

"An assessment of bioeconomic modeling of pest resistance with new insights into dynamic refuge fields"

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An assessment of bioeconomic modeling of pest resistance with new insights into dynamic refuge fields

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Abstract

We examine the optimal time-variant refuge policy to manage pest resistance to *Bt* crops in a finite-horizon discrete-time model. We identify analytically the intertemporal effects of refuge fields on the pest population and its susceptibility. The shape of the optimal refuge policy and whether or not pest susceptibility should be exhausted completely at the end of the time horizon depend crucially on the values of a cost premium of *Bt* seeds and the fitness cost of resistance (over-mortality of resistant pests) and are addressed via numerical simulations. We demonstrate the importance of modeling the dynamics of the biological system accurately, of defining a diploid (and not haploid) biological model, and of using a discrete-time (rather than continuous-time) framework.

Keywords: *Bt* crop, optimal control, (non-)renewable resource, pest resistance management, refuge policy.

1 Introduction

Agricultural biotechnology research has increased the scope for developing pest-toxic crops in order to limit pest damage. However, pest-toxic crop varieties may lose their toxicity to pests over time as selection pressures increase the prevalence of resistant pests. Designing the best way to use pest-toxic varieties over time is thus a question of obvious economic interest, but with no easy answer. The design involves the intertemporal trade-off between controlling the pest population now and preserving its susceptibility to the pest-toxic crop in the future. This trade-off depends on the rather complex dynamics of two interdependent

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variables, namely, the size of the pest population and the susceptibility of pests to pest-toxic crop varieties.

Various papers in the literature have addressed the corresponding optimal control problem. Some rely on simplified biological models to extend the scope for explicit resolution, while others introduce many real-world features in simulation models based on particular pest-crop examples. Different prescriptions on the use of the pest-toxic variety have emerged from this literature. In this paper, we identify which modeling assumptions may explain this divergence in the results and we propose a modeling framework that, we believe, provides robust simulation results. We present the necessary conditions for optimality and then provide a sensitivity analysis of key biological and economic parameters based on simulations. We characterize the effect of three parameters, the fitness cost of resistance, the overcost of the pest-toxic variety seeds, as well as the relative dominance of resistance.

After early contributions in the 1970s-80s on chemical pesticide use, starting with the seminal paper by Hueth and Regev (1974), the economic analysis of pest resistance management has met a renewed interest with the advent of pest-toxic genetically modified (GM) crops. All presently commercialized pest-toxic transgenic crops have obtained their toxicity through the insertion and expression of the toxins of a soil bacterium, namely *Bacillus Thuringiensis* (*Bt*). Commercialization of these crops in the United States has raised concerns that pest populations may adapt to the transgenic crop, especially among scientists, and among environmental groups who fear that organic farmers may no longer be able to use *Bt* sprays for pest control. Due to active pressure from these groups, the large-scale adoption of *Bt* crops in the United States has been accompanied by the most comprehensive mandatory system ever developed for pest resistance management (EPA 2001, Bourguet *et al.* 2005). Since 1995 for *Bt* cotton and since 2000 for *Bt* corn, the U.S. Environmental Protection Agency (EPA) requires that all farmers growing a *Bt* crop devote a given percentage of their area to a non-GM, non-insect-toxic variety, which is referred to as the *refuge field*.

Refuge fields have been introduced as a tool to manage pest resistance, as they prevent a

quick selection of the *Bt* resistant pest population. By placing refuges near *Bt* crops, resistant insects that emerge from *Bt* crops will mate with susceptible pests emerging from refuge fields, thus maintaining the susceptibility to *Bt* crops within the gene pool. Leaving some of the crop unprotected in the refuge area causes immediate and future profit losses for farmers via higher pest prevalence. But by increasing the pest population's future susceptibility to the *Bt* crop, it decreases pest pressure in the future and improves future crop protection, thereby increasing future profits.

Bio-economic models on *Bt* crops in the literature obtain differing characterizations of the optimal refuge size as a function of the state of the system and thus provide differing policy advice on how to manage refuge fields over time. This paper discusses the assumptions used in these models and their impact on the optimal refuge size. Secchi *et al.* (2006), who run simulations with a realistic bio-economic model, conclude that the optimal refuge is initially low, then increases to a maximum, and finally decreases over the remaining time horizon. Livingston *et al.* (2004) obtain analogous results with a simulation model of resistance to *Bt* and to a chemical insecticide. Within a more stylized model, Laxminarayan and Simpson (2002) derive the optimal refuge at the steady state analytically and approximate it at the beginning of the time horizon. Qiao *et al.* (2008) run simulations on the entire optimal refuge path with a similar model. As opposed to Secchi *et al.* (2006), they find that no refuge should be planted initially. When the fitness cost of resistance is low, this initial phase is followed by a phase of “bang-bang” controls in which the optimal decision alternates back and forth between no *Bt* crop and no refuge, and next, by a singular path of varying intermediate refuges.¹ When the fitness cost of resistance is high, it is optimal to maintain no refuge for some years at the beginning of the period of analysis and an intermediate and constant refuge level over the remaining time horizon. Applying ‘singular-perturbation’ reduction methods, Grimsrud and Huffaker (2006) find that the size of the refuge should decrease monotonically over time. Qiao *et al.* (2009) simulate a pest's development of resistance to both a *Bt* toxin and a conventional pesticide. They find non-linear dynamics for refuges and conventional

pesticide use, possibly alternating the use of *Bt* cotton and pesticides to control the pest.

In order to derive a basis for comparison for the existing models in the literature, we develop a stylized biological model of pest-toxic crops and refuges that is similar to the pioneering model of Alstad and Andow (1995) in the biology literature on the high-dose/refuge strategy (section 2). A detailed comparison of this model with others in the literature shows that it is similar to that of Secchi *et al.* (2006), but different in various respects from Laxminarayan and Simpson (2002), Qiao *et al.* (2008, 2009) and Grimsrud and Huffaker (2006). More precisely, in all these models, we find that some events of the biological cycle described by the authors are actually not reflected in the equations of the biological model. Also, the biological models used by Laxminarayan and Simpson (2002) and Qiao *et al.* (2008) are written for haploid populations, while the refuge policy implemented by the EPA corresponds to the high-dose/refuge strategy defined by population geneticists, which has been explicitly designed for diploid insects.² A key element of this strategy is that most heterozygous individuals, which have received a susceptible allele from one parent and a resistant allele from their other parent, die on *Bt* crops (which are the "high-dose" component of the strategy). The role of the refuges is to maintain an available pool of homozygous susceptible pests, which can cross with homozygous resistant pests selected on GM crops, such that most of their heterozygous progeny will die on GM crops. This key factor is not accounted for in haploid models. Furthermore, all models mentioned in this paragraph use a continuous-time framework while, as we will show, a discrete-time model is better adapted to account for high selection pressures which may occur during some periods of the optimal refuge path. Finally, in addition to a continuous-time framework, Grimsrud and Huffaker (2006) postulate the speed of coevolution of the pest population and the resistance at a specific level rather than allowing it to be endogenously determined by the biological model.

Having set up the biological model in section 2, we add an economic component similar to that of Secchi *et al.* (2006). We follow the work of these authors, which relies exclusively on simulations, by analytically identifying the intertemporal effects of refuge fields on the pest

population and its susceptibility (section 3). Then, we illustrate the optimal time-variant refuge by means of an exhaustive dynamic comparative exercise on parameter values based on simulations (section 4). We assess in detail how the optimal refuge size should adjust over time and whether or not the susceptibility of the pest population should be completely exhausted (which depends entirely on the model parameters). In particular, we describe the effects of the overcost of *Bt* seeds and the fitness cost of resistance. In section 5, we present simulations related to a more general version of our model, in which the effective dominance of resistance is addressed.

2 Biological modeling

We start by developing a biological model that is consistent with the pioneering work by Alstad and Andow (1995) on pest resistance management with transgenic *Bt* crops and refuge fields. We then compare its properties with those of alternative models found in the economic literature thus far.

2.1 A biological model consistent with the population genetics literature

Following Alstad and Andow (1995), we assume a deterministic environment, with selection driving the evolution of a pest population and its genetic composition over time. Resistance is determined at a single locus with two alleles, where an allele can either be susceptible (*s*) or resistant (*r*) to *Bt*.³ Each insect inherits one allele from its father and one from its mother, and is thus either homozygous resistant (*rr*), homozygous susceptible (*ss*), or heterozygous (*rs*).

The biological model is written in discrete time. We define the pest population at the beginning of year t , $N(t)$, as the average number of larvae per plant, which may be distributed unevenly over *Bt* and refuge fields. Omitting the time notation, the average pest population is then $N = N_{rr} + N_{ss} + N_{rs}$, where N_{rr} , N_{ss} and N_{rs} respectively denote the average number of homozygous resistant, homozygous susceptible, or heterozygous larvae per plant. The total number of alleles is $N_r + N_s = 2N$, with $N_r = 2N_{rr} + N_{rs}$ resistant alleles and

$N_s = 2N_{ss} + N_{rs}$ susceptible alleles. The proportions of resistant and susceptible alleles are respectively $p_r = N_r/(2N)$ and $p_s = N_s/(2N)$, with $p_r + p_s = 1$. We assume that there is one generation of insects per year, with non-overlapping generations. Each generation has two development phases, detailed below. How they differ from the model of Alstad and Andow (1995) is discussed in Appendix A.

– **Stage 1:** Migration of emerging adults, reproduction and density dependence. Larvae pupate and eclose as adult moths that migrate, reproduce and lay eggs. We assume a logistic growth model with a growth rate g and a carrying capacity K .⁴ This growth rate is the average number of larvae per adult minus the natural mortality rate, which is equal to 1 because all adult moths die soon after laying eggs. The average number of larvae per plant at the end of stage 1, denoted as N_1 , is thus $N_1 = [1 + g(1 - N/K)]N$.

We assume that moths are sufficiently mobile to ensure random mating between moths emerging from the *Bt* and the refuge fields. We also assume that genotype proportions in each field are not affected by density dependence. The proportions of the three genotypes are therefore given by the Hardy-Weinberg ratios (Roughgarden, 1998).⁵ At the end of stage 1, the average numbers of larvae per plant for *rr*, *rs* and *ss* individuals are the same in each of the two fields (*Bt* and refuge), and are given by $N_{rr,1} = p_r^2 N_1$, $N_{rs,1} = 2p_r p_s N_1$, and $N_{ss,1} = p_s^2 N_1$.⁶

– **Stage 2:** Genotype-induced mortality. The high-dose/refuge strategy involves the use of a large enough concentration of *Bt* toxins to kill nearly all *rs* and *ss* larvae in the transgenic field (Bourguet *et al.*, 2005). Formally, let us define m_{ij} as the mortality rate of genotype *ij* on the *Bt* crop. The effective dominance of resistance, which characterizes the relative mortality rate of heterozygous pests on *Bt*, is defined as $h = \frac{m_{rs} - m_{ss}}{m_{rr} - m_{ss}}$; pest survival on *Bt* crops is recessive when $h = 0$ and is dominant when $h = 1$. The high-dose/refuge strategy requires m_{ss} to be close to 1 and h to be close to 0, so that almost all susceptible and heterozygous pests die on *Bt* crops (Bourguet *et al.*, 2000). In accordance with the empirical literature, we make the simplifying assumption that $m_{rr} = 0$ and $m_{ss} = 1$ (implying $h = 1 - m_{rs}$), so that

the *Bt* toxin in the transgenic field causes all larvae of genotype *ss* to die, while having no effect on *rr* larvae.⁷ In addition, larvae with genotype *rr* may experience increased mortality, regardless of which crop variety they feed on, if they face a positive fitness cost of resistance, $c \geq 0$. A strictly positive fitness cost implies that larvae acquire resistance to the detriment of their general fitness to the environment (Coustau *et al.*, 2000).⁸

We denote the proportion of the refuge area in year t as ϕ . The proportion of the *Bt* area is therefore $1 - \phi$. At the end of stage 2, the average population of each of the three genotypes $i = rr, rs$ and ss corresponds to its weighted average in the *Bt* and refuge fields. Specifically, $N_{rr,2} = (1 - c)N_{rr,1}$, $N_{rs,2} = ((1 - \phi)h + \phi)N_{rs,1}$ and $N_{ss,2} = \phi N_{ss,1}$.

Combining these two stages in year t yields the average population per plant at the beginning of year $t + 1$, N'_i , for each of the three genotypes: $N'_{rr} = (1 - c)p_r^2 N_1$, $N'_{rs} = 2((1 - \phi)h + \phi)p_r p_s N_1$ and $N'_{ss} = \phi p_s^2 N_1$. From the definition of N_1 and the Hardy-Weinberg ratios, we can then write:

$$N'_{rr} = p_r^2 N \left[1 + g \left(1 - \frac{N}{K} \right) \right] (1 - c), \quad (1)$$

$$N'_{rs} = 2p_r p_s N \left[1 + g \left(1 - \frac{N}{K} \right) \right] ((1 - \phi)h + \phi), \quad (2)$$

$$N'_{ss} = p_s^2 N \left[1 + g \left(1 - \frac{N}{K} \right) \right] \phi. \quad (3)$$

The first term in each of the above equations relates to random mating and Hardy-Weinberg genotype proportions. The bracketed terms in the three equations are identical and relate to the logistic regeneration of the pest population and its density dependence (the latter via the fraction N/K). The respective last terms, $1 - c$, $(1 - \phi)h + \phi$ and ϕ , are the genotype-dependent mortalities of each insect category. It is of interest to point out that the successive events of the insect lifecycle, namely, random mating, reproduction and density dependence, and genotype-dependent mortality, result in a multiplicative form in the three equations above. Note that several models in the literature instead use an additive form to model these biological events, and unlike us, do so without deriving this particular form from a rigorous biological lifecycle.

The three equations may be combined in order to write our model in terms of the evolution of allele numbers:

$$N'_r = N_r \left[1 + g \left(1 - \frac{N_r + N_s}{2K} \right) \right] \frac{(1-c)N_r + ((1-\phi)h + \phi)N_s}{N_r + N_s}, \quad (4)$$

$$N'_s = N_s \left[1 + g \left(1 - \frac{N_r + N_s}{2K} \right) \right] \left(\phi + \frac{(1-\phi)hN_r}{N_r + N_s} \right). \quad (5)$$

Interestingly, the two types of alleles interact not only via density dependence, *i.e.* the fraction $(N_r + N_s)/(2K)$ in the bracketed terms, but also via the impact of random mating on the genotypic composition, which is captured by the inclusion of both N_r and N_s in the far right multiplicative term of equations (4) and (5). This will turn out to be a major difference with haploid models that feature in the literature, in which, as we discuss below, resistant and susceptible insects interact only via density dependence.

2.2 Discussion of biological models used in the economic literature

Our biological model is very close to that of Secchi *et al.* (2006).⁹ It is also close to the biological model of Ambec and Desquilbet (2012), who focus on spatial instead of intertemporal aspects as we do here. However, as we detail below, it differs along several dimensions from models by Laxminarayan and Simpson (2002), Grimsrud and Huffaker (2006) and Qiao *et al.* (2008, 2009).

First, as an extensive use of pest-toxic crop varieties induces a high selection pressure and therefore a quick change in the number of susceptible alleles, the biological model should allow for the representation of such an evolution. However, as these models are written in continuous time to achieve mathematical tractability, they do not allow for jumps in the state variables, and, consequently, are only equivalent to their more realistic discrete-time counterpart as long as state variables change slowly (Otto and Day, 2007). For example, equation (5) states that if pest survival on *Bt* crops is fully recessive (*i.e.*, $h = 0$), planting only *Bt* crops (*i.e.*, $\phi = 0$) causes the number of susceptible alleles to drop to zero in one year (and therefore the level of resistance p_r to jump to its maximum value, $p_r = 1$). Laxminarayan and Simpson (2002) and Qiao *et al.* (2008) both find that no refuge

should be planted initially. However, in their continuous-time model, this initial policy maintains an intermediate level of resistance, while a discrete-time counterpart, like our model, would yield a jump to the maximum resistance of $p_r = 1$ within one year. In this context, the robustness of their policy recommendation can be challenged, since it propagates the benefits of decreasing the pest population in the short run when no refuge is planted, without accounting for the resulting quick rise in the level of pest resistance in the future.¹⁰

In our discrete-time setting, the robustness of results is not affected by the extent of the selection pressure.

The biological models developed by these authors differ from our model in other ways as well. We discuss these differences below using the discrete-time versions of their models.¹¹ In discrete time and with our notation, the model used in Laxminarayan and Simpson (2002) and Qiao *et al.* (2008) becomes:

$$n'_r = n_r \left[1 - c + g \left(1 - \frac{n_r + n_s}{K} \right) \right], \quad (6)$$

$$n'_s = n_s \left[1 - (1 - \phi)z + g \left(1 - \frac{n_r + n_s}{K} \right) \right], \quad (7)$$

where n_r and n_s correspond to the number of resistant and susceptible alleles (*and* insects because the authors use a haploid model) and where z corresponds to the proportion of susceptible insects that die on *Bt* fields (with $z = 1$ in Laxminarayan and Simpson, 2002).

First, with an additive form in the bracketed terms instead of a multiplicative form like in our model, these equations actually do not reproduce the biological events described by the authors. They define that all susceptible insects die on the *Bt* crop when $z = 1$. However, when all fields are planted with *Bt* varieties at one period ($\phi = 0$) and when $z = 1$, the population of susceptible insects remains strictly positive in the next period, $n'_s = n_s g \left(1 - \frac{n_r + n_s}{K} \right)$. For all susceptible insects to die on the *Bt* crop when $z = 1$, and for all resistant insects to experience a fitness cost of resistance, as the authors describe, the right-hand terms of equations (6) and (7) would have to be multiplied by $(1 - c)$ and ϕ respectively.

Second, the populations of resistant and susceptible insects in these other authors' models interact only via density dependence, while in our equations (4) and (5) they also interact via random mating and reproduction. A noteworthy consequence is that the population of resistant insects in the other authors' models is necessarily increasing over time if the fitness cost of resistance is zero and as long as the population is below its carrying capacity. This is shown by equation (6). In this particular case, as shown by equation (4), the number of resistant alleles is possibly decreasing through time in our model. We note that because of these shortcomings, refuge areas are only warranted in the model of Qiao *et al.* (2008) if there is a cost premium for *Bt* seeds, as shown in Appendix B. However, the high-dose/refuge strategy is expected to be of interest even when *Bt* and refuge seeds sell at the same price (see e.g. Alstad and Andow, 1995, or Hurley *et al.*, 2001).

Qiao *et al.* (2009) extend Qiao *et al.* (2008) by incorporating the assumption of diploid pests as we do. A discrete-time simplified version of their model is:¹²

$$\begin{aligned} N_{rr}' &= N_{rr} [1 + g(1 - N) - c], \\ N_{rs}' &= N_{rs} [1 + g(1 - N) - (1 - \phi)(1 - h) - ch], \\ N_{ss}' &= N_{ss} [1 + g(1 - N) - (1 - \phi)]. \end{aligned}$$

Again, the additive, as opposed to multiplicative form of the bracketed terms of these equations does not fit with the biological events that the authors describe. For example, the right-hand term of the third equation should be multiplied by ϕ , so that all insects of genotype *ss* die on the *Bt* crop.

Figure 1 illustrates the differing evolution of the discrete-time analogue to these authors' biological model compared to our biological model, assuming a constant refuge $\bar{\phi} = 0.5$, a growth rate and a carrying capacity $g = K = 1$, no fitness cost of resistance and a dominance of resistance to zero ($c = h = 0$), an initial resistance $p_{r_0} = 0.01$, an initial pest population $N_0 = K$, and a time horizon $T = 100$. With our biological model, the combination of *Bt* crops and refuges allows to decrease not only the number of susceptible alleles, but also, at

least initially, the number of resistant alleles. The total pest population decreases to a very low level and the resistance level remains small for most of the time horizon. This evolution is qualitatively different from the discrete-time versions of the models by Laxminarayan and Simpson (2002) and Qiao et al. (2009).

Grimsrud and Huffaker (2006) apply the geometric singular perturbation theory to pest resistance management. Singular perturbation reduction methods reduce the dimension of dynamic problems in which some variables change on widely different time scales, thereby simplifying their analysis. In their model, they define the evolution of the total pest population as a function of the evolution of the three genotype populations, rr , rs and ss . However they postulate the speed of resistance rather than deriving it endogenously from the evolution of these three genotypes (a detailed discussion is provided in Appendix D).

Figure 2 illustrates the difference between our model and the discrete-time analogue to the Grimsrud and Huffaker (2006) model, for a time horizon $T = 4000$ and using identical parameter values as in Figure 1.¹³ When survival of susceptible alleles on Bt crops is totally recessive ($h = 0$, that is, all insects of genotypes ss and rs die on Bt crops), the number of susceptible alleles constantly decreases in our model. In theirs, the evolution is non-monotonous, as it first decreases, then increases, and lastly decreases again. To our understanding, this is caused by the postulated relative speed of their two state variables N and p_r .

Since the diverging modeling assumptions above have lead to diverging results with respect to the refuge field size in the bio-economic literature, without delivering a uniform policy prescription, we feel that a further investigation is necessary. In what follows, using our biological model, we extend the results obtained by Secchi *et al.* (2006) in two ways: first we incorporate an explicit analysis of intertemporal effects; next, relying on simulations, we extend the assessment of the sensitivity of the optimal refuge path to parameters that are not included in their analysis, namely, the fitness cost of resistance and the overcost of Bt seeds.

3 Explicit analysis of the bio-economic model

In the remainder of the paper we use an alternative formulation of the biological model presented before, in which our variables of interest are the evolution of the average number of larvae per plant and the proportion of resistant alleles. For ease of exposition, we assume that $h = 0$, *i.e.* all heterozygous pests die on *Bt*-fields. The more general case with $h > 0$ will be addressed in our numerical simulations, where the sensitivity analysis shows its slight impact on the results. Equations (4) and (5) can be used to derive our variables of interest at the beginning of year $t + 1$, $N' = (N'_r + N'_s)/2$ and $p'_r = N'_r/(N'_r + N'_s)$:

$$N' = f_N(N, p_r, \phi) = [(1 - c)p_r^2 + \phi(1 - p_r^2)] \left[1 + g \left(1 - \frac{N}{K} \right) \right] N, \quad (8)$$

$$p'_r = f_r(p_r, \phi) = \frac{(1 - c)p_r^2 + p_r(1 - p_r)}{(1 - c)p_r^2 + \phi(1 - p_r^2)}. \quad (9)$$

The pest population, N , and pest resistance as measured by the average frequency of the resistant allele in the gene pool, p_r , are the state variables of our model. The control variable is the percentage of the total area allocated to the refuge in year t , $\phi \in [0, 1]$. Straightforward calculations show that $f_N(\cdot)$ is increasing in N if and only if $N < \frac{1+g}{2g}K$, increasing in p_r as long as $\phi < 1 - c$, and increasing in ϕ ; while $f_r(\cdot)$ is increasing in p_r and decreasing in ϕ . As a result, a positive refuge implies an immediate loss in agricultural returns by allowing the pest population to increase, but may imply future benefits by slowing down the evolution of resistance, and therefore slowing down the future evolution of the population, which confirms the intuition on the high-dose/refuge strategy given above.

The change in resistance as a difference equation is $\Delta p_r \equiv p'_r - p_r = \frac{(1-p_r)p_r^2(1-c-\phi)}{(1-c)p_r^2+\phi(1-p_r^2)}$. For a zero fitness cost ($c = 0$), resistance is non-decreasing and therefore the pest population's susceptibility, the mirror image of resistance, can be interpreted as a non-renewable resource. With a positive fitness cost of resistance ($c > 0$) and as long as $p_r < 1$, resistance increases over time if $\phi < 1 - c$ and decreases over time if $\phi > 1 - c$. Pest susceptibility is then a renewable resource because sufficiently high refuge levels allow susceptibility to increase over time, as pointed out, for example, by Laxminarayan and Simpson (2002).

Starting with (8) and (9), we set $\Delta p_r = 0$ and $\Delta N \equiv N' - N = 0$ to derive the steady-state configurations of the biological system. For any $\phi \neq 1 - c$, there are two distinct steady states, which are given by:

$$S_0 \equiv (N^{S_0}, p_r^{S_0}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{\phi} - 1 \right) \right], 0 \right), \quad (10)$$

$$\text{and } S_1 \equiv (N^{S_1}, p_r^{S_1}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{1-c} - 1 \right) \right], 1 \right). \quad (11)$$

For $\phi = 1 - c$, we have $\Delta p_r = 0$ for any value of p_r and hence all

$$S_i \equiv (N^{S_i}, p_r^{S_i}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{1-c} - 1 \right) \right], p_r \in [0, 1] \right). \quad (12)$$

constitute steady states where p_r may take ‘‘interior’’ values. Under mild conditions on the parameter values, which are detailed in Appendix E and assumed to hold in what follows, convergence to each steady state occurs eventually for appropriate values of the refuge size ϕ .

In order to analyze the dynamics of the biological system in greater detail, we draw a phase diagram in $N \times p_r$ space in Figure 3. The isoclines for N and p_r are the geometric loci for which $\Delta N = 0$ and $\Delta p_r = 0$. The dynamic forces driving the system when we find ourselves away from the isoclines are represented by arrows. Setting $\Delta p_r = 0$ in equation (9) yields three isoclines, either $p_r = 0$, or $p_r = 1$, or p_r takes a value strictly between 0 and 1 if the refuge area takes the critical value $\phi = 1 - c$. The shape of the ΔN isocline and the forces driving the pest population when we are away from it depend on the relative values of ϕ and $1 - c$.¹⁴ In Figure 3, steady states S_0 , S_1 and S_i lie at the intersection of the Δp_r and ΔN isoclines (note that the Δp_r isocline for $\phi = 1 - c$ is not drawn).

Two representative trajectories for the two state variables p_r and N for a constant refuge $\phi(t) = \bar{\phi}$, starting from an interior state (N_1, p_{r1}) , are also represented in Figure 3. As the system evolves in discrete time, the trajectories are, strictly speaking, sequences of points. When the refuge area is strictly lower than $1 - c$, the driving dynamic forces are represented in Figure 3 by the solid arrows and the solid line $\Delta N = 0$ (the dashed line $\Delta N = 0$ does not

apply). In this case, pest resistance monotonically increases over time until the *Bt* corn has entirely lost its efficacy ($p_r = 1$) (see equation (9)). The level of the pest population may initially decrease, in which case it eventually crosses the ΔN isocline. From that point in time onwards, the pest population then increases up to its long-run steady-state value. The arc linking the initial state (N_1, p_{r1}) to steady state S_1 shows this qualitative evolution of the state variables. In the particular case where $\bar{\phi} = 0$, pest resistance jumps to its maximum value $p_r = 1$ immediately, while the pest population evolves along the Δp_r isocline at $p_r = 1$ toward S^1 . When $\bar{\phi} > 1 - c$, the dynamic forces are represented by the dashed arrows and line. Such a large refuge reduces resistance over time. The pest population increases monotonically over time from the initial state and the dynamic system converges to S_0 . Finally, when the refuge area takes the critical value $\bar{\phi} = 1 - c$, pest resistance remains at its initial value p_{r1} and the pest population converges to $N^{S_i} = N^{S_1}$, so that the interior steady state S_i is reached.¹⁵

This preliminary analysis allows us to postulate some principles relating to the use of a constant refuge as a pest resistance management strategy. To start with, extensive use of *Bt* corn reduces the pest population, but comes at the cost of potentially exhausting susceptibility to *Bt* in the long run (steady state S_1 as defined in (11)). Moreover, avoiding any resistance (*i.e.* reaching $p_r = 0$) comes at the cost of a higher steady-state level of pests in the long run, *i.e.* $N^{S_0} > N^{S_1}$, where N^{S_0} in (10) is evaluated at $\phi > 1 - c$. Finally, there is only one constant refuge size which allows us to reach the interior steady state S_i , in which resistance is neither eradicated nor fully spread in the pest population, but instead remains constant. This steady state, which is the only one explicitly analyzed by Laxminarayan and Simpson (2002), can therefore be reached only in a very particular case.

3.1 The economic objective

We assume that yield losses are proportional to the number of larvae per plant after genotype-dependent mortality has occurred in each field. We allow GM seeds to be more expensive than non-GM seeds, with an exogenous cost premium $c_s \geq 0$ per unit of GM-planted area.

The current cost supported by farmers per unit of area in year t is given by:

$$C(N, p_r, \phi) = \alpha f_N(N, p_r, \phi) + c_s(1 - \phi), \quad (13)$$

where the time indices have been omitted. Let $\delta \equiv \frac{1}{1+\rho}$ be the discount factor, where ρ represents the annual social discount rate. In accordance with the related literature, the economic objective is to minimize the total discounted sum of the average yield loss encountered by farmers on the Bt and refuge fields and the cost premium of GM plantings:

$$V(N_1, p_{r1}) = \min_{0 \leq \phi \leq 1} \sum_0^T \delta^t C(N, p_r, \phi), \quad (14)$$

subject to the laws of motion of the state variables, p_r and N , as defined in (8) and (9) and where the time horizon $T < \infty$ is exogenous.¹⁶

4 The optimal refuge policy

We first characterize the necessary conditions that have to be satisfied by a refuge field and then analyze the optimal refuge field at the last period. Subsequently, we investigate the evolution of the dynamic system numerically.

4.1 Characterization of an interior solution for the refuge field

The Lagrangian function for our problem is:

$$L = \sum_{t=0}^T \delta^t \{ -C(N_t, p_{rt}, \phi_t) + \delta \lambda_{t+1} [f_N(N_t, p_{rt}, \phi_t) - N_{t+1}] + \delta \mu_{t+1} [f_r(p_{rt}, \phi_t) - p_{rt+1}] \}.$$

The unknowns in this problem are the series $\{\phi_t\}$, $t = 0, 1, \dots, T$, and $\{N_t, p_{rt}, \lambda_t, \mu_t\}$, $t = 0, 1, \dots, T + 1$ (see Conrad, 1999, for a detailed derivation of the necessary conditions for optimality of the unknown variables).

An interior solution of the refuge field at t is characterized by $\partial L / \partial \phi_t = 0$, which can be written explicitly as:

$$c_s - \delta \mu_{t+1} \frac{(1-c)(1-p_{rt})p_{rt}^2}{[\phi_t + p_{rt}^2(1-c-\phi_t)]^2} = \left(1 + g \left(1 - \frac{N_t}{K} \right) \right) N_t (1 - p_{rt}^2) (\alpha - \delta \lambda_{t+1}) \quad (15)$$

Following Leonard (1981), we presume the shadow values of the pest population and the allelic resistance to be non-positive (*i.e.*, $\lambda_{t+1}, \mu_{t+1} \leq 0$) when these state variables represent “bad stocks” in the sense that they negatively affect the objective function.

The left-hand side of equation (15) represents the social (marginal) cost of using Bt seeds, which includes both the additional cost of Bt seeds and the shadow cost of building up resistance. The right-hand side of (15) represents the social (marginal) benefit of avoided pest damage. Whenever marginal costs equal marginal benefits, an interior solution is optimal. However, when the cost of using Bt seeds outweighs its benefits at the margin, no Bt seeds should be used and $\phi_t = 1$.

4.2 The final period optimal refuge field

Since we are considering a finite time horizon, the problem can be solved by backward induction. At time T , the control ϕ_T is chosen for a given state of the system (N_T, p_{rT}) . As future changes in the state variables are not accounted for, the corresponding shadow values must satisfy $\lambda_{T+1} = \mu_{T+1} = 0$. Equation (15) then specifies a concave hyperbola $c_s = \alpha \left(1 + g \left(1 - \frac{N_T}{K}\right)\right) N_T(1 - p_{rT}^2)$ that divides the $N \times p_r$ space into two regions of extreme controls. For relatively low levels of resistance p_r and high values for the pest population N ($c_s < \alpha \left(1 + g \left(1 - \frac{N_T}{K}\right)\right) N_T(1 - p_{rT}^2)$), it is optimal to incur the additional cost of Bt seeds by planting no refuge ($\phi = 0$). In the opposite case, no Bt seeds should be used in the last period as no social benefit can be derived from a perfectly resistant pest population ($\phi = 1$).

It is important to note that the optimal refuge policy determined in the last period is independent of the length of the time horizon T , the discount factor δ and the initial state of the system (N_1, p_{r1}) . Which control applies in T depends on how the dynamic system has evolved over time and thus on the refuge policy. The following subsections address the optimal evolution of the refuge and the state dynamics of the system by simulations. The baseline parameters, which we will retain unless specified otherwise, are given in Table 1.¹⁷

Parameter		Value
N_1	initial average pest population per plant	K
p_{r1}	initial resistance frequency	0.05
c	fitness cost	0.05
g	growth rate	1
α	damage rate	0.064
K	logistic carrying capacity	1
c_s	additional cost of Bt seed	0.03
ρ	discount rate	0.03
δ	discount factor	$\frac{1}{1+\rho}$
T	time horizon	25

Table 1: Parameter values

Note: We calibrate the bioeconomic parameters on the European corn borer and Bt corn: c is the fitness cost of resistance in Vacher *et al.* (2007); α is taken from Calvin (1995) who reports a 6.4% annual yield reduction for corn due to the European corn borer; c_s is the ratio of the additional cost of Bt seeds reported by Onstad and Guse (1999) (10\$/acre) and the crop value when damages are negligible in Hurley *et al.* (2001) (305\$/acre); ρ is the discount rate in Hurley *et al.* (2001). Their shorter time horizon of $T = 15$ years is also used in our sensitivity analysis. We choose $p_{r1} = 0.05$ so that the initial resistance is low, but not too low (which would make the convergence of simulations harder). We adopt *ad-hoc* values of $g = K = 1$ for two reasons. First, Bt crops are mostly commercialized in areas where pests go through several generations per year. Our simplified model has one generation per year and is thus an imperfect approximation. Second, these parameters do not have rigorously established values and are calibrated with a variety of values in the literature (e.g., for the European corn borer on Bt corn, Hurley *et al.* (2001) calibrate $g_1 = 0.243$, $g_2 = 8.76$, $K_1 = 4.58$ and $K_2 = 0.85$; while Onstad *et al.* (2002) calibrate $g_1 = 1$, $g_2 = 10$, $K_1 = K_2 = 22$).

4.3 Comparative dynamic analysis of the fitness cost of resistance and the cost premium of Bt seeds

We first analyze the dynamics of the system when there is no fitness cost of resistance, in which case the susceptibility of the pest population is a non-renewable resource. As shown in Figure 4, the optimal refuge when the cost premium of Bt seeds is zero is at an intermediate level in the first period, leading to a drastic reduction of the pest population while maintaining relatively low resistance. The optimal refuge then increases over time, which slows down the increase in resistance. As resistance gets bigger, the refuge loses some of its efficiency and the share of land allocated to the refuge is progressively reduced over time. In a final phase, the optimal refuge is set to zero, causing a complete exhaustion

of susceptibility and an increase of the pest population along its logistic growth function (that would eventually lead to convergence to steady state of type S^1 , albeit beyond the time horizon of the economic program). This refuge path is comparable to that described by Secchi *et al.* (2006), but differs from the monotonically decreasing path obtained by Grimsrud and Huffaker (2006), as well as from the no-refuge path that Qiao *et al.* (2008) find when there is no cost premium of Bt seeds.

Still with a zero fitness cost of resistance, a different kind of refuge path arises when the cost premium of Bt seeds is strictly positive ($c_s > 0$). It is shown in Figure 5. In this case, the refuge does not only slow down the emergence of resistance, but is also cheaper. It is optimal in the initial phase to plant some positive, intermediate refuge field, which slows down the evolution of resistance. The optimal refuge is initially increasing, then drops to a very low value. The substantial increase in resistance occurring after this sharp decline in the refuge makes further use of the more costly Bt seeds sub-optimal and the refuge finally reaches $\phi_T = 1$ (see Figure 5). Over the planning horizon the refuge is always strictly positive, and, therefore, pest susceptibility is never completely exhausted. If ϕ were maintained at 1 beyond the time horizon, the system would eventually reach the interior steady state S^i .

The impact of a positive fitness cost is addressed in Figures 6 and 7. In the absence of a cost premium of Bt seeds ($c_s = 0$), results are similar to the case of $c = 0$. As can be seen from Figure 6, a higher fitness cost c tends to reduce the optimal refuge for each period and to cause the optimal refuge to become zero sooner, which is equivalent to exhausting pest susceptibility at an earlier date. This occurs because a higher fitness cost implies higher mortality among the resistant population, with the result that the smaller number of (more resistant) surviving individuals causes less damages. The argument that higher mortality among resistant pests tends to reduce the optimal refuge also applies when $c_s > 0$. A positive fitness cost then causes a complete exhaustion of the pest susceptibility, which occurs earlier if the fitness cost is higher, as depicted in Figure 7.¹⁸

In summary, our numerical evidence suggests that the susceptibility to Bt should gen-

erally be exhausted and that the higher the value of the fitness cost, the earlier exhaustion of pest susceptibility occurs. Only when resistant pests incur no fitness cost ($c = 0$) and Bt seeds are characterized by a cost premium ($c_s > 0$), is it optimal to preserve some susceptibility to Bt . In this case, there is no long-run advantage to facing a resistant pest population given that its steady-state level is identical to that of a susceptible pest population.

Finally, it is interesting to compare the optimal refuge policy described above with a refuge which is constrained to be constant over time ($\phi_t = \bar{\phi}$), while the economic objective, laws of motion and time horizon stay the same. Let $V(p_{r1}, N_1; \phi_t)$ and $V(p_{r1}, N_1; \bar{\phi})$ denote the intertemporal costs farmers face due to crop damage when the refuge adjusts optimally over time or remains constant, at an optimally set level. A constant refuge field necessarily increases costs, such that $\Delta V \equiv V(p_{r1}, N_1; \phi_t) - V(p_{r1}, N_1; \bar{\phi}) > 0$, because it represents an additional constraint in the optimization problem.

Table 2 shows that the constant refuge $\bar{\phi}$ is increasing in the additional cost of Bt seeds, and decreasing in the level of the fitness cost. It is never optimal to have $\bar{\phi} = 0$, and therefore pest susceptibility is never completely exhausted in the simulations.

	c	0	0.05	0.15	0.25
$c_s = 0$	$\bar{\phi}$ ($\Delta V/V(., \phi_t)$)	0.4033 (6.91%)	0.3567 (22.26%)	0.2467 (58.61%)	0.1367 (84.09%)
$c_s = 0.03$	$\bar{\phi}$ ($\Delta V/V(., \phi_t)$)	0.4367 (13.53%)	0.4100 (25.63%)	0.3833 (66.93%)	0.3833 (158.75%)
$c_s = 0.06$	$\bar{\phi}$ ($\Delta V/V(., \phi_t)$)	0.4633 (18.03%)	0.4500 (31.29%)	0.4433 (78.07%)	0.4467 (177.51%)

Table 2: Comparative analysis of intertemporal costs associated to constant *versus* dynamic refuges

Intertemporal costs are approximately 26% higher with a constant refuge when the benchmark parameters are used. In other words, our simulations show the potentially high advantage of varying the refuge size optimally. This result differs from that obtained by Secchi *et al.* (2006), whose simulations suggest that the optimal time-variant refuge offers very little economic gain over the optimal time-invariant refuge. These authors conclude that a static

policy may be preferable because it avoids the administrative cost associated with varying the refuge size. Our simulation results do not support this policy recommendation.

However, a difficulty resides in the fact that the shape of the optimal refuge path can differ markedly when parameters values vary slightly. As shown in Figure 8 for a fitness cost $c = 0$, the optimal refuge size may exhibit multiple sharp changes when a higher cost premium has to be incurred for Bt seeds. This “back and forth” in the optimal policy may include, in several periods, the maximum value $\phi = 1$. As can be seen from this figure, higher cost premiums increase the frequency of the back and forth pattern in the policy. Varying the fitness cost of resistance may also cause sizeable variation in the trajectory of the optimal refuge policy, as discussed before and as evidenced by Figures 6 and 7. As a result, varying the refuge size over time can only prove to be welfare enhancing when accompanied by a good knowledge of the economic and biological parameters pertaining to a particular pest-crop interaction – which may in effect be a real challenge.

4.4 Comparative dynamic analysis of further parameters

When all other parameters take the baseline values as given in Table 1 ($c_s = 0.3$, $c = 0.05$ and $\alpha = 0.064$), increasing the time horizon causes pest susceptibility to become completely exhausted at a later point in time (not shown). Furthermore, when considering the problem of optimally choosing a constant refuge $\bar{\phi}$, pest resistance is controlled for more intensively, as indicated by the increasing size of the constant refuge in Table 3. Interestingly, when the cost premium takes a relatively high value, such as $c_s = 0.05$, the susceptibility of the pest population is not completely exhausted for relatively long time horizons. This makes it possible to decrease the pest population by momentarily decreasing the refuge size and thereby controlling susceptible pests (not shown).

Comparative dynamics on the discount rate yield the intuitive result that increasing the discount rate ρ (and therefore decreasing the discount factor $\delta = 1/(1 + \rho)$) tends to lead to earlier exhaustion of pest susceptibility (not shown). Table 3 shows that the constant refuge field $\bar{\phi}$ decreases with an increase in the discount rate. This is due to the fact that a

T	15	25	55	100
$\bar{\phi}$ ($\Delta V/V(; \phi_t)$)	0.34 (110.54%)	0.41 (25.63%)	0.51 (16.06%)	0.52 (18.00%)
ρ	0	0.03	0.15	0.25
$\bar{\phi}$ ($\Delta V/V(; \phi_t)$)	0.4233 (26.88%)	0.41 (25.63%)	0.40 (61.63%)	0.37 (117.34%)

Table 3: Comparative dynamics on the time horizon and the discount rate ($c_s = 0.03$ and $c = 0.05$)

higher discount rate increases the current value of planting the Bt crop in order to achieve a reduction in the pest population. A higher discount rate also reduces the discounted value of using the refuge to maintain low resistance, a measure that improves pest population control in the future.

Finally, equation (13) shows that the economic objective is linear in both the damage rate, α , and the over-cost of Bt seeds, c_s . This implies that the dynamic comparative effect of increasing α is similar to that of decreasing c_s , which we analyzed above.

5 Addressing the effective dominance of resistance

Here we consider the general case of our model in which the relative mortality rate of heterozygous pests on Bt , h , is positive (not only homozygous, but also some heterozygous pests survive on a Bt fields). The following laws of motion, which are the general versions of equations (8) and (9) for $h \geq 0$, can then be derived from our biological model:

$$N' = [(1-c)p_r^2 + \phi(1-p_r^2) + 2(1-\phi)hp_r(1-p_r)] \left[1 + g \left(1 - \frac{N}{K} \right) \right] N, \quad (16)$$

$$p_r' = \frac{(1-c)p_r^2 + ((1-\phi)h + \phi)p_r(1-p_r)}{(1-c)p_r^2 + \phi(1-p_r^2) + 2(1-\phi)hp_r(1-p_r)} \quad (17)$$

From equation (17) the refuge size that keeps resistance constant, which was equal to $1-c$ in the previous case of $h = 0$, is now given by $\tilde{\phi} \equiv 1 - c \frac{p_r}{p_r + h(1-2p_r)}$. Resistance increases when the refuge size is below this threshold value and decreases when the refuge size is above the threshold. When c and h are positive, it is easily shown that $\tilde{\phi} > 1-c$ when $p_r < 1/2$,

and that $\tilde{\phi} < 1 - c$ when $p_r > 1/2$. In order to keep resistance constant when h is positive, a higher level of refuge has to be implemented than would be the case if h was equal to zero, as long as resistance is low (that is, less than half); the reverse holds when resistance is high. The reason is that heterozygous pests, of which some now survive on *Bt* fields, each contribute with one allele to the pool of resistant alleles, and with one allele to the pool of susceptible ones. Consider a situation in which N_r and N_s resistant and susceptible alleles would survive if no heterozygous pests survived (that is, if $h = 0$). Then, resistance would evolve to $p'_r = \frac{N_r}{N_r + N_s}$. Consider then the same planting pattern when a number n of heterozygous pests survives ($h > 0$). Then, resistance evolves to ($p_r^* = \frac{N_r + n}{N_r + N_s + 2n}$). It can easily be shown that $p_r^* > p'_r \Leftrightarrow N_s < N_r \Leftrightarrow p_r > 1/2$. The reverse holds when $p_r < 1/2$. This is illustrated on the right panel of Figure 9: as long as $p_r < 1/2$, the higher the level of relative dominance h , the faster resistance evolves; while the reverse holds for $p_r > 1/2$. To mitigate this evolution, a bigger refuge size is initially optimal when h increases; a pattern which is reversed towards the end of the planning horizon (left panel of Figure 9).

Another new characteristic we obtain with $h > 0$ relates to the maximum value that resistance p_r can reach. As heterozygous pest can now survive on *Bt* fields, the susceptible allele cannot be eradicated. This implies that $p_r < 1$. In particular, when the selection pressure for resistant alleles is at its maximum (no refuge is planted, $\phi = 0$), equation (17) yields: $p'_r = \frac{(1-c)p_r^2}{(1-c)p_r^2 + 2hp_r(1-p_r)} < 1$. Figure 10 illustrates this characteristic. In this simulation, pest susceptibility is renewable and *Bt* seeds imply an additional cost. With higher levels of the relative dominance, resistance increase faster, with an optimal back and forth pattern of the refuge field. The additional cost for *Bt* seeds, in combination with high levels of resistance, renders the use of *Bt* seeds economically inefficient at some point of time after which only non-*Bt* seeds are planted ($\phi = 1$). Resistance, which remained below its maximum value, then starts decreasing. We can conjecture that if this decrease in resistance occurs fast enough (or if the planning horizon is long enough), starting to plant *Bt* seeds may become efficient again.

6 Conclusions

We have presented a combined entomological-economic model in which a pest population damages a crop yield. Crop damage can be diminished by using pest-resistant varieties, such as *Bt* seed varieties. This, however, comes at the cost of decreasing the susceptibility of the pest population because *Bt* seeds select genes that contain the information on how to be resistant to the *Bt* toxin. One way to preserve the susceptibility of the pest population and the effectiveness of the *Bt* seed is to use refuge fields.

Our paper focuses on characterizing the optimal time-variant policy of the refuge field in a finite discrete time horizon model. Our analytical and numerical results characterize the refuge field as a function of the state of the system, as well as bioeconomic parameters such as the cost premium of *Bt* seeds and the fitness cost of resistance. All refuge paths share the pattern of considerably declining pest population in early periods, which considerably reduces crop damage in the short run. The induced increase in pest resistance to *Bt* is managed by increasing the size of refuge fields. An important result of our simulations is that the trajectory of the optimal refuge policy depends on the price differential between *Bt* seeds and refuge seeds. When there is no difference in seed prices, the optimal refuge starts at an intermediate level, first increases and then decreases to remain at zero in all our simulations. Depending on parameter values, when *Bt* seeds are more costly, the optimal policy may be characterized by a “back and forth” pattern for relatively high cost premiums; for relatively long time horizons, it may include a final phase in which the refuge is periodically decreased from its maximum size for one period in order to lower pest prevalence. In all simulations in which *Bt* seeds are more costly, the optimal refuge remains at its maximum size in the final periods of the time horizon.

In general, our numerical evidence suggests the complete extraction of pest susceptibility before the last period or at least the near extraction in the case of some heterozygous pests surviving on *Bt* fields, in which case complete extraction of susceptibility is not possible. However, we find evidence that some susceptibility should be preserved in situations in

which the social benefits of using *Bt* are the lowest. This happens when *Bt* seeds are more expensive than refuge seeds and when either the fitness cost of resistance is zero (*i.e.* there is no additional mortality among resistant insects), or the effective dominance of resistance is positive (*i.e.* some heterozygous pests survive on *Bt* crops). Finally, our simulations suggest that significant gains may be realized by using a time-variant (rather than time-invariant) refuge policy. The practical success of such a policy is, however, contingent on a good knowledge of the biological and economic parameters pertaining to the pest/crop interaction of interest, which may pose challenges for public regulators. In particular, the biological parameters characterizing pests targeted by *Bt* crops are not always well-known, as is evidenced by the variety of values used for calibration in the literature.

Further research may look into the cross-dynamics of the *Bt* crop/refuge strategy and conventional pesticides or transgenic crops with different toxins. However, introducing such additional complexity will make the model more difficult to interpret. Therefore, we believe our analysis of *Bt* crops represents a useful benchmark to gain intuition for future research on more complex situations. It would also be of interest to extend our work, which focuses on pest-toxic varieties, to the similar questions raised for pesticides. In this literature too (notably Regev *et al.*, 1983; Lazarus and Dixon, 1984; Plant *et al.*, 1985), a consensus on the design of the optimal intertemporal path of pesticide use has yet to emerge.

Appendix

A Discussion of the assumptions of our biological model

Our biological model includes several modifications relative to the initial model of Alstad and Andow (1995). As opposed to theirs, our model includes a fitness cost of resistance, which has been recognized as a major factor of the evolution of resistance with the refuge strategy (Lenormand and Raymond, 1998; Carrière and Tabashnik, 2001) We also make a few changes to the initial model for the sake of simplicity. In our model, density dependence happens before toxin-induced mortality while these events take place in the opposite order in the

model by Alstad and Andow (1995). We use a logistic function that models reproduction and density-dependence as concomitant (as is done for example by Onstad *et al.*, 2002). We assume only one generation of insects per year. This assumption holds neither for the European corn borer (ECB), the main target pest of *Bt* corn, which has two generations per year throughout much of the central Corn Belt (Alstad and Andow, 1995), nor for the tobacco budworm, the main target pest of *Bt* cotton, which has five generations each year in the U.S. Midsouth (Livingston *et al.*, 2004). However, one generation of ECB per season applies to more northern regions, e.g. parts of Ontario, Canada. While we assume perfect migration, Alstad and Andow (1995) assume that only 95% of moths fly away from the field in which they emerge. We assume that all insects of genotype *ss* die on *Bt* fields whereas Alstad and Andow (1995) assume that 0.1% of them survive on *Bt* corn.

B Analysis of the time-variant refuge strategy in the model of Qiao *et al.* (2008) with no cost premium of *Bt* seeds

In terms of our notation, in the model of Qiao *et al.* (2008) with no overcost of *Bt* seeds, the optimization problem is $\min_{\phi} \int_0^{\infty} e^{-\rho t} [\alpha(n_r + n_s)] dt$, subject to:

$$\dot{n}_r = [g(1 - n_r - n_s) - c]n_r, \quad (\text{B-1})$$

$$\dot{n}_s = [g(1 - n_r - n_s) - (1 - \phi)h]n_s. \quad (\text{B-2})$$

The current value Hamiltonian function is $H = -\alpha(n_r + n_s) + \lambda_r[g(1 - n_r - n_s) - c]n_r + \lambda_s[g(1 - n_r - n_s) - (1 - \phi)h]n_s$, where λ_r and λ_s respectively represent the shadow values associated with the populations of resistant and susceptible pests. An optimal solution must satisfy:

$$\dot{\lambda}_r - \rho\lambda_r = \alpha - \lambda_r[g(1 - 2n_r - n_s) - c] + \lambda_s g n_s, \quad (\text{B-3})$$

$$\dot{\lambda}_s - \rho\lambda_s = \alpha + \lambda_r g n_r - \lambda_s [g(1 - n_r - 2n_s) - (1 - \phi)h]. \quad (\text{B-4})$$

The Hamiltonian function is linear in the control. We define the switching function as $\Omega(t) \equiv \partial H / \partial \phi = \lambda_s h n_s$. The optimal refuge zone can be expressed as:

$$\phi(t) = \begin{cases} 0 & \text{if } \Omega(t) < 0 \\ \hat{\phi}(t) \in [0, 1] & \text{if } \Omega(t) = 0 \\ 1 & \text{if } \Omega(t) > 0 \end{cases}$$

where $\hat{\phi}$ is the singular control that applies whenever the switching function $\Omega(t)$ is zero, which happens either if $n_s = 0$ or if $\lambda_s = 0$.

There cannot be a singular path of the control variable with $n_s = 0$: in this case, we also have $\dot{n}_s = 0$, and the optimization problem reduces to $\min_{\phi} \int_0^{\infty} e^{-\rho t} \alpha n_r dt$ subject to $\dot{n}_r = [g(1 - n_r) - c]n_r$, and does not depend upon ϕ . Therefore, if a singular control exists, it must satisfy $\lambda_s = 0$, in which case $\dot{\lambda}_s = 0$ must also hold. Then from (B-4) we must have:

$$\lambda_r n_r = -\frac{\alpha}{g}. \quad (\text{B-5})$$

Differentiating (B-5), it must also hold that $\lambda_r \dot{n}_r + \dot{\lambda}_r n_r = 0$. Introducing \dot{n}_r and $\dot{\lambda}_r$ into this last equation using (B-1) and (B-3), we find that this last equation simplifies to:

$$\lambda_r n_r = 0. \quad (\text{B-6})$$

Equations (B-5) and (B-6) are incompatible. Therefore, no singular path to this minimization problem exists when $c_s = 0$.

C Simplified version of the model of Qiao et al. (2009)

Qiao et al. (2009) use a two-locus four-allele model to simulate resistance evolution to both *Bt* toxin and conventional pesticide. Here we write a simplified version of their model considering a one-locus two-allele model of resistance to *Bt* toxin alone and keeping our simplifying assumption that all homozygous susceptible pests die on *Bt* crops.

For each genotype $i = rr, rs$ or ss , define f_i as the fraction of genotype i , MR_i as the mortality rate of genotype i , and $m_{i,Bt}$ (respectively, $m_{i,ref}$) as the mortality rate of genotype i on the *Bt* (respectively, refuge) crop. From their Appendix 1 and their equation (1), their model with only one locus of two alleles is written $\dot{N}_i = f_i g N (1 - N) - N MR_i$, with $MR_i = f_i [(1 - \phi)m_{i,Bt} + \phi m_{i,ref}]$. These two equations imply that $\dot{N}_i = [g(1 - N) - (1 - \phi)m_{i,Bt} - \phi m_{i,ref}] f_i N$.

Our assumptions on mortality rates are: $m_{rr,Bt} = m_{rr,ref} = c$, $m_{ss,Bt} = 1$, $m_{ss,ref} = 0$, $m_{rs,Bt} = 1 - h + c$, $m_{rs,ref} = ch$. Using the same methodology as in the paper to write the discrete-time version of the model, we obtain:

$$\begin{cases} N_{rr}' = N_{rr} [1 + g(1 - N) - c], \\ N_{rs}' = N_{rs} [1 + g(1 - N) - (1 - \phi)(1 - h) - ch], \\ N_{ss}' = N_{ss} [1 + g(1 - N) - (1 - \phi)]. \end{cases}$$

Then, using that $N_r = 2N_{rr} + N_{rs}$, $N_s = 2N_{ss} + N_{rs}$ and $N_r + N_s = 2N$, after simplification, we obtain:

$$\begin{cases} N_r' = \left[1 + g\left(1 - \frac{N_r + N_s}{2}\right) - \frac{cN_r + ((1-\phi)(1-h) + ch)N_s}{N_r + N_s} \right] N_r, \\ N_s' = \left[1 + g\left(1 - \frac{N_r + N_s}{2}\right) - \frac{(1-\phi)N_s + ((1-\phi)(1-h) + ch)N_r}{N_r + N_s} \right] N_s. \end{cases}$$

D Evolution of resistance in the model of Grimsrud and Huffaker (2008)

For each genotype $ij = rr, rs$ or ss , Grimsrud and Huffaker define absolute selective values, W^{ij} , and genotype-specific mortality rates on transgenic fields, $M^{ij}(\phi)$ (where the time indices are omitted and use is made of our notation ϕ for the refuge proportion). From this, the authors calculate the evolution of the total pest population. From the above definitions, we calculate the evolution of each of the three genotype populations, $dN_{ij}/dt = (W^{ij} - M^{ij}(\phi))N_{ij}$, where $W^{ij} = \phi W_{rf}^{ij}(N) + (1 - \phi)W_{tg}^{ij}(N)$. Making use of $N = N_{rr} + N_{rs} + N_{ss}$ yields the same result for the total pest population as in Grimsrud and Huffaker (2008) (see their equation (23)). With N_{ij} and dN_{ij}/dt in hand, we can derive the evolution of pest resistance – a fact that remains unnoticed in the other authors' article. Indeed, given that $N_r = 2N_{rr} + N_{rs}$, one can easily compute that $dN_r/dt = N_r(W^r - M^r(\phi, p_r))$, where W^r is defined as in their paper and where $M^r(\phi, p_r) = p_r M^{rr}(\phi) + p_s M^{rs}(\phi)$. Introducing this equation as well as their equation (13) in the following equation defining the evolution of resistance, $dp_r/dt = p_r \left[\frac{1}{N_r} \frac{dN_r}{dt} - \frac{1}{N} \frac{dN}{dt} \right]$ one obtains the evolution of resistance that is consistent with the assumptions of the biological model, $dp_r/dt = p_r [W^r - W - (M^r(\phi, p_r) - M_{tr}(\phi, p_r))]$ where $M_{tr}(\cdot)$ is defined as in their paper. This differs from the definition $dp_r/dt = p_r \epsilon [W^r - W]$ they use in their paper, with the perturbation parameter ϵ set to 0.1 in the simulations.

E Stability analysis of the biological model

The interested reader may refer to Azariadis (1993) for technical details on what follows. The stability of steady state $S \in \{S_0, S_1, S_i\}$ as defined in (10) – (12) can be addressed by evaluating, at each steady state, the Jacobian matrix J of the linearized counterpart to (8) and (9), given by:

$$J = \begin{pmatrix} \frac{\partial f_N(N, p_r, \phi)}{\partial N} & \frac{\partial f_N(N, p_r, \phi)}{\partial p_r} \\ \frac{\partial f_r(p_r, \phi)}{\partial N} & \frac{\partial f_r(p_r, \phi)}{\partial p_r} \end{pmatrix} \quad (\text{B-7})$$

Solving the characteristic equation $|J - \nu I| = 0$, where I is the identity matrix, allows us to determine the eigenvalues ν associated with each steady state. We find:

$$\begin{array}{l} S \quad \{ \nu_1, \nu_2 \} \\ \hline S_0 \quad \{ 1, \quad 2 - (1 + g)\phi \quad \} \\ S_1 \quad \{ \frac{\phi}{1-c}, \quad 1 + c - (1 - c)g \quad \} \\ S_i \quad \{ 1, \quad 1 + c - (1 - c)g \quad \} \end{array}$$

Following theorem 6.2 in Azariadis (1993), a steady state of a non-linear system is asymptotically stable (called a sink), if it has two eigenvalues strictly smaller than unity. Only steady state S_1 may satisfy this condition. Indeed, this steady state can only be reached when $\bar{\phi} < 1 - c$, which implies that $\nu_1 < 1$; additionally, $\nu_2 < 1$ if and only if $g > c/(1 - c)$. With parameter values such that $\nu_2 > 1$, steady state S_1 is a saddle point and therefore unstable. If we have $\nu_2 = 1$, then S_1 is a non-hyperbolic equilibrium, which (following the aforementioned theorem) may be stable, asymptotically stable, or unstable and the ensuing discussion applies.

Being characterized by at least one unit eigenvalue, steady states S_0 and S_i (and possibly S_1) represent non-hyperbolic equilibria. In order to reach S_0 , we must have $\bar{\phi} > 1 - c$. Using this, it can be shown that $g > c/(1 - c)$ is a sufficient condition to ensure $\nu_2 < 1$. Moreover, a steady state represents a saddle-node bifurcation if the trace $Tr = \nu_1 + \nu_2$ of the corresponding Jacobian matrix satisfies $0 \leq Tr \leq 2$.¹⁹ This can be shown to hold for parameter values satisfying the additional condition $g \leq (2 + c)/(1 - c)$.

In summary, if parameters satisfy $c < g(1 - c) < 2 + c$, then S_1 represents a stable sink, while the stability of S_0 and S_i cannot be determined analytically. We believe that this

condition is relatively mild if we assume a relatively small fitness cost and an intermediate growth rate. Numerical analysis available on request shows that the particular case of $g = 1$ and $c = 0.05$ has stable sinks for all steady states.

Notes

¹In short, the fitness cost of resistance represents the additional mortality incurred by *Bt* resistant pests. It can be interpreted as the “opportunity cost” of the advantage of being resistant to the *Bt* crop variety. Secchi *et al.* (2006) assume that resistant pests incur no fitness cost of resistance.

²All diploid individuals have two alleles of each gene. A high level of resistance to *Bt* toxins is often due to variations in a single gene. Copies of this gene that confer susceptibility to *Bt* toxins are called susceptible alleles whereas those decreasing this susceptibility are referred to as resistant alleles. Individuals with two susceptible alleles are called homozygous susceptible whereas those with two resistance alleles are called homozygous resistant. Finally, individuals with one susceptible and one resistance allele are referred to as heterozygous individuals. Haploid pests each have only one allele of each gene, either susceptible or resistant.

³As stated in Roughgarden (1998), “[F]or our purposes, a “locus” is a spot on a chromosome. Two different genes that can occupy the same spot are called “alleles”.”

⁴We choose the logistic growth function because of its widespread use and convenience. However, Hurley *et al.* (2001) show that with this growth function an extensive use of *Bt* crops results in near eradication of pests, which may be unrealistic. See Secchi *et al.* (2006) for a sensitivity analysis on the degree of pest suppression with a modified logistic growth function.

⁵The probability that an allele of type i is paired with an allele of type j ($i, j = r$ or s) is determined as if alleles collided with one another at random. For example, the probability that a larva is of type rr is the probability that the first allele is of type r , p_r , times the probability that the second allele is of type r , p_r . The probability that a larva is of type rs is the probability that the first allele is of type r times the probability that the second one is of type s , $p_r p_s$, plus the probability that the first one is of type s times the probability that the second one is of type r , $p_s p_r$.

⁶We have that $p_r^2 + 2p_r p_s + p_s^2 = (p_r + p_s)^2 = 1$. Therefore, the identity $N_1 = N_{rr,1} + N_{ss,1} + N_{rs,1}$ holds.

⁷For example, for the European corn borer on *Bt* corn, Onstad *et al.* (2002) assume that $m_{rs} = 0.99$ and $m_{ss} = 0.999$, while Secchi *et al.* (2006) assume that $m_{rs} = 0.98$ and $m_{ss} = 1$; for the tobacco budworm on *Bt* cotton, Livingston *et al.* (2002) assume that $m_{rs} = 0.998$ and $m_{ss} = 0.999$, while Vacher *et al.* (2003) assume that $m_{rs} = 0.98$ and $m_{ss} = 1$. In all these papers, it is assumed that $m_{rr} = 0$.

⁸The actual fitness cost of resistance for the European corn borer and *Bt* corn remains unknown, as no case of *Bt* resistance has been confirmed for this pest. Onstad *et al.* (2002) and Secchi *et al.* (2006) assume that $c = 0$, while Vacher *et al.* (2007) assume that $c = 0.05$ (with heterozygous pests facing no fitness cost of resistance, which is also our assumption). For the tobacco budworm on *Bt* cotton, Vacher *et al.* (2003) and Livingston *et al.* (2002) respectively assume a fitness cost of 0.15 and 0.05 for homozygous resistant pests. They also assume that heterozygous pests face a positive but much lower fitness cost of resistance (respectively equal to 0.03 and 0.005 in these papers).

⁹It is actually easier to compare our biological model with that of Hurley *et al.* (2001), which is the one-toxin version of the more complex two-toxin model defined in Secchi *et al.* (2006). It is readily obtained that the biological model of Hurley *et al.* (2001) with random mating is equivalent to ours for the evolution of resistance, and very close for the evolution of the total pest population.

In the next section, we derive the evolution of the total population as $N' = [1 + g(1 - \frac{N}{K})] S$, with $S = [p_r^2 + \phi(1 - p_r^2) + 2(1 - \phi)hp_r(1 - p_r)] N$. Hurley *et al.* (2001) define it as $N' = [1 + g(1 - \frac{S}{K})] S$. It is easily shown that their biological model is consistent with a lifecycle composed of successive stages of migration, reproduction, genotype-induced mortality and density dependence – as long as we allow that the stages of reproduction and density-dependence, which are usually modeled as concomitant with the logistic function, may be modeled with this function as taking place at different moments.

¹⁰Such a rapid jump to a maximum resistance is unlikely to occur in real-world situations because other fields (of the same crop or of other crops) usually play the role of natural refuges (see for example Secchi and Babcock, 2003, or Qiao *et al.*, 2009)

¹¹The discrete-time analogs to these continuous-time models are derived by assuming that the laws of motion of each state variable x , written for the differential dx/dt , are also valid for the difference $x' - x$ (see Otto and Day, 2007, chapter 2).

¹²Qiao *et al.* (2009) simulate resistance evolution to both a *Bt* toxin and a conventional pesticide. We simplify their model by considering resistance to the *Bt* toxin alone, and we keep our simplifying assumption that all homozygous susceptible pests die on *Bt* crops, as detailed in Appendix C.

¹³Their parameter R_1 is analogue to our parameter g . We set their parameter R_2 to 1. With a definition of the dominance of resistance h analogous to ours, Grimsrud and Huffaker (2006) set h to 0.5 in all their analysis, stating that the available empirical evidence does not favor any particular dominance value. To our understanding, the literature is consistent with a level of h close to zero (see endnote 7). We draw the figure for a value $h = 0$. The discrete-time analogue to their model is then written: $N' = [1 + (\phi + (1 - \phi)p_r^2)g(1 - \frac{N}{K}) - (1 - p_r)^2(1 - \phi)] N$ and $p_r' = [1 + \epsilon(1 - p_r)p_r(1 - \phi)g(1 - \frac{N}{K})] p_r$.

¹⁴Setting $\Delta N = 0$ in equation (8) gives us the ΔN isocline as a function of p_r , $N(p_r) = K \left[1 - \frac{1}{g} \left(\frac{1}{(1-c)p_r^2 + \phi(1-p_r^2)} - 1 \right) \right]$. The forces driving the pest population N when away from the ΔN isocline are derived by calculating the derivative $\partial N(p_r)/\partial p_r$, which is negative for any $\phi > 1 - c$ (respectively, positive for any $\phi < 1 - c$). The derivative $\partial^2 N(p_r)/\partial p_r^2 = 2K(1 - c - \phi)[\phi - 3p_r^2(1 - c - \phi)]/(g[\phi + p_r^2(1 - c - \phi)]^3)$ gives the curvature of the ΔN isocline. For $\phi > 1 - c$, it is negative and the ΔN isocline is concave in p_r . For $\phi < 1 - c$, its denominator is positive, whereas the sign of the numerator is positive (negative) for $\phi > (<)(1 - c)3p_r^2/(1 + p_r^2)$. Since the right-hand side of the last equation is increasing in p_r and its maximum value is given at $p_r = 1$ by $3(1 - c)/4$, the ΔN isocline is convex for $\phi > 3(1 - c)/4$, but may also be concave.

¹⁵The dynamic system is non-stationary with respect to the refuge field ϕ . If ϕ changes over time, the ΔN isoclines will also change. Values of ϕ closer to the critical value $1 - c$ imply steeper ΔN isoclines. We presume that if the sequence of ϕ eventually converges to a particular value $\bar{\phi} \gtrless 1 - c$ (either from above or from below), the dynamic system will approach the corresponding steady-state configuration for sufficiently long sequences of ϕ .

¹⁶We do not include a salvage function, which would presumably depend on the levels of remaining pest susceptibility and pest population at time T . As seen in Secchi *et al.* (2006), this would tend to increase the social cost of using *Bt* seeds via its implied building up of pest resistance, particularly when the pest population cannot be eradicated or brought down to very low levels.

¹⁷In order to carry out the numerical simulations, we formulate the problem recursively and calculate the corresponding value function by backward induction. This approach is explained, for example, in Judd (1998), p. 409.

¹⁸Figure 7 shows that for $c = 0.25$, $\phi = 0$ in the fourth period, and therefore susceptibility is exhausted at the end of that period.

¹⁹A saddle-node bifurcation equilibrium lies on the limit of two regions in which an equilibrium can be characterized as a stable sink or a saddle.

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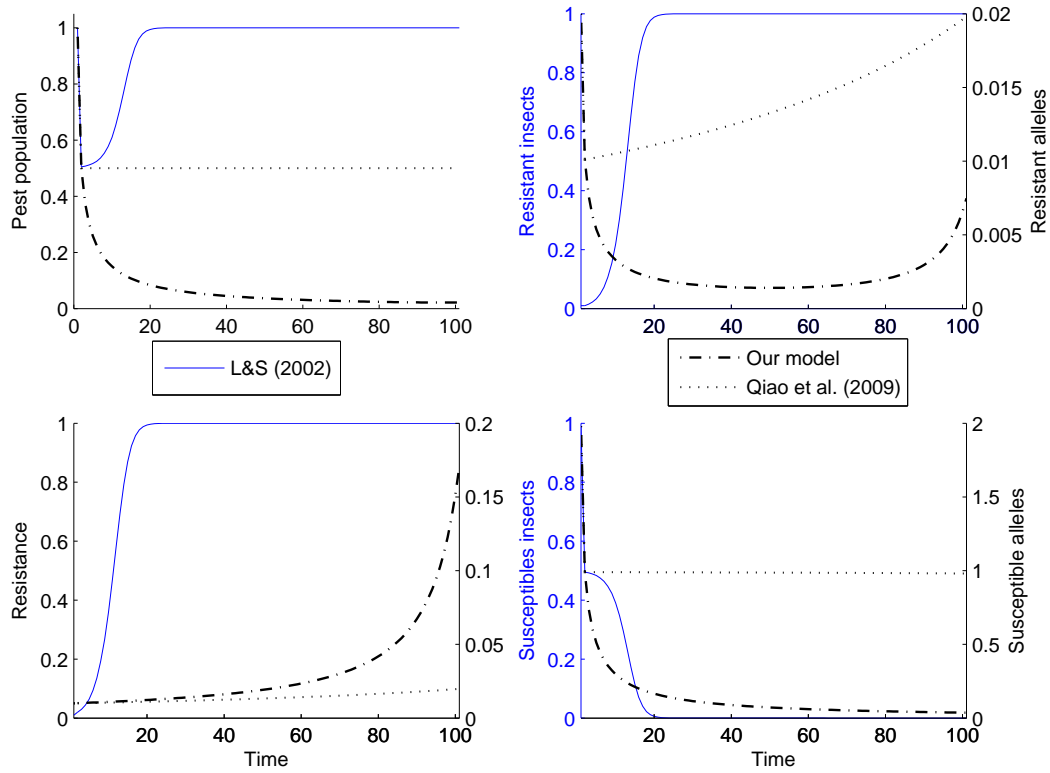


Figure 1: Comparison with L&S (2002) and Qiao *et al.* (2009)

Note: In the two graphs on the upper right, the vertical axis on the left refers to L&S (2002) (resistant and susceptible insects, respectively on top and bottom); the vertical axis on the right refers to Qiao *et al.* (2009) and our model (resistant and susceptible alleles, respectively on top and bottom).

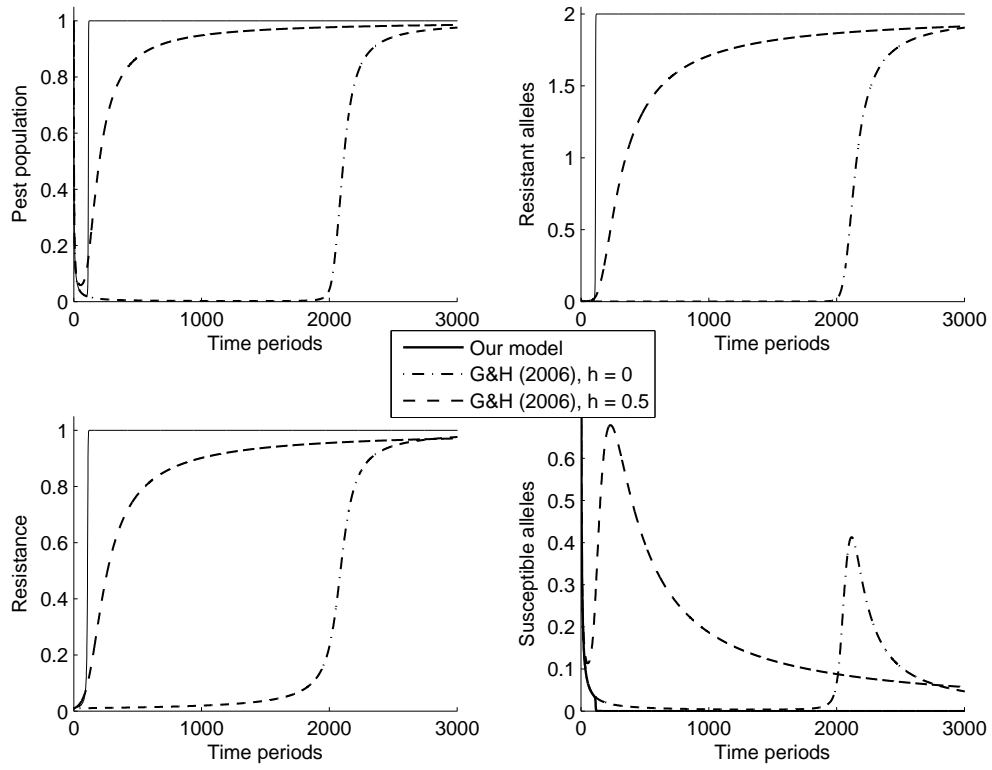


Figure 2: Comparison with Grimsrud and Huffaker (G&H, 2006)

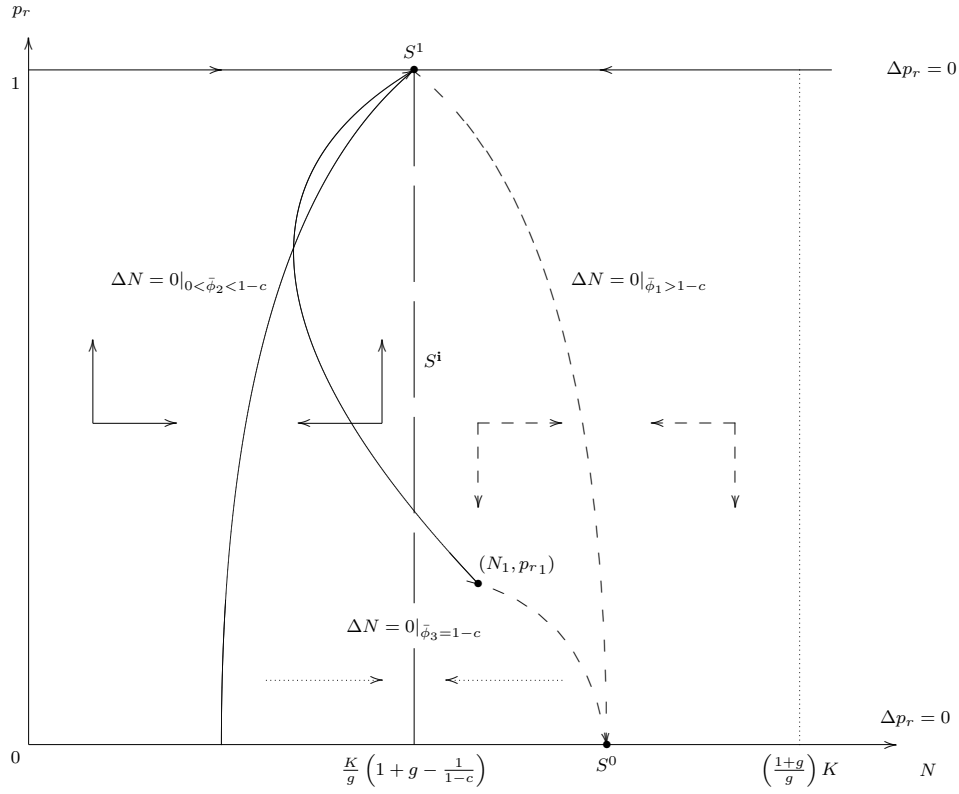


Figure 3: The phase diagram

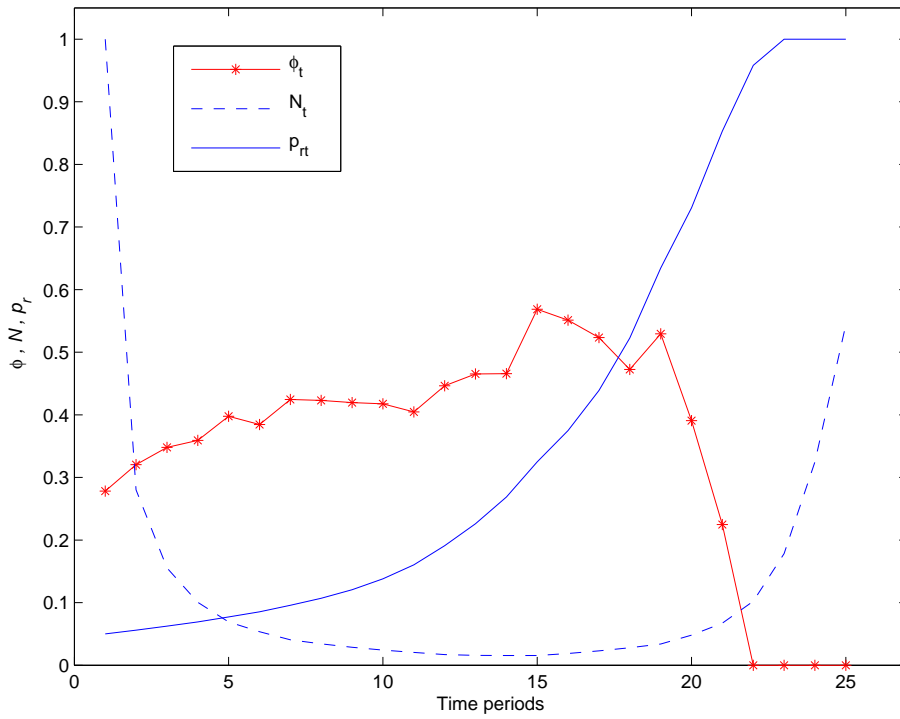


Figure 4: Evolution of (N_t, p_{rt}, ϕ_t) with $c_s = c = 0$

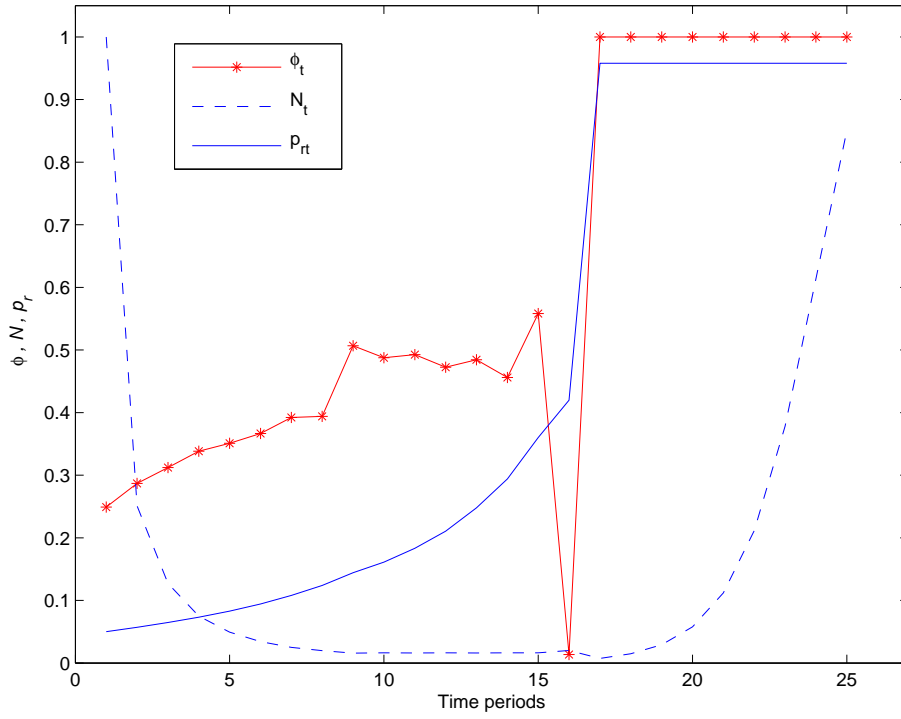


Figure 5: Evolution of (N_t, p_{rt}, ϕ_t) with $c_s > 0$ and $c = 0$

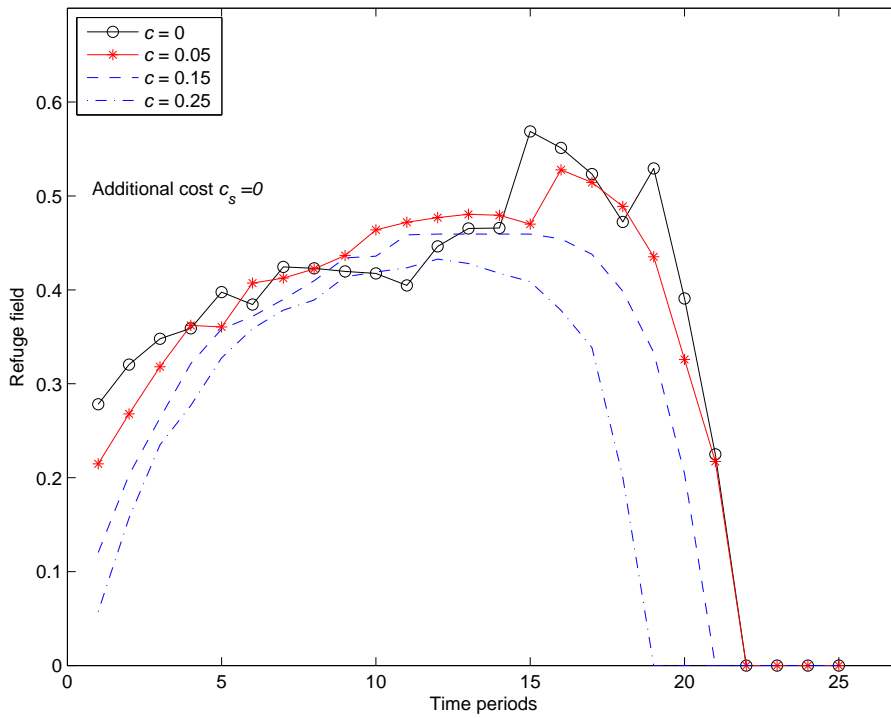


Figure 6: Comparative dynamics of the refuge policy with $c_s = 0$

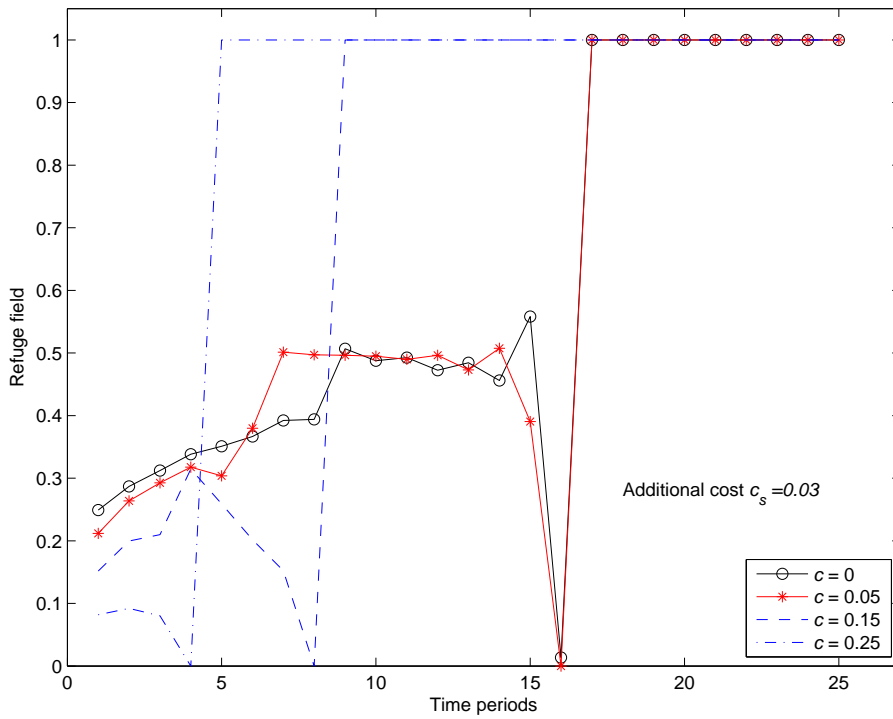


Figure 7: Comparative dynamics of the refuge policy with $c_s > 0$

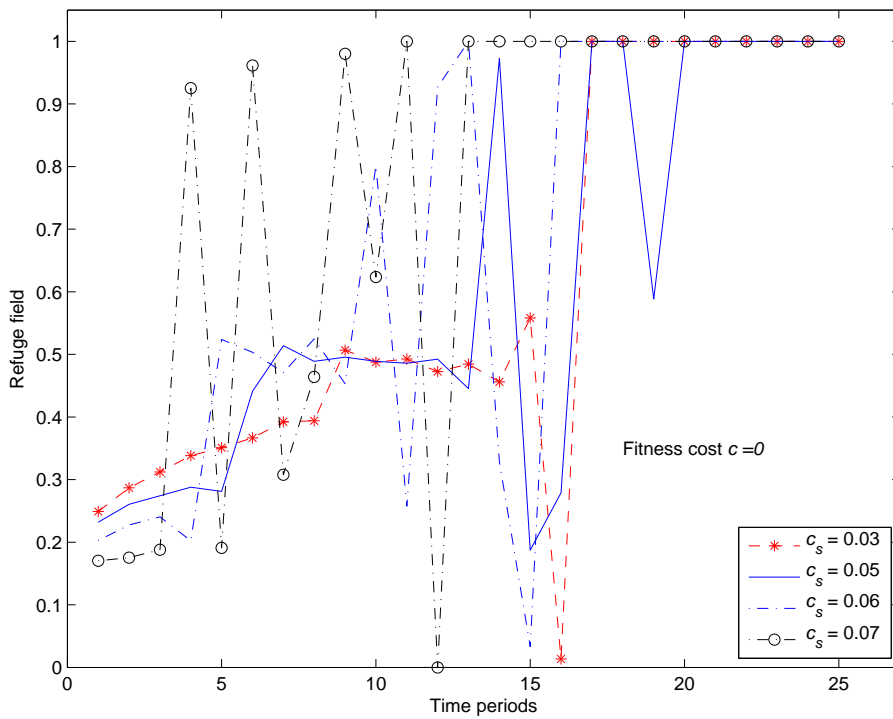


Figure 8: Comparative dynamics of the refuge policy and the cost premium (c_s) when $c = 0$

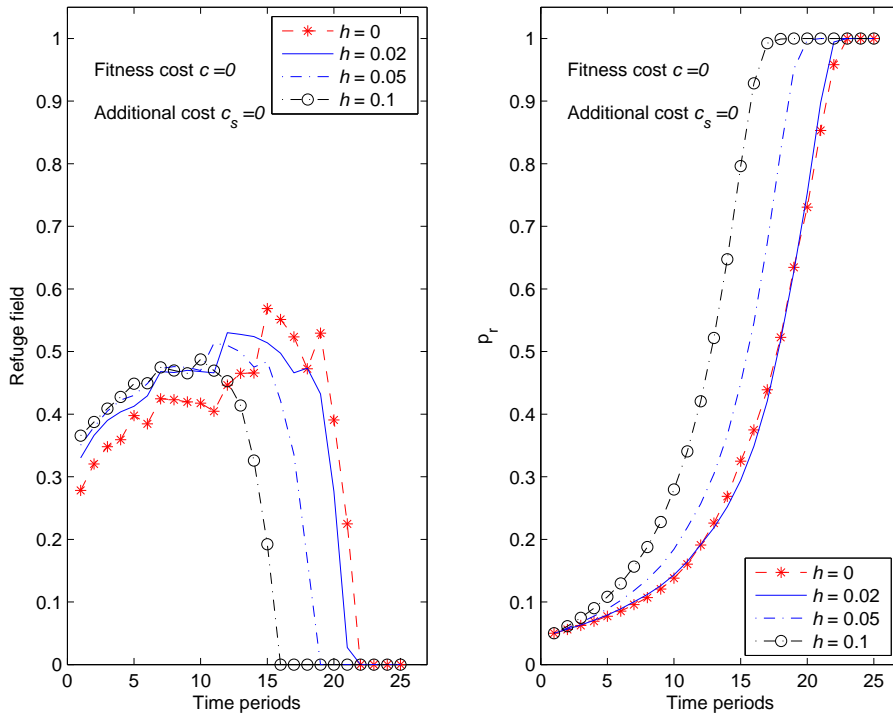


Figure 9: Comparative dynamics of the refuge policy and relative dominance (h) when $c = c_s = 0$

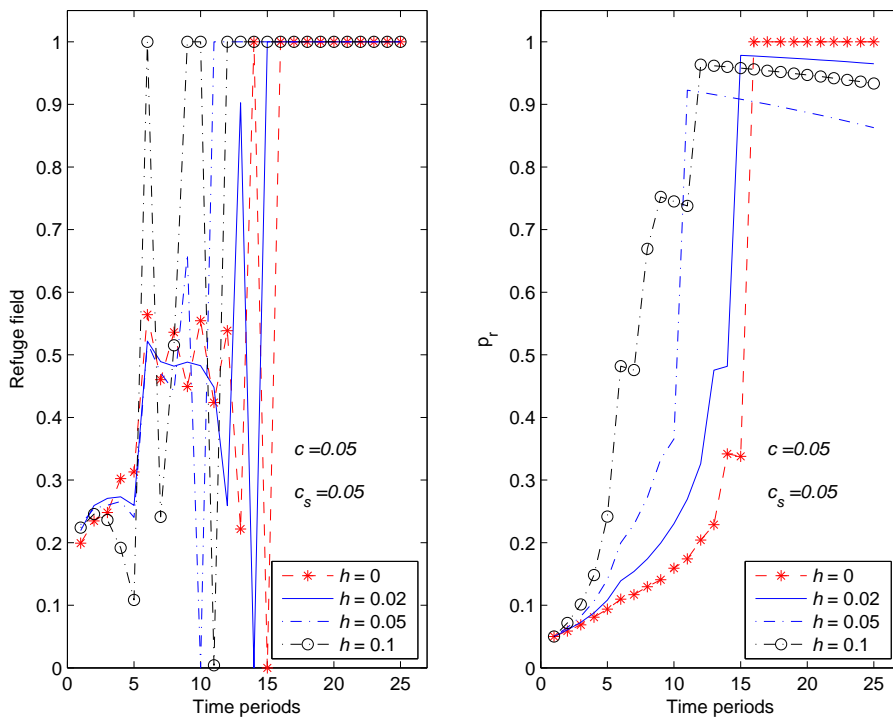


Figure 10: Comparative dynamics of the refuge policy and relative dominance (h) when $c = c_s = 0.05$