9]). Several common strategies are currently in use, namely the conservation biological control, in which the biocontrol agents are already present in the environment and are favored and enhanced via habitat management or other methods, the classical biological control, in which a small population of biocontrol agents is introduced at once in the environment with the purpose of stabilizing the system near a long-term equilibrium between pests and biocontrol agents and the augmentative biological control, in which biocontrol agents are repeatedly introduced in the environment with the purpose of totally eradicating the pest population. The chemical control method is generally avoided and used only as a last resort, as pesticides are sometimes the most effective way to contain a pest outbreak, selective pesticides which target the undesirable pest and minimize the damage inflicted on non-target organisms being preferred. See Brudea [4] for an overview of the various mechanisms of action of biocontrol agents and pesticides towards the incapacitation or removal of pests.

A perspective to augmentative biological control is to periodically release infective pest individuals, with the purpose of maintaining the endemicity of a disease in the target pest population, on the grounds that infective pests are usually less likely to reproduce or to damage the environment. In this case, the usual considerations on compartmental models for disease propagation apply, the dynamics of the pest population being then described via a SEIR model or one of its variations. To account for the immediate variation of the pest population sizes after the release of infective pest individuals and to reflect the discontinuous character of human control activities, models involving impulsive perturbations are often used (see Mailleret and Grognaud [13], Shi and Chen [15], Georgescu and Moroşanu [6]). See also Apreutesei [1] and Apreutesei and Dimitriu [2] for optimal control problems associated to a three-dimensional food chain and to a Lotka-Volterra model, respectively,

the purpose being to maximize the total population size at the end of a given interval $[0, T]$.

The more specialized and hard to find the food on which a species lives, the stronger it is constrained to live in resource-rich patches. Ultimately, the populations of all species are patchily distributed at one scale or another. Also, patchiness may be a feature of the physical environment itself (oceanic islands, mountain tops, forest patches) or may be created by the species itself through the depletion of local resources (Begon, Harper and Townsend [5]). Alternatively, patchiness may be an outcome of human interference or disturbance, which forces certain species to live in habitat remnants (Hastings and Wolin [8]). In this regard, destruction and fragmentation of habitats are viewed as some of the most serious threats to biodiversity worldwide (Wilcox and Murphy [18]).

A metapopulation is then a system of local populations which live in spatially separated habitat patches which are surrounded by inhospitable environment and are connected through dispersal fluxes. Individuals of local populations are often moving between patches on a daily or seasonal basis to maintain themselves within the same type of environment (the movement of crabs on a shoreline with the tide) or to escape major changes in food supply or climate (the seasonal migration of grazing animals). Also, density-dependent migration is a frequent way of avoiding overcrowding, inbreeding and kin competition.

Mathematical models of populations dispersing between patches are a subject of growing interest (see, for instance, Kuang and Takeuchi [12], Takeuchi [16], Hsieh, van den Driessche and Wang [10], Takeuchi, Wang and Saito [17]). It has been shown in Takeuchi [16] that dispersion between patches does not destabilize population dynamics, in the sense that in a patchy system occupied by a single species, if the species is able to survive at a globally stable equilibrium point when the patches are isolated, it will still do so at a different equilibrium (depending on the dispersal rate) for any dispersion rate. It has also been shown in Takeuchi, Wang and Saito [17] that if the species goes extinct in the absence of delays, it will do so for any delay lengths, while suitable time delays may drive the populations from coexistence to extinction.

The purpose of this paper is to construct a two-patch *SI* integrated pest management model which relies on the impulsive use of a biological control, in the form of periodic release of infective pest individuals, in a constant amount, and of a chemical control, in the form of periodic pesticide spraying. The

controls are used with the same periodicity, but not simultaneously. A non-linear force of infection g in an abstract, unspecified form, is used to model the spread of the disease inflicted by the release of infective individuals. Of concern is the stability of the susceptible-pest eradication periodic solution, our results extending the corresponding ones in Georgescu and Moroşanu [6], which are obtained for a single-patch version of our model.

The remaining part of this paper is organized as follows: in Section 2, the single-patch model treated in [6] is described together with the result obtained therein, the main biological assumptions on which our model relies being also formulated. In Section 3, several boundedness and stability results for impulsively perturbed systems of ordinary differential equations are introduced, together with a discussion on the exponential representation formula for the solution of a time-dependent system of ordinary differential equations. In Section 4, our patched model is formulated on the basis of the biological assumptions introduced in Section 2 and its well-posedness is established. Section 5 is concerned with a discussion of the stability of the susceptible-pest eradication periodic solutions, several comments on the biological significance of our results being also formulated.

2. A SI PEST MANAGEMENT MODEL

The following impulsively controlled system has been employed in Georgescu and Moroşanu [6] to characterize the dynamics of a SI integrated pest management model which is subject to periodic impulsive biological and chemical controls

$$\begin{aligned}
 S'(t) &= S(t)b(S(t)) - g(I(t))S(t), & t \neq (n+l-1)T, t \neq nT; \\
 I'(t) &= g(I(t))S(t) - wI(t), & t \neq (n+l-1)T, t \neq nT; \\
 \Delta S(t) &= -\delta_1 S(t), & t = (n+l-1)T; \\
 \Delta I(t) &= -\delta_I I(t), & t = (n+l-1)T; \\
 \Delta S(t) &= 0, & t = nT; \\
 \Delta I(t) &= \mu, & t = nT.
 \end{aligned} \tag{1}$$

In the above model, S and I denote the sizes of the susceptible and infective pest population, respectively, and it is supposed that all pests are either susceptible or infective. Also, $T > 0$ is the common periodicity of the biological and chemical controls, $\Delta\varphi(t) = \varphi(t+) - \varphi(t)$ for $\varphi \in \{S, I\}$ represent the instantaneous jumps of the pest population sizes after the use of controls,

$l \in (0, 1)$ is used to describe the time lag between the successive use of biological and chemical controls, $0 < \delta_1, \delta_I < 1$, $n \in \mathbf{N}^*$. The functions b and g satisfy the hypotheses indicated below.

(B) $b(0) = r$, b is decreasing on $[0, \infty)$, $\lim_{S \rightarrow \infty} b(S) < -w$, $S \mapsto Sb(S)$ is locally Lipschitz on $(0, \infty)$.

(G) $g(0) = 0$, g is increasing and globally Lipschitz on $[0, \infty)$.

To derive the above model, the following biological assumptions have been made

(A1) The intrinsic growth rate of the susceptible pest population in the absence of infection is given by the nonlinear function $S \mapsto Sb(S)$, where b satisfies (B).

(A2) The infective pests may neither recover nor reproduce.

(A3) The infective pests neither damage crops nor contribute to the total size of the environment-supported population.

(A4) The incidence rate of the infection is nonlinear in I and given by $g(I)S$, where the nonlinear force of infection g satisfies (G).

(A5) Infected pests are released in an impulsive and periodic fashion with periodicity T , in a fixed amount μ each time.

(A6) Pesticides are sprayed in an impulsive and periodic fashion, with the same periodicity T as the action of releasing infective pests but not simultaneously. As a result, fixed proportions δ_1 and δ_I of susceptible pests and infective pests, respectively, are removed each time.

Let us consider the following subsystem of (1)

$$\begin{aligned}
 I'(t) &= -wI(t), & t &\neq nT, (n+l-1)T; \\
 \Delta I(t) &= -\delta_I I(t), & t &= (n+l-1)T; \\
 \Delta I(t) &= \mu, & t &= nT; \\
 I(0+) &= I_0,
 \end{aligned} \tag{2}$$

which describes the dynamics of the susceptible pest eradication state. It has been seen in [4, Lemma 3.5] that the system consisting in the first three equations of (2) has a periodic solution I_w^* given by

$$I_w^*(t) = \begin{cases} \frac{\mu e^{-w(t-(n-1)T)}}{1 - e^{-wT}(1 - \delta_I)}, & t \in ((n-1)T, (n+l-1)T]; \\ \frac{\mu e^{-w(t-(n-1)T)}(1 - \delta_I)}{1 - e^{-wT}(1 - \delta_I)}, & t \in ((n+l-1)T, nT], \end{cases} \quad (3)$$

to which all solutions of (2) tend as $t \rightarrow \infty$. The following result, which establishes the existence of threshold parameter for the stability of (1) has also been proven in [6].

Theorem 1 ([6]) *The following statements hold.*

1. *The susceptible pest-eradication periodic solution $(0, I_w^*)$ of (1) is globally asymptotically stable provided that*

$$\int_0^T g(I_w^*(s))ds - \ln(1 - \delta_1) > rT. \quad (4)$$

2. *The susceptible pest-eradication periodic solution $(0, I_w^*)$ of (1) is unstable provided that*

$$\int_0^T g(I_w^*(s))ds - \ln(1 - \delta_1) < rT. \quad (5)$$

In this case, (1) is also permanent.

It may also be seen that if the equality

$$\int_0^T g(I_w^*(s))ds - \ln(1 - \delta_1) = rT \quad (6)$$

holds, then the susceptible pest-eradication periodic solution $(0, I_w^*)$ is stable, but not necessarily asymptotically stable. To complement the above result, it has also been shown in Georgescu, Zhang and Chen [7] that a supercritical bifurcation occurs in the limiting case, that is, the following result holds.

Theorem 2 ([7]) *A supercritical bifurcation occurs if (6) holds, in the sense that there is $\varepsilon > 0$ such that for all $0 < \tilde{\varepsilon} < \varepsilon$ there is a stable positive nontrivial periodic solution of (12) with period $T + \tilde{\varepsilon}$.*

The biological meaning of the conditions mentioned above is as follows. Suppose that (S, I) approaches the susceptible pest-eradication periodic solution $(0, I_w^*)$. Then, since the incidence rate of the infection is given by $g(I)S$, the integral $\int_0^T g(I^*(t))dt$ approximates the normalized (per susceptible pest) loss of susceptible pests in a period due to their movement to the infective class, while since the production of newborn susceptible pests is given by $Sb(S)$ and $b(0) = r$, rT approximates the normalized gain of susceptible pests in a period. A correction term $-\ln(1 - \delta_1)$ accounts for the loss of the susceptible pests due to pesticide spraying. In these settings, conditions (4), (5), (6) represent the fact that the total normalized loss of susceptible pests for a period due to disease transmission or pesticide spraying is larger than, less than or respectively equal to the total normalized gain of newborn susceptible pests for a period. Consequently, the above conditions can be understood as balance conditions near the susceptible pest-eradication periodic solution.

It is also seen that the classical concept of a basic reproduction number, which is related to the behavior of the system near the infective-free state and gives information about whether or not a single infective pest can cause a disease outbreak when introduced in an infective-free population at equilibrium, does not carry out well to impulsively perturbed systems with pulsed supply of infectives. This happens since for such systems of concern is whether or not the susceptibles (and not infectives) may persist, the survival of infectives being assured by the pulsed periodic supply. Consequently, although the stability of (12) is characterized by the threshold parameter $R_T = \frac{\int_0^T g(I_w^*(s))ds - \ln(1 - \delta_1)}{rT}$, as seen from Theorems 1 and 2, this is not a basic reproduction number in the classical sense, although it can be regarded as a reproduction number for the susceptible pest population.

3. PRELIMINARIES

The local stability results mentioned in Theorem 1 are obtained in [6] through the use of Floquet theory for impulsive and periodic ordinary differential equations. To state the theoretical result employed in [6] in order to obtain Theorem 1, which shall be of interest for this paper as well, let us consider the system

$$\begin{aligned} x'(t) &= A(t)x, & t &\neq \tau_k, t \in \mathbf{R}; \\ \Delta x &= B_k x, & t &= \tau_k, \tau_k < \tau_{k+1}, k \in \mathbf{Z}, \end{aligned} \tag{7}$$

under the following hypotheses.

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

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

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

In the above, by $PC(\mathbf{R}_+, \mathbf{R})$ ($PC^1(\mathbf{R}_+, \mathbf{R})$) is meant the class of real piecewise continuous (real piecewise continuously differentiable) functions defined on $[0, \infty)$. Let $\Phi(t)$ be a fundamental matrix of (7). Then there is a unique nonsingular matrix $M \in M_n(\mathbf{R})$ such that $\Phi(t+T) = \Phi(t)M$ for all $t \in \mathbf{R}$, which is called the monodromy matrix of (7) corresponding to Φ . Actually, all monodromy matrices of (7) are similar and consequently they have the same eigenvalues $\lambda_1, \lambda_2, \dots, \lambda_n$ regardless of the fundamental matrix Φ . These eigenvalues are then called the Floquet multipliers of (7). Under these hypotheses, the following result holds.

Lemma 1 ([3]) *Suppose that conditions (H1)-(H3) hold. Then*

1. *The system (7) is stable if and only if all Floquet multipliers λ_k , $1 \leq k \leq n$, satisfy $|\lambda_k| \leq 1$ and if $|\lambda_k| = 1$, then to λ_k there corresponds a simple elementary divisor.*
2. *The system (7) is asymptotically stable if and only if all Floquet multipliers λ_k , $1 \leq k \leq n$, satisfy $|\lambda_k| < 1$.*
3. *The system (7) is unstable if there is a Floquet multiplier λ_k such that $|\lambda_k| > 1$.*

Here, by elementary divisors of a square matrix we understand the characteristic polynomials of its Jordan blocks. We are now concerned with an exponential representation formula for the solutions of a general time-dependent system. Let us consider the system

$$x'(t) = B(t)x, \quad t \geq 0, \quad (8)$$

under the hypothesis

(H4) $B(\cdot) \in C([0, \infty), M_n(\mathbf{R}))$.

Then the fundamental matrix of (8) satisfying $\Phi(0) = I_n$ can be expressed as a Peano-Baker series in the form

$$\begin{aligned} \Phi(t) = & I_n + \int_0^t B(s_1)ds_1 + \int_0^t B(s_1) \int_0^{s_1} B(s_2)ds_2ds_1 \\ & + \int_0^t B(s_1) \int_0^{s_1} B(s_2) \int_0^{s_2} B(s_3)ds_3ds_2ds_1 + \dots \end{aligned} \quad (9)$$

If B commutes with its integral, that is,

$$B(t) \left(\int_0^t B(s)ds \right) = \left(\int_0^t B(s)ds \right) B(t) \quad \text{for } t \geq 0, \quad (10)$$

condition which is satisfied if the following hypothesis hold

(H5) $B(t)B(s) = B(s)B(t)$ for $t, s \geq 0$,

then the matrix fundamental matrix Φ can be expressed as

$$\Phi(t) = \exp \left(\int_0^t B(s)ds \right),$$

where, given $M \in M_n(\mathbf{R})$, $\exp(M)$ is defined as

$$\exp(M) = \sum_{k=0}^{\infty} \frac{1}{k!} M^k.$$

Note that, in general, the fundamental matrix Φ given by (9) may be different from $\exp(\int_0^t B(s)ds)$ if B does not satisfy (10).

We now indicate a result which provides an estimation for the solution of an impulsively perturbed system of differential inequalities.

Lemma 2 ([3]) *Let the function $u \in PC^1(\mathbf{R}_+, \mathbf{R})$ satisfy the inequalities*

$$\begin{aligned} \frac{du}{dt} & \leq (\geq) p(t)u(t) + f(t), & t \neq \tau_k, t > 0; \\ u(\tau_k+) & \leq (\geq) d_k u(\tau_k) + h_k, & k \geq 0; \\ u(0+) & \leq (\geq) u_0, \end{aligned} \quad (11)$$

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

$$\begin{aligned} u(t) \leq (\geq) & u_0 \left(\prod_{0 < \tau_k < t} d_k \right) e^{\int_0^t p(s)ds} + \int_0^t \left(\prod_{0 \leq \tau_k < t} d_k \right) e^{\int_s^t p(\tau)d\tau} f(s)ds \\ & + \sum_{0 < \tau_k < t} \left(\prod_{\tau_k < \tau_j < t} d_j \right) e^{\int_{\tau_k}^t p(\tau)d\tau} h_k. \end{aligned}$$

For other similar quantitative results on impulsive differential equations, see Bainov and Simeonov [3].

4. A TWO-PATCH SI PEST MANAGEMENT MODEL WITH DISPERSAL

We now suppose that the environment consists in two distinct patches and that the susceptible pests can travel freely between patches, but the infected pests are confined to the patch they live in. Further, it is supposed that assumptions (A1)-(A6) are valid in each patch, which gives rise to two subsystems of type (12), connected through the equations for the susceptible classes, in the form

$$\begin{aligned}
 S_1'(t) &= S_1(t)b_1(S_1(t)) - g(I_1(t))S_1(t) \\
 &\quad + d_{21}S_2(t) - d_{12}S_1(t), & t \neq (n+l-1)T, t \neq nT; \\
 S_2'(t) &= S_2(t)b_2(S_2(t)) - g(I_2(t))S_2(t) \\
 &\quad + d_{12}S_1(t) - d_{21}S_2(t), & t \neq (n+l-1)T, t \neq nT; \\
 I_1'(t) &= g(I_1(t))S_1(t) - wI_1(t), & t \neq (n+l-1)T, t \neq nT; \\
 I_2'(t) &= g(I_2(t))S_2(t) - wI_2(t), & t \neq (n+l-1)T, t \neq nT; \\
 \Delta S_1(t) &= -\delta_1 S_1(t), & t = (n+l-1)T; \\
 \Delta S_2(t) &= -\delta_2 S_2(t), & t = (n+l-1)T; \\
 \Delta I_i(t) &= -\delta_I I_i(t), & t = (n+l-1)T, i = 1, 2; \\
 \Delta S_i(t) &= 0, & t = nT, i = 1, 2; \\
 \Delta I_i(t) &= \mu, & t = nT, i = 1, 2.
 \end{aligned} \tag{12}$$

For the model above, $S_i(t)$, $I_i(t)$, $i = 1, 2$ denote the sizes of the susceptible and infective pest classes in patch i , respectively. The nonnegative constants d_{12} and d_{21} represent the dispersal rates of the susceptible pest population from patch 1 to patch 2 and from patch 2 to patch 1, respectively. In this regard, it is easy to see that if the dispersion coefficients d_{12} and d_{21} are null, then the equations for patches 1 and 2 decouple and we are led to consider two isolated subsystems of type (1). Also, a common situation is $d_{12} = d_{21} = D$, in which the migration from one patch to another is proportional to the difference in population sizes. The functions b_1 , b_2 , g , as well as the constants δ_1 , δ_2 , δ_I , μ are assumed to satisfy the conditions mentioned in Section 2. We shall denote $b_1(0) = r_1$, $b_2(0) = r_2$. Note also that the terms $\delta_i S_i$ and $\delta_I I_i$ may also describe the effects of selective catching, apart from characterizing the sensitivity of the pest species to the chemical control.

The impulsive control of a formally related Lotka-Volterra model has recently been considered by Yang and Tang in [19]. We shall use a similar

approach here, based on the use of Floquet theory for systems of periodic equations, although it should be noted that a computation of the exponential of a time-dependent matrix is done in [19] without verifying the commutation condition (10), which does not seem to hold in that particular setting.

In the remaining part of this section we shall establish the well-posedness of (12) in a mathematical and biological sense. First, it is easy to see that (12) has a unique solution for all sets of initial data. Using Lemma 2, it is possible to prove that all solutions of (12) which start with strictly positive initial data remain strictly positive on their entire interval of existence.

Lemma 3 *The set \mathbf{R}_+^4 is an invariant region for the system (12).*

Proof. Let us consider $X = (S_1, S_2, I_1, I_2) : [0, T_0) \rightarrow \mathbf{R}^4$ a solution for (12) defined on its maximal interval of existence which starts with strictly positive $S_1(0), S_2(0), I_1(0), I_2(0)$. It follows that

$$\begin{aligned} S_1'(t) &\geq S_1(t) [b_1(S_1(t)) - g(I_1(t)) - d_{12}], & t \neq (n+l-1)T, t \neq nT; \\ S_2'(t) &\geq S_2(t) [b_2(S_2(t)) - g(I_2(t)) - d_{21}], & t \neq (n+l-1)T, t \neq nT; \\ I_1'(t) &\geq -wI_1(t), & t \neq (n+l-1)T, t \neq nT; \\ I_2'(t) &\geq -wI_2(t), & t \neq (n+l-1)T, t \neq nT, \end{aligned} \tag{13}$$

as long as X remains positive component-wise. By integrating the above inequalities, using Lemma 2 and accounting for the effect of impulsive perturbations which occur for $t = nT$ and $t = (n+l-1)T$, it follows that

$$\begin{aligned} S_1'(t+) &\geq S_1(0)e^{\int_0^t p_1(s)ds} (1 - \delta_1)^{\lfloor \frac{t}{T} \rfloor}; \\ S_2'(t+) &\geq S_2(0)e^{\int_0^t p_2(s)ds} (1 - \delta_2)^{\lfloor \frac{t}{T} \rfloor}; \\ I_1'(t+) &\geq I_1(0)e^{-wt} (1 - \delta_I)^{\lfloor \frac{t}{T} \rfloor}; \\ I_2'(t+) &\geq I_2(0)e^{-wt} (1 - \delta_I)^{\lfloor \frac{t}{T} \rfloor}, \end{aligned} \tag{14}$$

where

$$\begin{aligned} p_1(t) &= b_1(S_1(t)) - g(I_1(t)) - d_{12}; \\ p_2(t) &= b_2(S_1(t)) - g(I_2(t)) - d_{21}, \end{aligned}$$

on the interval on which X stays positive component-wise, so X is actually strictly positive on $[0, T_0)$. ■

Using the above positivity Lemma, it is now possible to prove that all solutions of (12) are bounded.

Lemma 4 *There is $M > 0$ such that $S_i(t) \leq M$, $I_i(t) \leq M$ for $t \geq 0$, $i = 1, 2$.*

Proof. Let us define $u : \mathbf{R}_+ \rightarrow \mathbf{R}_+$ by

$$u(t) = S_1(t) + S_2(t) + I_1(t) + I_2(t), \quad t > 0.$$

Then

$$\frac{du}{dt} + wu = S_1(b_1(S_1) + w) + S_2(b_2(S_2) + w), \quad t > 0, t \neq (n+l-1)T, t \neq nT. \quad (15)$$

Since $\lim_{S \rightarrow \infty} b_i(S) < -w$, $i = 1, 2$, it follows that the right-hand side of (15) is bounded from above and consequently there is $C > 0$ such that

$$D^+u + wu \leq C, \quad t > 0, t \neq (n+l-1)T, t \neq nT.$$

One also sees that

$$u((n+l-1)T+) \leq (1-\delta)u((n+l-1)T)$$

and

$$u(nT+) = u(nT) + 2\mu,$$

where $\delta = \min(\delta_1, \delta_2, \delta_I)$. It follows from Lemma 2 that

$$\begin{aligned} u(t) \leq & u(0+) \left[\prod_{0 < (n+l-1)T < t} (1-\delta) \right] e^{-wt} \\ & + C \int_0^t \left[\prod_{s \leq (n+l-1)T < t} (1-\delta) \right] e^{-w(t-s)} ds \\ & + \sum_{0 < nT < t} 2\mu e^{-w(t-nT)}, \quad t > 0, \end{aligned} \quad (16)$$

so

$$u(t) \leq u(0+)e^{-wt} + \frac{C(1-e^{-wt})}{w} + 2\mu \frac{e^{wT}}{e^{wT}-1}, \quad t > 0. \quad (17)$$

Since the limit of the right-hand side of (17) as $t \rightarrow \infty$ is $C/w + 2\mu e^{wT}/(e^{wT}-1)$, it easily follows that u is bounded on $[0, \infty)$. ■

5. THE STABILITY OF THE SUSCEPTIBLE PEST-ERADICATION PERIODIC SOLUTION

As previously mentioned, when the dispersion coefficients d_{12} and d_{21} are null, one is led to consider two independent subsystems of type (1). Consequently, it is seen that the system consisting in the first four equations of (12) has a periodic solution $E^* = (0, 0, I_w^*, I_w^*)$, which gives the behavior of the system in the absence of susceptible pests. This solution shall be called in the following the susceptible pest-eradication periodic solution. We now attempt to discuss its stability by using the method of small amplitude perturbations. To this purpose, let us denote

$$\begin{aligned} S_1(t) &= u_1(t); \\ S_2(t) &= u_2(t); \\ I_1(t) &= v_1(t) + I_w^*(t); \\ I_2(t) &= v_2(t) + I_w^*(t), \end{aligned} \tag{18}$$

in which $u_i, v_i, i = 1, 2$, are understood to be small amplitude perturbations. Substituting (18) into the first four equations of (12), one obtains

$$\begin{aligned} u_1'(t) &= u_1(t)b_1(u_1(t)) - g_1(v_1(t) + I_w^*(t))u_1(t) + d_{21}u_2(t) - d_{12}u_1(t); \\ u_2'(t) &= u_2(t)b_2(u_2(t)) - g_2(v_2(t) + I_w^*(t))u_2(t) + d_{12}u_1(t) - d_{21}u_2(t); \\ v_1'(t) &= g(v_1(t) + I_w^*(t))u_1(t) - wv_1(t); \\ v_2'(t) &= g(v_2(t) + I_w^*(t))u_2(t) - wv_2(t). \end{aligned} \tag{19}$$

The corresponding linearization of (19) around $(0, 0, 0, 0)$ is

$$\begin{aligned} u_1'(t) &= r_1u_1(t) - g(I_w^*(t))u_1(t) + d_{21}u_2(t) - d_{12}u_1(t); \\ u_2'(t) &= r_2u_2(t) - g(I_w^*(t))u_2(t) + d_{12}u_1(t) - d_{21}u_2(t); \\ v_1'(t) &= g(I_w^*(t))u_1(t) - wv_1(t); \\ v_2'(t) &= g(I_w^*(t))u_2(t) - wv_2(t), \end{aligned} \tag{20}$$

while the linearization of the jump conditions at $(n + l - 1)T$ is

$$\begin{aligned} \Delta u_1 &= -\delta_1 u_1(t), & t &= (n + l - 1)T; \\ \Delta u_2 &= -\delta_2 u_2(t); \\ \Delta v_1 &= -\delta_I v_1(t); \\ \Delta v_2 &= -\delta_I v_2(t), \end{aligned} \tag{21}$$

and the linearization of the jump conditions at nT is

$$\Delta u_1 = \Delta u_2 = \Delta v_1 = \Delta v_2 = 0, \quad t = nT. \tag{22}$$

Let us denote

$$\begin{aligned} & \begin{pmatrix} (r_1 - d_{12}) - g(I_w^*(t)) & d_{21} & 0 & 0 \\ d_{12} & (r_2 - d_{21}) - g(I_w^*(t)) & 0 & 0 \\ g(I_w^*(t)) & 0 & -w & 0 \\ 0 & g(I_w^*(t)) & 0 & -w \end{pmatrix} \\ &= \begin{pmatrix} A(t) & O_2 \\ g(I_w^*(t))I_2 & -wI_2 \end{pmatrix} \end{aligned}$$

We note that

$$A(t)A(s) = \begin{pmatrix} a_1(t)a_1(s) + d_{21}d_{12} & d_{21}(a_1(t) + a_2(s)) \\ d_{12}(a_1(s) + a_2(t)) & a_2(t)a_2(s) + d_{12}d_{21} \end{pmatrix},$$

where

$$a_1(t) = (r_1 - d_{12}) - g(I_w^*(t)), \quad a_2(t) = (r_2 - d_{21}) - g(I_w^*(t)).$$

Then

$$\begin{aligned} a_1(t) + a_2(s) &= (r_1 - d_{12}) + (r_2 - d_{21}) + g(I_w^*(s)) - g(I_w^*(t)) \quad (23) \\ &= a_1(s) + a_2(t), \quad t, s \geq 0, \end{aligned}$$

which implies that

$$A(t)A(s) = A(s)A(t), \quad t, s \geq 0.$$

Specifically, the need for the commutation condition (23) constitutes the technical motivation for the use of the same nonlinear force of infection g and removal rate w for infectives in both patches; if different forces of infection g_1 and g_2 or removal rates w_1, w_2 were employed, then the equality

$$g_1(I_{w_1}^*(t)) + g_2(I_{w_2}^*(s)) = g_1(I_{w_1}^*(s)) + g_2(I_{w_2}^*(t))$$

would not necessarily hold. Note that a general approach towards solving a large class of 2-dimensional nonautonomous systems which overlap with the (reduced) system $u'(t) = A(t)u(t)$, with $A(t)$ as above, has been devised by Martinuși in [14] using a certain matrix representation theorem. A fundamental matrix of (20) is then

$$\Phi(t) = \begin{pmatrix} \exp(\int_0^t A(s)ds) & O_2 \\ Z(t) & e^{-wt}I_2 \end{pmatrix},$$

where

$$Z(t) = \int_0^t e^{w(s-t)} g(I_w^*(s)) e^{\int_0^s A(\tau) d\tau} ds.$$

Consequently, the corresponding monodromy matrix is

$$M = \begin{pmatrix} \begin{pmatrix} 1 - \delta_1 & 0 \\ 0 & 1 - \delta_2 \end{pmatrix} \exp(\int_0^T A(s) ds) & O_2 \\ (1 - \delta_I)Z & (1 - \delta_I)e^{-wT} I_2 \end{pmatrix}$$

and since $0 < (1 - \delta_I)e^{-wT} < 1$, the stability of the susceptible pest-eradication periodic solution E^* is then determined by the eigenvalues of the matrix

$$M_1 = \begin{pmatrix} 1 - \delta_1 & 0 \\ 0 & 1 - \delta_2 \end{pmatrix} \exp\left(\int_0^T A(s) ds\right).$$

Let us note that

$$\int_0^T A(s) ds = \begin{pmatrix} (r_1 - d_{12})T - \int_0^T g(I_w^*(t)) dt & d_{21}T \\ d_{12}T & (r_2 - d_{21})T - \int_0^T g(I_w^*(t)) dt \end{pmatrix}.$$

The eigenvalues of $\int_0^T A(s) ds$ are given by

$$\begin{aligned} \lambda_1 &= \frac{1}{2}(\alpha_1 + \alpha_2 + \sqrt{(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2}) \\ \lambda_2 &= \frac{1}{2}(\alpha_1 + \alpha_2 - \sqrt{(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2}), \end{aligned}$$

where

$$\begin{aligned} \alpha_1 &= (r_1 - d_{12})T - G; \\ \alpha_2 &= (r_2 - d_{21})T - G; \\ G &= \int_0^T g(I_w^*(t)) dt. \end{aligned}$$

Note that

$$\begin{aligned} \lambda_1 &\geq \frac{1}{2}(\alpha_1 + \alpha_2 + |\alpha_1 - \alpha_2|) = \max(\alpha_1, \alpha_2); \\ \lambda_2 &\geq \frac{1}{2}(\alpha_1 + \alpha_2 - |\alpha_1 - \alpha_2|) = \min(\alpha_1, \alpha_2); \\ \lambda_1 &= \alpha_i \Leftrightarrow \alpha_i = \max(\alpha_1, \alpha_2) \text{ and } d_{12}d_{21} = 0; \\ \lambda_2 &= \alpha_i \Leftrightarrow \alpha_i = \min(\alpha_1, \alpha_2) \text{ and } d_{12}d_{21} = 0; \\ \lambda_1 &= \lambda_2 \Leftrightarrow \alpha_1 = \alpha_2 \text{ and } d_{12}d_{21} = 0. \end{aligned}$$

If both dispersal rates d_{12} and d_{21} are null (there is no dispersal between patches, that is), then

$$\int_0^T A(s)ds = \begin{pmatrix} \alpha_1 & 0 \\ 0 & \alpha_2 \end{pmatrix}$$

and

$$\exp\left(\int_0^T A(s)ds\right) = \begin{pmatrix} e^{\alpha_1} & 0 \\ 0 & e^{\alpha_2} \end{pmatrix}, \quad M_1 = \begin{pmatrix} (1 - \delta_1)e^{\alpha_1} & 0 \\ 0 & (1 - \delta_2)e^{\alpha_2} \end{pmatrix}.$$

In this case, the eigenvalues of M_1 are

$$\mu_1 = (1 - \delta_1)e^{\alpha_1}, \quad \mu_2 = (1 - \delta_2)e^{\alpha_2}$$

and it follows that E^* is locally stable if

$$\max((1 - \delta_1)e^{\alpha_1}, (1 - \delta_2)e^{\alpha_2}) \leq 1 \tag{24}$$

and unstable if the opposite inequality holds.

If both dispersal rates d_{12} and d_{21} are nonzero, then $\int_0^T A(s)ds$ can be put into the diagonal form as

$$\int_0^T A(s)ds = \begin{pmatrix} d_{21}T & d_{21}T \\ \lambda_1 - \alpha_1 & \lambda_2 - \alpha_1 \end{pmatrix} \begin{pmatrix} \lambda_1 & 0 \\ 0 & \lambda_2 \end{pmatrix} \begin{pmatrix} d_{21}T & d_{21}T \\ \lambda_1 - \alpha_1 & \lambda_2 - \alpha_1 \end{pmatrix}^{-1}.$$

It follows that

$$\begin{aligned} & \exp\left(\int_0^T A(s)ds\right) \\ &= \begin{pmatrix} d_{21}T & d_{21}T \\ \lambda_1 - \alpha_1 & \lambda_2 - \alpha_1 \end{pmatrix} \begin{pmatrix} e^{\lambda_1} & 0 \\ 0 & e^{\lambda_2} \end{pmatrix} \begin{pmatrix} d_{21}T & d_{21}T \\ \lambda_1 - \alpha_1 & \lambda_2 - \alpha_1 \end{pmatrix}^{-1} \\ &= \begin{pmatrix} \frac{(\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1}}{\lambda_1 - \lambda_2} & \frac{d_{21}T(e^{\lambda_1} - e^{\lambda_2})}{\lambda_1 - \lambda_2} \\ \frac{(\lambda_1 - \alpha_1)(\lambda_2 - \alpha_1)(e^{\lambda_2} - e^{\lambda_1})}{d_{21}T(\lambda_1 - \lambda_2)} & \frac{(\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}}{\lambda_1 - \lambda_2} \end{pmatrix}. \end{aligned}$$

Consequently,

$$M_1 = \begin{pmatrix} (1 - \delta_1) \frac{(\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1}}{\lambda_1 - \lambda_2} & (1 - \delta_1) \frac{d_{21}T(e^{\lambda_1} - e^{\lambda_2})}{\lambda_1 - \lambda_2} \\ (1 - \delta_2) \frac{(\lambda_1 - \alpha_1)(\lambda_2 - \alpha_1)(e^{\lambda_2} - e^{\lambda_1})}{d_{21}T(\lambda_1 - \lambda_2)} & (1 - \delta_2) \frac{(\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}}{\lambda_1 - \lambda_2} \end{pmatrix}.$$

It is easy to see that

$$\begin{aligned} \text{Tr } M_1 &= (1 - \delta_1) \frac{(\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1}}{\lambda_1 - \lambda_2} \\ &\quad + (1 - \delta_2) \frac{(\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}}{\lambda_1 - \lambda_2} \end{aligned}$$

and since $\lambda_1 - \alpha_1 > 0$, $\lambda_2 - \alpha_1 < 0$, $\lambda_1 - \lambda_2 > 0$ it is seen that $\text{Tr } M_1 > 0$. Also,

$$\begin{aligned} \det M_1 &= \frac{(1 - \delta_1)(1 - \delta_2)}{(\lambda_1 - \lambda_2)^2} (T_1 + T_2) \\ &= (1 - \delta_1)(1 - \delta_2)e^{\lambda_1 + \lambda_2} \\ &= (1 - \delta_1)(1 - \delta_2)e^{\alpha_1 + \alpha_2} \\ &\geq 0, \end{aligned}$$

where

$$\begin{aligned} T_1 &= ((\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1})((\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}) \\ T_2 &= (e^{\lambda_1} - e^{\lambda_2})^2(\lambda_1 - \alpha_1)(\lambda_2 - \alpha_1) \end{aligned}$$

and, by a similar computation,

$$\begin{aligned} &(\text{Tr } M_1)^2 - 4 \det M_1 \\ &= (T_3 + T_4)^2 - 4(1 - \delta_1)(1 - \delta_2)e^{\lambda_1 + \lambda_2} \\ &= (T_3 - T_4)^2 \\ &\quad + \frac{4(1 - \delta_1)(1 - \delta_2)}{(\lambda_1 - \lambda_2)^2} [-(\lambda_1 - \alpha_1)(\lambda_2 - \alpha_1)] (e^{\lambda_1} - e^{\lambda_2})^2 > 0, \end{aligned}$$

where

$$\begin{aligned} T_3 &= (1 - \delta_1) \frac{(\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1}}{\lambda_1 - \lambda_2} \\ T_4 &= (1 - \delta_2) \frac{(\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}}{\lambda_1 - \lambda_2} \end{aligned}$$

It follows that M_1 has two distinct positive eigenvalues μ_1, μ_2 given by

$$\begin{aligned} \mu_1 &= \frac{1}{2} \left(\text{Tr } M_1 + \sqrt{(\text{Tr } M_1)^2 - 4 \det M_1} \right), \\ \mu_2 &= \frac{1}{2} \left(\text{Tr } M_1 - \sqrt{(\text{Tr } M_1)^2 - 4 \det M_1} \right). \end{aligned}$$

Consequently, since $0 < \mu_2 < \mu_1$, the trivial periodic solution E^* is locally stable if

$$\text{Tr } M_1 + \sqrt{(\text{Tr } M_1)^2 - 4 \det M_1} \leq 2 \quad (25)$$

and unstable if the converse inequality holds. Note also that

$$\text{Tr } M_1 = \frac{(\alpha_2 - \alpha_1)(\delta_2 - \delta_1)}{2\sqrt{(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2}}(e^{\lambda_2} - e^{\lambda_1}) + \frac{1}{2}(2 - \delta_1 - \delta_2)(e^{\lambda_1} + e^{\lambda_2})$$

and

$$\begin{aligned} & (\text{Tr } M_1)^2 - 4 \det M_1 \\ &= \frac{1}{4[(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2]} \left[(\alpha_2 - \alpha_1)(2 - \delta_1 - \delta_2)(e^{\lambda_2} - e^{\lambda_1}) \right. \\ & \quad \left. + \sqrt{(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2}(\delta_1 - \delta_2)(e^{\lambda_1} + e^{\lambda_2}) \right]^2 \\ & \quad + \frac{4(1 - \delta_1)(1 - \delta_2)}{(\alpha_1 - \alpha_2)^2 + 4d_{12}d_{21}T^2} d_{12}d_{21}T^2 (e^{\lambda_1} - e^{\lambda_2})^2. \end{aligned}$$

If one dispersal rate is zero and the other is nonzero, the stability of E^* depends upon whether or not $\alpha_1 = \alpha_2$. If $\alpha_1 \neq \alpha_2$, one arrives again by a similar argument at the same stability condition (25) via computing the eigenvectors of $\int_0^T A(s)ds$ in terms of d_{12} instead of d_{21} . If $\alpha_1 = \alpha_2 = \alpha$, then

$$\exp\left(\int_0^T A(s)ds\right) = e^\alpha \begin{pmatrix} 1 & d_{21}T \\ d_{12}T & 1 \end{pmatrix}$$

and $\int_0^T A(s)ds$ is triangular. It follows that

$$M_1 = e^\alpha = \begin{pmatrix} 1 - \delta_1 & (1 - \delta_1)d_{21}T \\ (1 - \delta_2)d_{12}T & 1 - \delta_2 \end{pmatrix}$$

and M_1 is triangular as well. The eigenvalues of M_1 are then

$$\mu_1 = e^\alpha(1 - \delta_1), \quad \mu_2 = e^\alpha(1 - \delta_2)$$

and the stability condition is

$$\max(e^\alpha(1 - \delta_1), e^\alpha(1 - \delta_2)) \leq 1, \quad \text{if } \delta_1 \neq \delta_2,$$

respectively

$$\max(e^\alpha(1 - \delta_1), e^\alpha(1 - \delta_2)) < 1, \quad \text{if } \delta_1 = \delta_2. \quad (26)$$

As the interpretation of the local stability condition is rather difficult, we shall concentrate in the following on several particular cases.

5.1. NO DISPERSAL ($d_{12} = d_{21} = 0$)

In this case, as previously seen, the stability condition reads as

$$\max((1 - \delta_1)e^{\alpha_1}, (1 - \delta_2)e^{\alpha_2}) \leq 1.$$

It then follows that E^* is stable if the pests can be eradicated in each isolated patch.

5.2. NO CHEMICAL CONTROL ($\delta_1 = \delta_2 = 0$)

If one dispersal rate is nonzero, the other is zero and $\alpha_1 = \alpha_2 = \alpha$, it follows that the stability condition (26) reduces to $\alpha < 0$, which does not depend on the value of the nonzero dispersal rate. This is justified, since in this situation the conditions in both patches are similar, so the movement of pests from one patch to another does not modify essentially the structural properties of the system.

If both dispersal rates are nonzero or only one dispersal rate is nonzero but $\alpha_1 \neq \alpha_2$, it follows that

$$\begin{aligned} \text{Tr } M_1 &= \frac{(\lambda_1 - \alpha_1)e^{\lambda_2} - (\lambda_2 - \alpha_1)e^{\lambda_1}}{\lambda_1 - \lambda_2} + \frac{(\lambda_1 - \alpha_1)e^{\lambda_1} - (\lambda_2 - \alpha_1)e^{\lambda_2}}{\lambda_1 - \lambda_2} \\ &= e^{\lambda_1} + e^{\lambda_2} \end{aligned}$$

and

$$\det M_1 = e^{\lambda_1 + \lambda_2},$$

from which we deduce that

$$\mu_1 = \max(e^{\lambda_1}, e^{\lambda_2})$$

and the sufficient condition for stability is

$$E(r_1, r_2, d_{12}, d_{21}, G, T) \leq 0,$$

where we denote

$$\begin{aligned} E(r_1, r_2, d_{12}, d_{21}, G, T) &= (r_1 - d_{12})T + (r_2 - d_{21})T - 2G \\ &\quad + \sqrt{[(r_1 - d_{12})T - (r_2 - d_{21})T]^2 + 4d_{12}d_{21}T^2}. \end{aligned}$$

We now further specialize this result to several particular cases in order to discuss the effects of dispersal upon the success of the impulsive control strategy.

5.2.1. BOTH PATCHES ARE ASYMPTOTICALLY STABLE WITHOUT DISPERSAL

In this situation, $r_1T - G < 0$, $r_2T - G < 0$. Then

$$\begin{aligned} E(r_1, r_2, d_{12}, d_{21}, G, T) &= (r_1T - G) + (r_2T - G) - (d_{12}T + d_{21}T) \\ &\quad + \sqrt{(r_1T - r_2T)^2 - 2(r_1T - r_2T)(d_{12}T - d_{21}T) + (d_{12}T + d_{21}T)^2} \\ &\leq (r_1T - G) + (r_2T - G) + |r_1T - r_2T| \\ &= 2 \max(r_1T - G, r_2T - G) \\ &< 0, \end{aligned}$$

so E^* remains stable regardless of the value of the dispersion rates d_{12} and d_{21} . That is, the susceptible pests can be eradicated regardless of the value of the dispersion rates if they can be eradicated in each patch in isolation.

5.2.2. BOTH PATCHES ARE UNSTABLE WITHOUT DISPERSAL

In this situation, $r_1T - G > 0$, $r_2T - G > 0$. Then

$$\begin{aligned} E(r_1, r_2, d_{12}, d_{21}, G, T) &= (r_1T - G) + (r_2T - G) - (d_{12}T + d_{21}T) \\ &\quad + \sqrt{(r_1T - r_2T)^2 - 2(r_1T - r_2T)(d_{12}T - d_{21}T) + (d_{12}T + d_{21}T)^2} \\ &\leq (r_1T - G) + (r_2T - G) - |r_1T - r_2T| \\ &= 2 \min(r_1T - G, r_2T - G) \\ &> 0, \end{aligned}$$

so E^* is unstable regardless of the value of the dispersal rates. That is, in this situation the susceptible pests persist regardless of the value of the dispersal rates if they persist in each patch in isolation.

5.2.3. ONE PATCH IS UNSTABLE AND ONE PATCH IS ASYMPTOTICALLY STABLE WITHOUT DISPERSAL

It is seen that

$$\begin{aligned} \lim_{d_{12} \rightarrow 0, d_{21} \rightarrow 0} E(r_1, r_2, d_{12}, d_{21}, G, T) &= r_1T + r_2T - 2G + |r_1T - r_2T| \\ &= \max(r_1T - G, r_2T - G) \end{aligned}$$

and consequently E^* is unstable for small dispersal rates. Also

$$E(r_1, r_2, d_{12}, d_{21}, G, T) = (r_1T - G) + (r_2T - G) + \frac{(r_1T - r_2T)^2 - 2(r_1T - r_2T)(d_{12}T - d_{21}T)}{T_5 + (d_{12}T + d_{21}T)},$$

where

$$T_5 = \sqrt{(r_1T - r_2T)^2 - 2(r_1T - r_2T)(d_{12}T - d_{21}T) + (d_{12}T + d_{21}T)^2}.$$

As a result, if $d_{12} = d_{21} = D > 0$, then

$$E(r_1, r_2, D, D, G, T) = (r_1T - G) + (r_2T - G) + \frac{(r_1 - r_2)^2T^2}{\sqrt{(r_1 - r_2)^2T^2 + 4D^2T^2} + 2DT}$$

and

$$\begin{aligned} \lim_{D \rightarrow \infty} \left[(r_1 - D)T + (r_2 - D)T - 2G + \sqrt{(r_1 - r_2)^2T^2 + 4D^2T^2} \right] \\ = (r_1T - G) + (r_2T - G) \end{aligned}$$

It is seen that E^* can be stabilized by large equal dispersion rates provided that $(r_1T - G) + (r_2T - G) < 0$, since in this situation the dispersal levels the differences between patches in the long term. If the opposite inequality holds, then E^* is unstable regardless of the value of D , since the unstable patch has a larger destabilizing potential.

If one dispersal rate is zero and the other is nonzero, then

$$\begin{aligned} E(r_1, r_2, d_{12}, d_{21}, G, T) &= 2 \max((r_1 - d_{12})T, (r_2 - d_{21})T) - 2G \\ &= 2 \max((r_1 - d_{12})T - G, (r_2 - d_{21})T - G). \end{aligned}$$

Consequently, in a system with one stable and one unstable patch the pests can be always driven to extinction by a large enough dispersal rate from the unstable to the stable patch provided that the dispersal rate from the stable patch to the unstable patch is zero. Conversely, if the dispersal rate from the unstable patch to the stable patch is zero, E^* remains unstable regardless of the value of the dispersal rate from the stable patch to the unstable patch. A valid pest control strategy would then be to discourage the dispersal from the stable patch to the unstable patch (which is detrimental to the success of the

control strategy), while encouraging the dispersion from the unstable patch to the stable patch (which is beneficial to the success of the control strategy). Consequently, while in some situations large dispersion rates would be beneficial, in other situations they will just cause a more serious pest outbreak. It is also to be noted that the stability condition (25) is significantly more complicated than its single-patch counterpart (4) and consequently the patch structure induces a nontrivial level of complexity.

As a consequence, it is seen that if both individual patches are stable or unstable, the stability or lack thereof is transmitted to the system at large regardless of the values of the dispersal rate. Note the similarity between our result and those obtained by Takeuchi [16] for a non-patched, non-controlled model. If one patch is stable and the other is unstable, the susceptible pests can be eradicated in certain conditions if large dispersal rates are permitted, although the system will certainly be unstable for small dispersal rates. In the same conditions, the susceptible pests can always be eradicated if the dispersal from the stable patch to the unstable patch is 0, while the dispersal from the unstable patch to the stable patch is large enough. Similarly, the susceptible pests can be eradicated in both patches if G is large enough, that is, if enough many pests are released periodically (μ is large enough) or the chemical control is efficient enough (δ_1 and δ_2 are both large enough), although in concrete situations this effort may not necessarily be cost-effective.

Further developments of this model include considering the effect of dispersal of infective pests between patches as well as using different forces of infection and reduction rates of infective pest populations due to the use of the chemical control in each patch. Another possible research direction is to consider the permanence of the patched system. This, however, is left for future work.

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