Mangaging Pest Resistance to Bt Crops with Dynamic Refuge Size Adjustments

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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. 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 is addressed via numerical simulations. We point out 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: pest resistance management, *Bt* crop, refuge policy, optimal control, (non-)renewable resource.

1 Introduction

It is widely recognized that the efficacy of pesticides or pest-toxic crop varieties to control target pest populations is lost over time when selection pressures result in increasing prevalence of resistance among these pest populations. From the perspective of natural resource economics, the use of pesticides or pest-toxic varieties affects two interdependent "biological resources": (i) the level of the pest population, a detrimental resource which causes crop damages and (ii) the susceptibility of this population to pesticides or pest-toxic crop varieties, a beneficial resource.[1] The goal of this paper is to contribute to the theory of the optimal use of pest-toxic varieties over time in a context where the two aforementioned biological resources are involved and to assess the existing economic literature on the subject.

Following early contributions in the 1970s-80s on chemical pesticide use, the advent of pest-toxic genetically modified (GM) crops has led to a renewed interest in the economic analysis of pest resistance management. 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, especially among environmental groups, that pest populations may adapt to the transgenic crop which may have particularly negative effects on organic farmers who use *Bt* sprays for pest control. Active pressure from these groups, along with the calls from scientists for regulation has had the result that 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*.

The aim of using refuge fields is to prevent a quick selection of the *Bt* resistant pest

[1]Several antibiotics have also lost their effectiveness in fighting several bacterial infections. For a review of this akin problem and references to the economic literature, see Herrmann and Laxminarayan (2010).

population, which may occur if all fields are planted with *Bt* crops. The intended result of placing refuges near *Bt* crops is that resistant insects emerging from *Bt* crops will mate with susceptible pests emerging from refuge fields. This preserves the susceptibility to *Bt* crops within the gene pool and thus provides a tool to manage pest resistance. Introducing a refuge field has two opposite effects on farmers' profits: on the one hand, leaving some of the crop unprotected in the refuge area causes immediate and future profit losses via higher pest prevalence. On the other hand, this strategy increases the pest population's future susceptibility to the *Bt* crop, thus decreasing pest pressure in the future and improving future crop protection, thereby increasing future profits.

The literature on pest resistance to pesticides has yet to reach any consensus on the nature of optimal use of pesticides. The seminal paper by Hueth and Regev (1974) gives an economic interpretation of the intertemporal externalities at stake, but does not indicate how pesticide use should be adjusted over time. Regev *et al.* (1983) present a simulation exercise in which the optimal annual quantity of a chemical pesticide first decreases, then increases, falls to zero, becomes positive again and finally falls to zero. Lazarus and Dixon (1984) develop a simulation model with two control variables, the proportion of corn fields planted with a soybean rotation to reduce future pest population intensity and the presence or absence of insecticide treatment on corn fields. The optimum is characterized by oscillations of these two control variables. Plant *et al.* (1985) develop a simulation model with timing of application as the control variable. They show simulations where optimal applications occur later and later during the growing season as years pass, others in which applications occur earlier and earlier, still others in which they are non-monotonous.

More recently, several articles on *Bt* crops reach contradictory results in terms of the optimal evolution of the refuge size through time. Arguing that the optimal evolution of refuges is first and foremost an empirical question, Secchi *et al.* (2006) run simulations with a realistic bioeconomic model and conclude that the optimal refuge is initially low, then increases to a maximum, and finally decreases over the remaining time horizon. They also

find that it may or may not be optimal to fully exhaust pest susceptibility at the end of the time horizon depending on whether the target pest recovers slowly or rapidly after the decrease in the population level caused by *Bt* plantings. Livingston *et al.* (2004) report results that are consistent with Secchi *et al.* (2006), using a simulation model to examine the evolution of *Bt* and insecticide resistance in two pest populations when refuges may be sprayed with insecticides. Laxminarayan and Simpson (2002) analytically derive the optimal refuge at the steady state and approximate the optimal refuge at the beginning of the time horizon with a more stylized model in continuous time. As opposed to Secchi *et al.* (2006), they find it optimal that no refuge is planted initially, and that pest susceptibility is exhausted in the long run when there is no fitness cost of resistance among resistant pests.[2] With a positive fitness cost of resistance however, they find an interior steady state in which pest susceptibility is not exhausted. In a similar model, Qiao *et al.* (2008) run simulations on the entire optimal refuge path. 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 refuge and no *Bt* crop, followed by a singular path of varying intermediate refuges. When the fitness cost of resistance is high, the optimal path is simply not to plant a refuge over some number of years at the beginning of the period of analysis, and then to plant an intermediate constant refuge level over the remaining time horizon. Applying 'singular-perturbation' reduction methods from the literature on dynamic systems, Grimsrud and Huffaker (2006) find that the size of the refuge should decrease monotonically over time with a full exhaustion of pest susceptibility. Qiao *et al.* (2009) simulate a pest's development of resistance to both a *Bt* toxin and a conventional pesticide, allowing for other crops to function as natural refuges for this pest. They find non-linear dynamics for refuges and conventional pesticide use with the possibility of alternating use of *Bt* cotton and pesticides to control the pest.

[2]In 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.

This paper will focus on pest-resistant varieties to argue that the apparent contradictions in the literature can mostly be attributed to important shortcomings in the biological models of Laxminarayan and Simpson (2002), Qiao *et al.* (2008, 2009) and Grimsrud and Huffaker (2006). First, these models permit some insects to experience logistic growth although they have previously died, either because they are susceptible to *Bt* crops or because they are subject to a fitness cost of resistance. Secondly, these authors use a continuous-time framework which, as we will show, cannot account for high selection pressure on non-resistant pests when no refuge is planted. Third, 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.[3] A key element of this strategy is that 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 their heterozygous progeny will die on GM crops. This central factor is not accounted for in the models of Laxminarayan and Simpson (2002), or Qiao *et al.* (2008). Fourth, the biological model of Grimsrud and Huffaker (2006) is based on the ad-hoc assumption that resistance evolves much more slowly than pest population and an inconsistent interaction in the dynamics of these two state variables.

We develop a stylized bioeconomic model where the biological component is compatible with the biology literature on the high-dose/refuge strategy. Our biological model is similar to that of Secchi *et al.* (2006), who simulate the optimal time-variant refuge for *Bt* corn

[3]All 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.

resistant to the European corn borer. We analytically identify the intertemporal effects on the pest population and its susceptibility occurring in relation to refuge fields. Furthermore, we illustrate the optimal, time-variant refuge by showing an exhaustive dynamic comparative exercise on parameter values based on simulations. 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 itself depends entirely on the model parameters (notably the overcost of *Bt* seeds and the fitness cost of resistance). We also compare our results with previous literature in terms of the optimal adjustment of refuge size over time and the extent to which particular events in the pest's lifecycle, the assumption of diploid versus haploid pests and the assumption of discrete rather than continuous time are essential for our analysis.

This paper is structured as follows. In section 2 we present a combined intertemporal, bioeconomic model where a pest population and its susceptibility to *Bt* seeds are determined endogenously as a function of refuge field size. The optimal refuge is characterized analytically in section 3 as a function of the state of the system and the corresponding implicit shadow values. Numerical simulations are used to address the socially optimal evolution of the dynamic system. In section 4, we discuss our model and compare it to the aforementioned bioeconomic models. We conclude in section 5.

2 The bioeconomic model in discrete time

We start with a biological model in discrete time, which is a modified version of the pioneering work by Alstad and Andow (1995) on pest resistance management with transgenic *Bt* crops and refuge fields. The environment is assumed to be deterministic, with selection driving the evolution of a pest population and its genetic composition over time. In biological terms, we assume that resistance is determined at a single locus with two alleles, where an allele can either be susceptible (s) or resistant (r) to *Bt*.[4] In this diploid context, each insect inherits

[4]As 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"."

one allele from its father and one from its mother, and is thus either a homozygous resistant (rr), homozygous susceptible (ss), or heterozygous (rs).

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, 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 average shares of resistant and susceptible alleles are respectively $p_r = N_r/(2N)$ and $p_s = N_s/(2N)$, with $p_r + p_s = 1$. Furthermore, we assume that there is one generation of insects per year, with non-overlapping generations. Each generation has two development phases: (1) migration of emerging adults, reproduction and density dependence and (2) genotype-induced mortality. These two steps are detailed below, while its differences as compared to the model of Alstad and Andow (1995) are discussed in Appendix C (online appendix).

2.1 The life cycle two stages

– **Stage 1**: migration of emerging adults, reproduction and density dependence. Larvae pupate and eclose as adult moths which migrate, reproduce and lay eggs. We assume a logistic growth model with a growth rate g and a carrying capacity K.[5] The growth rate is defined as the birth rate, b (the average number of larvae per adult, which is itself determined as the number of eggs laid per adult minus the number of larvae that die naturally in young stages), minus the natural mortality rate, which is equal to 1 because all adult moths die just after laying eggs, such that $g = b - 1$. 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

[5]We 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.

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).[6] At the end of stage 1, the average numbers of larvae per plant for each 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 $N_{ss,1} = {p_s}^2 N_1$.[7]

– **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, such 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 throughout the paper that $m_{rr} = 0$ and $m_{ss} = m_{rs} = 1$ (implying $h = 0$), such that the Bt toxin in the transgenic field causes all larvae of genotypes rs and ss to die, while it has no effect on rr larvae.[8]

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

[6]The 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 collide 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, $p_s p_r$.

[7]We 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.

[8]For 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 $m_{rr} = 0$.

the environment (Coustau *et al.*, 2000).[9]

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, *i.e.* $N_{i,2} = (1-\phi)N_{i,2}^{Bt} + \phi N_{i,2}^{ref}$. Specifically, $N_{rr,2} = (1-c)N_{rr,1}$, $N_{rs,2} = \phi N_{rs,1}$ and $N_{ss,2} = \phi N_{ss,1}$.

2.2 The laws of motion of the biological model

Combining the change in the pest population over the two stages of the life cycle in year t yields the average population per plant, N_i', for each of the three genotypes, with $i = rr, rs$ and ss at the beginning of year $t+1$. The three corresponding populations are: $N_{rr}' = (1-c)p_r^2 N_1$, $N_{rs}' = 2\phi p_r p_s N_1$ and $N_{ss}' = \phi p_s^2 N_1$. The fact that $p_r + p_s = 1$ and the definition of N_1 can be used to write our model as:

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

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

The bracketed terms in equations (1) and (2) are identical and relate to the logistic regeneration of the pest population and its density dependence (the latter via the fraction $-(N_r + N_s)/(2K)$). In equation (2), the term ϕ can be re-interpreted as the fitness of all susceptible pests (*i.e.* of type ss or rs), which can only survive on the refuge field. The last term in equation (1), $((1-c)N_r + \phi N_s)/(N_r + N_s)$, reflects the impact of random mating on the genotypic composition. This term is decreasing in N_s so long as the mortality of homozygous resistant pests, c, is lower than the mortality of heterozygous pests, $1-\phi$: if

[9]The actual fitness cost of resistance for the European corn borer and Bt corn is yet unknown, as no case of Bt resistance has been confirmed yet 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).

the share of susceptible alleles increases, resistant alleles are carried by less numerous homozygous resistant pests, and by more numerous heterozygous pests.[10] This implies that an increase in the stock of susceptible alleles contributes to the decay of resistant alleles as long as the mortality of the former pests is lower than that of the latter.

Turning to our variables of interest, we use an alternative formulation of the biological model in terms of the evolution of the average number of larvae per plant and the proportion of resistant alleles. Equations (1) and (2) can be used to derive these two variables at the beginning of year $t+1$ as $N' = (N'_r + N'_s)/2 =$ and $p'_r = N'_r/(N'_r + N'_s)$. This gives:

$$N' = f_N(N, p_r, \phi) = \left[(1-c){p_r}^2 + \phi(1-{p_r}^2)\right]\left[1 + g\left(1 - \frac{N}{K}\right)\right]N, \tag{3}$$

$${p_r}' = f_r(p_r, \phi) = \frac{(1-c){p_r}^2 + \phi p_r(1-p_r)}{(1-c){p_r}^2 + \phi(1-{p_r}^2)}. \tag{4}$$

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 $f_N(.)$ 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 $f_r(.)$ is increasing in p_r and decreasing in ϕ. The result is that a positive refuge causes an immediate loss 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 population, which confirms the intuition on the high-dose/refuge strategy given above.

Writing the change in allelic resistance p_r as a difference equation $\Delta p_r \equiv {p_r}' - p_r$ allows us to determine whether the pest population's susceptibility – the mirror image of allelic resistance – can be interpreted as a renewable resource:

$$\Delta p_r = \frac{(1-p_r){p_r}^2(1-c-\phi)}{(1-c){p_r}^2 + \phi(1-{p_r}^2)}. \tag{5}$$

[10] Formally, $N_{rr} = p_r^2 N = \frac{N_r^2}{2(N_r+N_s)}$, which is decreasing in N_s for a given N_r, while $N_{rs} = 2p_r p_s N = \frac{N_r N_s}{N_r+N_s}$, which is increasing in N_s for a given N_r.

For a zero fitness cost ($c = 0$), resistance is non-decreasing. With a positive fitness cost of resistance ($c > 0$), as long as $p_r < 1$, resistance increases over time if $\phi < 1 - c$ and decreases over time if $\phi > 1 - c$. It follows that for $c > 0$, pest susceptibility is 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 (3) and (4), we set Δp_r=0 and $\Delta N \equiv N' - N = 0$ to derive three steady-state configurations of the biological system. For any $\phi \neq 1 - c$, there are two distinct steady states, given by:

$$S_0 \equiv (N^{S_0}, p_r^{S_0}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{\phi} - 1 \right) \right], 0 \right), \tag{6}$$

$$\text{and} \quad S_1 \equiv (N^{S_1}, p_r^{S_1}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{1-c} - 1 \right) \right], 1 \right). \tag{7}$$

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

$$S_{\mathrm{i}} \equiv (N^{S_{\mathrm{i}}}, p_r^{S_{\mathrm{i}}}) = \left(K \left[1 - \frac{1}{g} \left(\frac{1}{1-c} - 1 \right) \right], p_r \in [0, 1] \right). \tag{8}$$

constitute steady states where p_r may take "interior" values. Under mild conditions on the parameter values, which are detailed in Appendix A 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, particularly the simultaneous motion of both state variables p_r and N, we draw a phase diagram in space $N \times p_r$ in Figure 1. The isoclines for N and p_r are the geometric loci where $\Delta N = 0$ and $\Delta p_r = 0$. The dynamic forces driving the system when out of the isoclines are represented by arrows. Three different configurations must be distinguished depending on whether $\phi \gtreqless 1 - c$. Setting $\Delta p_r = 0$ in equation (4) yields the isoclines $p_r = 0$ and $p_r = 1$ for the resistant allelic frequency. If the refuge area takes the critical value $\phi = 1 - c$, then the $\Delta p_r = 0$ isocline is horizontal at some level strictly between 0 and 1 (not shown in Figure 1). Setting $\Delta N = 0$

in equation (3) gives us the ΔN isocline as a function of p_r:

$$N(p_r) = K\left[1 - \frac{1}{g}\left(\frac{1}{(1-c){p_r}^2 + \phi(1-{p_r}^2)} - 1\right)\right]. \tag{9}$$

The shape of this isocline and the forces driving the pest population when out of the ΔN isocline depend on the value of ϕ for the refuge. [11] In Figure 1, we show which of the Δp_r and ΔN isoclines apply depending on whether $\phi \gtreqless 1-c$. Steady states S_0, S_1 and S_i lie at the intersection of the Δp_r and ΔN isoclines (the Δp_r isocline for $\phi = 1-c$ has been omitted).

A representative trajectory 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}), is represented in Figure 1. As the system evolves in discrete time, the trajectories are, strictly speaking, sequences of points. When the refuge area is positive and relatively small, $0 < \bar{\phi} < 1-c$, the driving dynamic forces are represented in Figure 1 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 monotonously increases over time (see equation (4)). The level of the pest population may initially decrease, in which case it eventually crosses the ΔN isocline. From that point of time, the pest population 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 until the point in time when the *Bt* corn has entirely lost its efficacy ($p_r = 1$). 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. A relatively large refuge reduces resistance over time. The pest population increases monotonously over time from the initial state and the dynamic system converges to S_0. Finally, we consider the case where the refuge area takes

[11] The forces driving the pest population N when out of 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 $\partial^2 N(p_r)/\partial {p_r}^2 = 2K(1-c-\phi)[\phi - 3{p_r}^2(1-c-\phi)]/(g[\phi + {p_r}^2(1-c-\phi)]^3)$ 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 $\phi > (<)(1-c)3p_r^2/(1+p_r^2)$. As 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 else be concave.

the critical value $\bar{\phi} = 1 - c$. Pest resistance $p_r(t)$ remains unchanged and equal to its initial value p_{r1} and the pest population converges to $N^{S_\text{i}} = N^{S_1}$, such that the interior steady state S_i is reached.[12]

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 (7)). 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 (6) is evaluated at $\phi > 1 - c$). Finally, there is only one constant refuge size which allows to reach the interior steady state S_i, in which resistance is neither eradicated nor fully spread in the pest population, but controlled for to be 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.

2.3 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 also 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:

$$C(N, p_r, \phi) = \alpha f_N(N, p_r, \phi) + c_s(1 - \phi), \tag{10}$$

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

[12]The 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 $\bar{\phi} \gtreqless 1 - c$ (either from above or from below), the dynamic system will approach the corresponding steady-state configuration for sufficiently long sequences of ϕ.

encountered by farmers on the Bt and refuge fields and the cost premium of GM plantings:

$$V(N_1, p_{r1}) = \min_{0 \leq \phi \leq 1} \sum_{0}^{T} \delta^t C(N, p_r, \phi), \tag{11}$$

subject to the laws of motion of the state variables, p_r and N, as defined in (3) and (4) and where the time horizon $T < \infty$ is exogenous.[13]

3 The optimal refuge policy

We first characterize the necessary conditions which a refuge field has to satisfy and analyze in detail the last period optimal refuge field, before numerically addressing the evolution of the dynamic system.

3.1 Characterization of an interior solution for the refuge field

The Lagrangian function for our problem is:

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

The unknowns in this problem are the series $\{\phi_t\}$, $t = 0, 1, \ldots, T$, and $\{N_t, p_{rt}, \lambda_t, \mu_t\}$, $t = 0, 1, \ldots, T+1$ (see Conrad, 1999, for 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:

$$c_s - \delta\mu_{t+1} \frac{(1-c)(1-p_{rt})p_{rt}^2}{[\phi_t + p_{rt}^2(1-c-\phi_t)]^2} = \left(1 + g\left(1 - \frac{N_t}{K}\right)\right) N_t(1-p_{rt}^2)(\alpha - \delta\lambda_{t+1}) \tag{12}$$

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 affect negatively the objective function.

The left-hand side of equation (12) 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

[13] We 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.

resistance. The right-hand side of (12) 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 $\phi_t = 1$.

3.2 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 $\lambda_{T+1} = \mu_{T+1} = 0$. Omitting the time indice T, the partial derivative of the Lagrangian function with respect to the control variable thus becomes:

$$\frac{\partial L}{\partial \phi} = \delta^T \left\{ c_s - \alpha(1 - p_r^2) \left[1 + g \left(1 - \frac{N}{K} \right) \right] \right\}.$$

Depending on whether $\partial L / \partial \phi \lesseqgtr 0$, we can characterize the extreme controls for the refuge as:

$$\begin{aligned} \phi_T &= 0 \Leftrightarrow {p_r}^2 < 1 - \tfrac{c_s}{\alpha N[1+g(1-N/K)]}, \\ \phi_T &= 1 \Leftrightarrow {p_r}^2 > 1 - \tfrac{c_s}{\alpha N[1+g(1-N/K)]}. \end{aligned} \tag{13}$$

In the benchmark case where there is no cost premium for *Bt* seeds ($c_s = 0$), the first of the above equations simplifies to $\phi_T = 0 \Leftrightarrow p_r < 1$, indicating that no refuge should be planted in the last period T so long as resistance is not at its maximum value. Indeed, planting nothing but *Bt* crops in the last period is the most effective way to reduce crop damage. This last strategy obviously causes resistance to jump to its maximum value at the end of that period, but this does not carry any social cost beyond time horizon T.

With a cost premium of *Bt* seeds and $0 < c_s < \alpha \left[1 + g(1 - N/K)\right] N$, equation (13) specifies a concave hyperbola 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, it is optimal to incur the additional cost of *Bt* seeds by planting no refuge ($\phi = 0$). The opposite case of $\phi = 1$ sees the refuge at its maximum because incurring the additional cost of *Bt* seed is not efficient.

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 present numerical simulations and address the optimal evolution of the refuge and the state dynamics of the system. The baseline parameters which we will retain unless specified differently are given in Table 1.[14]

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 $g_1 = 1$, $g_2 = 10$, $K_1 = K_2 = 22$).

[14] In 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.

3.3 Comparative dynamic analysis of the fitness cost of resistance and the cost premium of *Bt* seed

We first analyze the dynamics of the system when the fitness cost of resistance and the cost premium of *Bt* seeds satisfy $c = c_s = 0$. In the absence of a positive fitness cost, the susceptibility of the pest population is a non-renewable resource. Furthermore, from (13), no refuge should be planted in the last period ($\phi_T = 0$). As shown in Figure 2, the optimal refuge in this case 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 beyond the time horizon of the economic program). This refuge path is coherent with that described by Secchi *et al.* (2006), but differs from the monotonously decreasing path obtained by Grimsrud and Huffaker (2006), as well as from the no-refuge path Qiao *et al.* (2008) find when there is no cost premium of *Bt* seeds (as we will show later). In section 4, we will discuss the models and results of these and other authors more thoroughly.

A different kind of refuge path arises when the cost premium of *Bt* seeds is non-zero ($c_s > 0$) and the fitness cost is $c = 0$, as shown in Figure 3. In this case, the refuge not only slows down the emergence of resistance, but it is also cheaper. The optimal refuge is initially increasing, then drops to a very low value, and finally reaches $\phi_T = 1$. Over the planning horizon, the refuge is always strictly positive, therefore pest susceptibility is never completely exhausted. It is optimal in the initial phase to plant some positive, intermediate refuge field, which slows down the evolution of resistance. But the substantial increase in resistance occurring after the sharp decline in the refuge makes further use of the more costly *Bt* seed sub-optimal (see Figure 3). If ϕ were maintained at 1 beyond the time horizon, the

system would eventually reach the interior steady state S^i.

Figures 4 and 5 show the change in the size of the socially optimal refuge field when there is a positive fitness cost ($c > 0$). In the absence of cost premium of *Bt* seeds ($c_s = 0$), results are similar to the case where $c = 0$. As can be seen from Figure 4, 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. This 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 shown in Figure 5.[15]

The impact of a positive fitness cost on the optimal refuge was not identified adequately in the previous literature thus far (Secchi *et al.* (2006) are the only authors with results consistent with ours, but they do not examine the effect of a positive fitness cost). In summary, our numerical evidence suggests that the susceptibility to *Bt* should generally 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 advantage in the longer run to face a resistant pest population, the steady-state level of which 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. Table 2 shows the intertemporal cost farmers face due to crop damage in each situation, $V(p_{r1}, N_1; \phi_t)$, when the refuge adjusts optimally over time, and when it is constrained to be constant over time, $V(p_{r1}, N_1; \bar{\phi})$. Of

[15]Figure 5 shows that for $c = 0.25$, $\phi = 0$ in the fourth period, and therefore susceptibility is exhausted at the end of that period.

course, this additional constraint on ϕ_t necessarily increases costs, and we always observe that $V(p_{r1}, N_1; \bar{\phi}) > V(p_{r1}, N_1; \phi_t)$. Table 2 shows that the constant refuge $\bar{\phi}$ is increasing in the additional cost of *Bt* seed, 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
$c_s = 0$	$V(p_{r1}, N_1; \phi_t)$	0.1230	0.0723	0.0244	0.0088
	$V(p_{r1}, N_1; \bar{\phi})$	0.1315	0.0884	0.0387	0.0162
	$\bar{\phi}$	0.4033	0.3567	0.2467	0.1367
	$(\Delta V / V(.; \phi_t))$	(6.91%)	(22.26%)	(58.61%)	(84.09%)
$c_s = 0.03$	$V(p_{r1}, N_1; \phi_t)$	0.3902	0.3340	0.2434	0.1566
	$V(p_{r1}, N_1; \bar{\phi})$	0.4430	0.4196	0.4063	0.4052
	$\bar{\phi}$	0.4367	0.4100	0.3833	0.3833
	$(\Delta V / V(.; \phi_t))$	(13.53%)	(25.63%)	(66.93%)	(158.75%)
$c_s = 0.06$	$V(p_{r1}, N_1; \phi_t)$	0.6257	0.5527	0.4040	0.2588
	$V(p_{r1}, N_1; \bar{\phi})$	0.7385	0.7256	0.7194	0.7182
	$\bar{\phi}$	0.4633	0.4500	0.4433	0.4467
	$(\Delta V / V(.; \phi_t))$	(18.03%)	(31.29%)	(78.07%)	(177.51%)

Table 2: Comparative analysis of inter-temporal 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.

A difficulty resides however in the fact that the shape of the optimal refuge path can differ markedly when parameters values vary slightly. As shown in Figure 6 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 important variations in the trajectory of the optimal refuge policy, as discussed before and as evidenced by Figures 4 and 5. 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.

3.4 Comparative dynamic analysis of other 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 shown by the increasing size of the constant refuge. 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 decreasing momentarily the refuge size (and thereby controlling susceptible pests). Figure 7 shows this sawtooth pattern of the refuge field for time horizons $T = 55$ and $T = 100$.

Comparative dynamics on the discount rate show 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 intuitive result is due to the fact that a 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 which would improve pest population control in the future.

Finally, equation (10) shows that the economic objective is linear in both the damage

T	15	25	55	100
$V(p_{r1}, N_1; \phi_t)$	0.1414	0.3340	0.9203	1.2218
$V(p_{r1}, N_1; \bar{\phi})$	0.2977	0.4196	1.0681	1.4417
$\bar{\phi}$	0.34	0.41	0.51	0.52
$(\Delta V/V(;\phi_t))$	(110.54%)	(25.63%)	(16.06%)	(18.00%)
ρ	0	0.03	0.15	0.25
$V(p_{r1}, N_1; \phi_t)$	0.4438	0.3340	0.1793	0.0950
$V(p_{r1}, N_1; \bar{\phi})$	0.5631	0.4196	0.2898	0.2065
$\bar{\phi}$	0.4233	0.41	0.40	0.37
$(\Delta V/V(;\phi_t))$	(26.88%)	(25.63%)	(61.63%)	(117.34%)

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

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 was formerly analyzed.

4 Discussion and significance of our biological modeling

We will now briefly point out several important characteristics distinguishing our model from Laxminarayan and Simpson (2002), Grimsrud and Huffaker (2006) and Qiao *et al.* (2008, 2009).

First, these models are in continuous time and do not allow for jumps of the state variables, as discrete models do (Otto and Day, 2007). Laxminarayan and Simpson (2002) and Qiao *et al.* (2008) both find that no refuge should be initially planted. However, in their continuous-time model, such an initial policy maintains an intermediate level of resistance – while its discrete-time counterpart would yield a jump to the maximum resistance of $p_r = 1$ within one year. Their policy recommendation is therefore flawed because it acknowledges 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.[16] In the second place, the biological models developed by these authors have several flaws that

[16]Such 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); but such a feature is not modeled in Laxminarayan and Simpson (2002) and Qiao *et al.* (2008).

we discuss below using the discrete-time versions of their models.[17]

In discrete time and with our notation, the model used in Laxminarayan and Simpson (2002) and Qiao *et al.* (2008) becomes:

$$n'_r = n_r \left[1 - c + g\left(1 - \frac{n_r + n_s}{K}\right)\right], \tag{14}$$

$$n'_s = n_s \left[1 - (1-\phi)z + g\left(1 - \frac{n_r + n_s}{K}\right)\right], \tag{15}$$

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, these equations are inconsistent with the effect of Bt crops on susceptible pests and with the effect of the fitness cost on resistant pests that the authors describe: neither do all susceptible insects die on the Bt crop when $z = 1$, nor do all resistant insects experience a fitness cost of resistance. If it were the case, the right-hand terms of equations (14) and (15) would respectively be multiplied by $(1-c)$ and ϕ. [18] This shortcoming becomes evident, for example, when considering equation (15): the assumption that $z = 1$ (all susceptible insects die on Bt fields) means that the population of susceptible insects should fall to zero after one period when all fields are planted with Bt varieties (that is when $\phi = 0$), whereas (15) defines this population as positive (given by $n'_s = n_s g\left(1 - \frac{n_r+n_s}{K}\right)$). Second, the populations of resistant and susceptible insects interact only via density-dependence – while our equations (1) and (2) also exhibited the impact of random mating and reproduction on the population's genotypic composition, a mechanism which lies at the core of the high-dose/refuge strategy. A noteworthy consequence is that the population of resistant insects is necessarily increasing over time in this model if the fitness cost of resistance is zero, as long as the population is

[17] The discrete-time analogs to these continuous-time model 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).

[18] If all insects experienced genotype-dependent mortality followed by logistic growth, the numbers of resistant and susceptible insects, respectively, would first decrease to $(1-c)n_r$ and $\phi h n_s$, then increase to $n'_r = (1-c)n_r\left[1 + g\left(1 - \frac{(1-c)n_r + \phi z n_s}{K}\right)\right]$ and $n'_s = \phi n_s z\left[1 + g\left(1 - \frac{(1-c)n_r + \phi z n_s}{K}\right)\right]$. If all insects experienced logistic growth followed by genotype-dependent mortality, these numbers would first increase to $n_i\left[1 + g\left(1 - \frac{n_r+n_s}{K}\right)\right]$ $(i = r, s)$, then decrease to the same n'_r and n'_s as before.

below its carrying capacity, as shown by equation (14). However, in our model the number of resistant alleles is possibly decreasing through time in this particular case, as shown by equation (1). We may also 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 the Appendix B. This is, we believe, inconsistent with the intuition of the high-dose/refuge strategy, which should be worth adopting even when Bt and refuge seeds sell at the same price.

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:[19]

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

These equations are incompatible with the anticipated effects that all insects of genotypes rs and ss die on the Bt crop, and also do not cohere with the notion that all insects of genotype rr experience a fitness cost of resistance: if it were the case, the right-hand terms of each of these two equations would respectively be multiplied by $(1-c)$ and ϕ for the same reason as described above. Their biological model is thus inconsistent with the expected effects of Bt crops and of the fitness cost on pest mortality.

Grimsrud and Huffaker (2006) apply the geometric singular perturbation theory to pest resistance management. Singular perturbation reduction methods simplify the analysis of dynamic problems by reducing their dimension. They are valid for systems in which some variables change on widely different time scales (e.g. Jones, 1994). The authors justify the use of these methods by claiming that the frequency of resistant alleles changes more slowly than the pest population. Yet, there is no reason why this should be the general case. Rather, the speed at which either state variable evolves should be determined endogenously

[19] Qiao *et al.* (2009) use a two-locus four-allele model to simulate resistance evolution to both a Bt toxin and a conventional pesticide. We simplify their model by considering a one-locus two-allele model of resistance to Bt toxin alone, and by keeping our simplifying assumption that all homozygous susceptible and heterozygous pests survive on refuge and die on Bt crops, as detailed in Appendix D (online appendix).

as occurs in our model, as opposed to adding an ad hoc parameter which slows down the evolution of resistance (Grimsrud and Huffaker, 2006, calibrate this parameter to $\epsilon = 0.1$). A further modeling assumption of Grimsrud and Huffaker (2006) is that the evolution of resistance is independent from pest mortality rates on transgenic fields. In other words, the proportions of resistant, heterozygous or susceptible pests dying on *Bt* crops do not affect the change in resistance in their model, while this should obviously be the case. Finally, and in line with the models discussed above, logistic growth and mortality on *Bt* crops are written additively in their paper, while the correct formulation would have a multiplicative form.

The differing behavior of these models is shown in Figures 8 and 9, which represent the evolution of our biological model and of the discrete-time analogue to these authors' biological model, assuming a constant refuge $\bar{\phi} = 0.5$, a growth rate $g = 1$, a carrying capacity $K = 1$, no fitness cost of resistance ($c = 0$), an initial resistance $p_{r0} = 0.01$ and an initial pest population $N_0 = K$.

Figure 8 is drawn for a time horizon of $T = 100$. In line with our discussion above, the population of resistant insects increases over time when using the discrete-time version of the model by Laxminarayan and Simpson (2002), while the number of resistant alleles in our model first decreases, and remains below its initial level during the whole time horizon. At the same time, the population of susceptible insects decreases in their model, less smoothly than the number of susceptible alleles in our model. As a result, the pest population in their model first decreases, and then increases back to the carrying capacity after 33 years, when the pest resistance becomes equal to 1. This contrasts with our biological model, where the pest population remains below its steady state across the entire, while pest resistance increases more slowly. At last, in the simplified discrete-time version of the model by Qiao et al. (2009), *Bt* crops do not decrease the number of susceptible alleles close to zero, whereas this does happen with our model.

For a time horizon $T = 4000$, Figure 9 compares our model with two versions of the

Grimsrud and Huffaker (2006) model, in which we set the dominance of resistance h to either 0.5, as was the case in their article, or to 0 as in ours.[20] The number of susceptible alleles first decreases, then increases, and lastly decreases again in both versions of their model, whereas it constantly decreases in our model. There is no rationale supporting this non-monotonous change in susceptible alleles, which we believe results from the lack of a coherent interdependence in their dynamics of the two state variables N and p_r (from which the dynamics of the variable N_s is recalculated).

5 Conclusions

We have presented a combined entomological-economic model, where a pest population damages a crop yield. Crop damage can be diminished by using genetically modified organisms, such as *Bt* seeds, which express genes which produce a lethal toxin. This comes at a cost, however, of decreasing the susceptibility of the pest population. In fact, this occurs 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 (or equivalently, the effectiveness of the *Bt* seed), is to use refuge fields.

Our paper has focused 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, notably 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

[20] With a definition of the dominance of resistance h analogous to ours, Grimsrud and Huffaker (2006) claim that "available empirical evidence does not favor any particular dominance value" and set h to 0.5 in all their analysis. We believe that both their claim and their calibration of h are at odds with the relevant literature, which argues for a level of h close to zero (see footnote 8). In Figure 9 we set their parameters g, K and R_2 to zero. The discrete-time analogue to their model is then written: $N' = \left[1 + (\phi + (1-\phi)p_r(p_r + 2(1-p_r)h))\, g\left(1 - \frac{N}{K}\right) - (1-p_r)\left(1 - p_r + 2p_r(1-h)\right)(1-\phi)\right] N$ and $p_r' = \left[1 + \epsilon(1-p_r)\left(p_r + (\phi + (1-\phi)h)\right)(1-2p_r) - (1-p_r)\phi\right) g\left(1 - \frac{N}{K}\right)\right] p_r$.

that the trajectory of the optimal refuge policy first depends on whether or not *Bt* seeds are priced as refuge seeds. When there is no difference in seed prices, the optimal refuge starts at an intermediate level, increases, then decreases and falls down 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 where the refuge is periodically decreased from its maximum size for one period in order to lower pest prevalence. In all simulations where *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 problem ends. However, we find evidence that some susceptibility should be preserved in situations where the social benefits of using *Bt* are the lowest, which happens when the fitness cost of resistance is zero (*i.e.* there is no additional mortality among resistant insects) and *Bt* seeds are more expensive than refuge seeds. 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 an effective knowledge of the biological and economic parameters pertaining to the pest/crop interaction of interest, which may pose certain challenges for public regulators. In particular, the biological parameters characterizing pests targeted by *Bt* crops are not always well-known as evidenced by the variety of values used for calibration in the literature.

We have also outlined the importance of accurately modeling the effects of *Bt* crops and of the fitness cost of resistance on pest populations. Irreconcilable results in the previous economic literature on optimal time-variant refuge size may be attributable to inadequate features of the biological models developed in several articles. We have highlighted the need to accurately and consistently depict which pests die and which pests reproduce on both, *Bt* crops and in refuges. The importance of modeling the gene pool with a diploid population to capture the effects of random mating on the share of susceptible and resistant alleles, which

is at the core of the high toxin dose / refuge strategy defined by population geneticists, has also been presented. Finally, we have pointed out the shortcomings of continuous-time modeling in comparison to our approach of modeling in discrete time.

Further research may look into the administrative cost of adjusting the refuge field, the cross-dynamics of the *Bt* crop/refuge strategy and conventional pesticides, as well as when transgenic crops with different toxins are available. However, introducing such additional complexity into the model will be 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.

Appendix

A Stability analysis of the biological model

The interested reader can 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 (6) – (8) can be addressed by evaluating, at each steady state, the Jacobian matrix J of the linearized counterpart to (3) and (4), given by:

$$J = \begin{pmatrix} \frac{\partial f_N(N,p_r,\phi)}{\partial N} & \frac{\partial f_N(N,p_r,\phi)}{\partial p_r} \\ \frac{\partial f_r(p_r,\phi)}{\partial N} & \frac{\partial f_r(p_r,\phi)}{\partial p_r} \end{pmatrix} \tag{A–1}$$

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:

S	$\{\, \nu_1, \nu_2 \,\}$
S_0	$\{\, 1, \quad 2-(1+g)\phi \quad \}$
S_1	$\{\, \frac{\phi}{1-c}, \; 1+c-(1-c)g \,\}$
S_i	$\{\, 1, \quad 1+c-(1-c)g \,\}$

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 only can 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 is 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 $0 \leq Tr \leq 2$.[21] 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.

Online appendix

B Online appendix: 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 notations, the optimization problem in the model of Qiao *et al.* (2008) when there is no overcost of Bt seeds is:

$$\min_{\phi} \int_0^{\infty} e^{-\rho t}[\alpha(n_r + n_s)]dt, \tag{B–1}$$

subject to the dynamic constraints of the model,

$$\dot{n}_r = [g\,(1 - n_r - n_s) - c]n_r, \tag{B–2}$$

$$\dot{n}_s = [g\,(1 - n_r - n_s) - (1-\phi)h]n_s. \tag{B–3}$$

[21] A 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.

Omitting the time indices, the current value Hamiltonian function associated with this dynamic problem is:

$$\begin{aligned} H(n_r, n_s, \phi, \lambda_r, \lambda_s) &= -\alpha(n_r + n_s) + \lambda_r[g\,(1 - n_r - n_s) - c]n_r \\ &\quad +\lambda_s[g\,(1 - n_r - n_s) - (1 - \phi)h]n_s, \end{aligned}$$

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 the following necessary conditions:

$$\dot{\lambda}_r - \rho\lambda_r = \alpha - \lambda_r[g\,(1 - 2n_r - n_s) - c] + \lambda_s g n_s, \tag{B–4}$$

$$\dot{\lambda}_s - \rho\lambda_s = \alpha + \lambda_r g n_r - \lambda_s[g\,(1 - n_r - 2n_s) - (1 - \phi)h]. \tag{B–5}$$

The Hamiltonian function is linear in the control. We define the switching function as the partial derivative of the Hamiltonian function with respect to the control variable:

$$\Omega(t) \equiv \partial H/\partial\phi = \lambda_s h n_s.$$

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 $\lambda_s = 0$.

We easily verify that there cannot be a singular path of the control variable with $n_s = 0$. In fact, the above state equation can be used to show that, 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. Setting λ_s and $\dot{\lambda}_s$ to zero in (B–5) above, we find that we must have:

$$\lambda_r n_r = -\frac{\alpha}{g}. \tag{B–6}$$

Differentiating (B–6), 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–2) and (B–4), use equation (B–6) and $\lambda_s = 0$ to find that this last equation simplifies to:

$$\lambda_r n_r = 0. \tag{B–7}$$

Equations (B–6) and (B–7) are uncompatible. Therefore, no singular path to this minimization problem exists when $c_s > 0$.

In the minimization problem, the refuge variable ϕ only contributes to increase the population of susceptible insects. This population, together with resistant insects, should be kept minimal together with the population of resistant insects to minimize losses as defined in the objective function. Therefore, since there exists no singular control to this linear optimization problem, it must be that ϕ is always kept equal to zero. In other words, refuges are never warranted in this model when no cost premium for *Bt* seeds applies.

C Online appendix: A discussion of the assumptions of our biological model

Our biological model includes several modifications relative to the intial model of Alstad and Andow (1995) which merit discussion. As opposed to Alstad and Andow (1995), 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) and has been included in some previous bioeconomic models of the refuge strategy (Laxminarayan and Simpson, 2002; Qiao *et al.*, 2008). We also make a view changes to the initial model for the sake of simplicity. They do not qualitatively affect our topic of concern in this paper, *i.e.* how refuge size should be adjusted over time. Our stages 2 and 3 happen in the opposite order compared to to Alstad and Andow. Our life cycle is consistent with the ordering adopted in other biological models of refuges, such as Onstad *et al.* (2002), and allows the evolution of resistance to be a function of the past level of resistance only, while being independent of the pest population level. Our assumption of one generation of insects per year differs from Alstad and Andow, who model two generations

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 where they emerge, and recent work has shown that the ECB probably mates at a more restricted spatial scale than previously assumed (Dalecky *et al.*, 2006). We assume that resistance to the *Bt* toxin is completely recessive whereas Alstad and Andow (1995) assume that around 2.6% of insects of genotype rs, and 0.1% of insects of genotype ss, survive on *Bt* corn. The logistic function that we use to characterize density dependence differs from the functional form chosen by Alstad and Andow (1995), who assume that on each crop, $N(1+4N)^{0.7}$ of the N surviving larvae die of density dependence.

D Online appendix: a 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 and heterozygous pests survive on refuge and die on *Bt* crops (the correspondence with their notations and assumptions is given in Table 4).

For each genotype $i = rr, rs$ or ss, define f_i as the fraction of genotype i, MR_i is 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. Their model with only one locus of two alleles is written (as defined in their Appendix 1 and in their equation (1)):

$$\dot{N}_i = f_i g N(1-N) - N MR_i,$$

Variable	Our notations and assumptions	Notations in Qiao et al. (2009)
pest population	N	D
homozygous resistant pests	rr	xx
heterozygous pests	rs	xX
homozygous susceptible pests	ss	XX
proportion of susceptible alleles	$p_s = 1 - p_r$	w
land fraction of Bt crops	$1 - \phi$	lf_{bt}
land fraction of refuges	ϕ	lf_{nbt}
mortality rate $m_{rr,Bt} = m_{rr,ref}$	c	r_{bt}
mortality rate $m_{ss,Bt}$	1	h_{bt}
mortality rate $m_{ss,ref}$	0	0
mortality rate $m_{rs,Bt}$	1	$h_{bt}d_{bt} + r_{bt}(1 - d_{bt})$
mortality rate $m_{rs,ref}$	0	$r_{bt}(1 - d_{bt})$

Table 4: Alternative notations and assumptions

with

$$MR_i = f_i \left[(1-\phi)m_{i,Bt} + \phi m_{i,ref}\right].$$

These two equations imply that:

$$\dot{N}_i = \left[g(1-N) - (1-\phi)m_{i,Bt} - \phi m_{i,ref}\right] f_i N.$$

Given our assumptions on mortality rates (summarized in Table 4), and given the genotype fractions $f_{rr} = {p_r}^2$, $f_{rs} = 2p_r p_s$ and $f_{ss} = {p_s}^2$, this equation implies:

$$\begin{cases} \dot{N}_{rr} = \left[g(1-N) - c\right] {p_r}^2 N, \\ \dot{N}_{rs} = \left[g(1-N) - (1-\phi)\right] 2p_r p_s N, \\ \dot{N}_{ss} = \left[g(1-N) - (1-\phi)\right] {p_s}^2 N. \end{cases}$$

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

$$\begin{cases} \dot{N}_r = \left[g(1 - \frac{N_r+N_s}{2}) - \frac{cN_r + (1-\phi)N_s}{N_r+N_s}\right] N_r, \\ \dot{N}_s = \left[g(1 - \frac{N_r+N_s}{2}) - (1-\phi)\right] N_s. \end{cases}$$

Using the same methodology as in the paper, the discrete-time analog of this model is:

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

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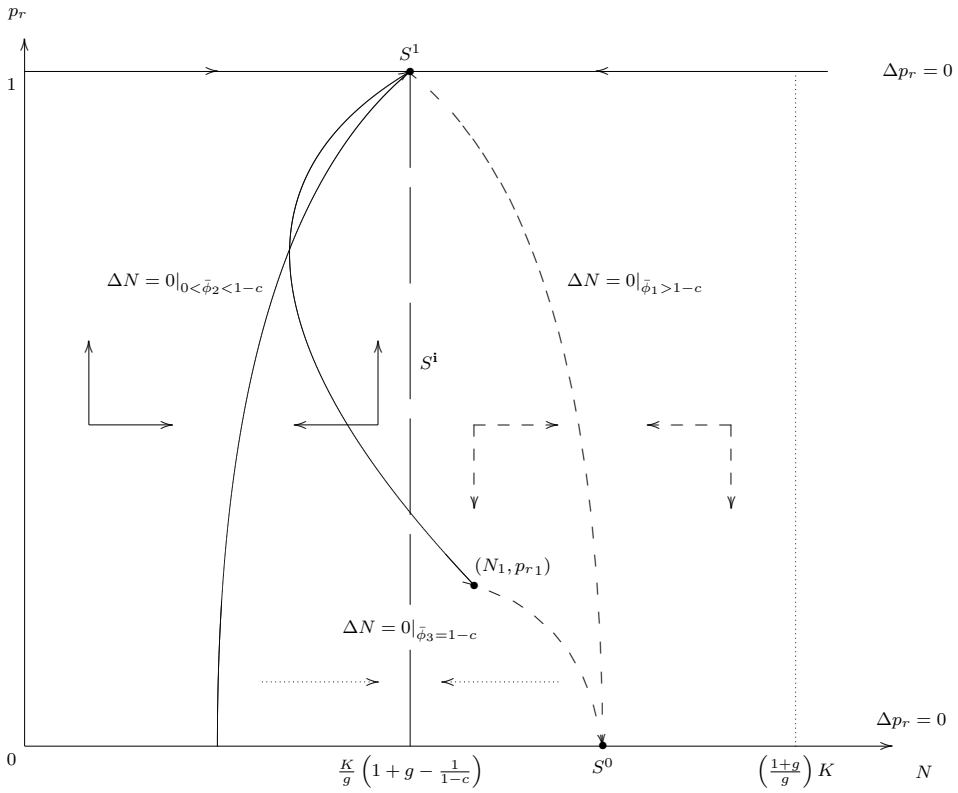

Figure 1: The phase diagram

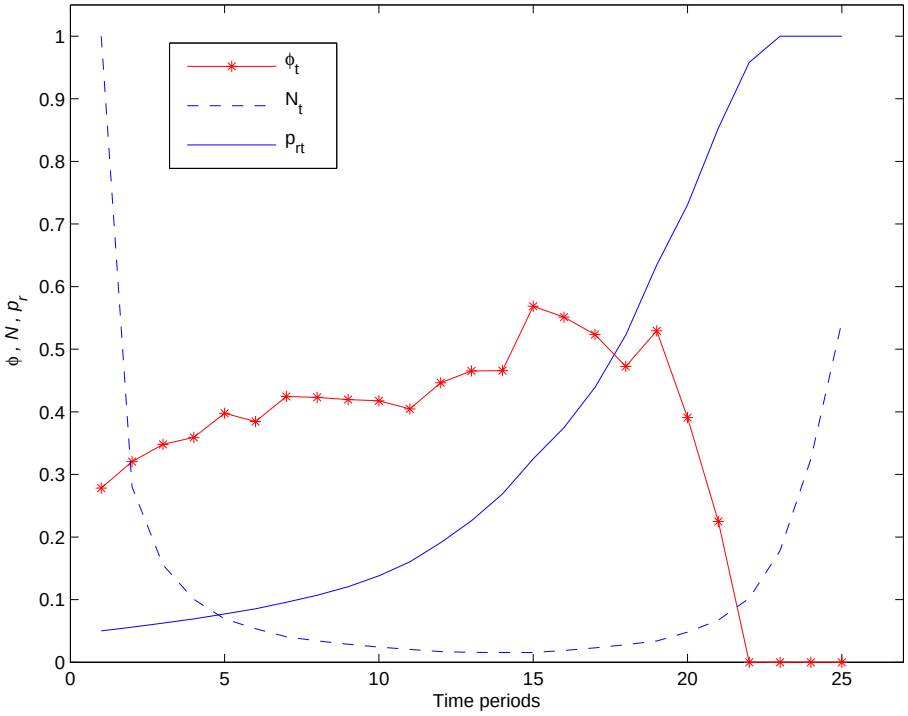

Figure 2: Evolution of (N_t, p_{rt}, ϕ_t) with $c_s = c = 0$

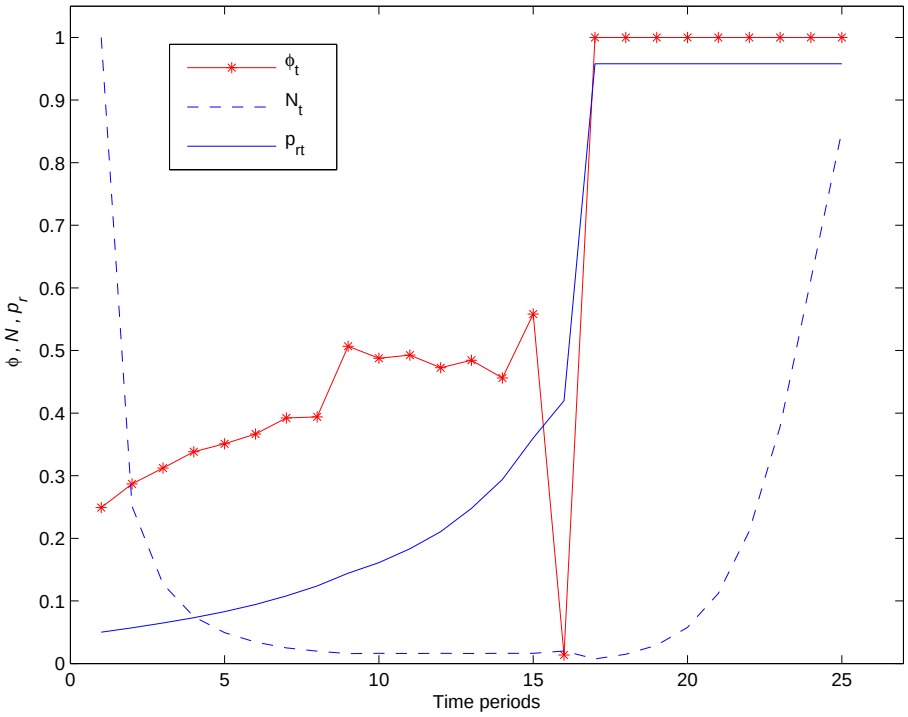

Figure 3: Evolution of (N_t, p_{rt}, ϕ_t) with $c_s > 0$ and $c = 0$

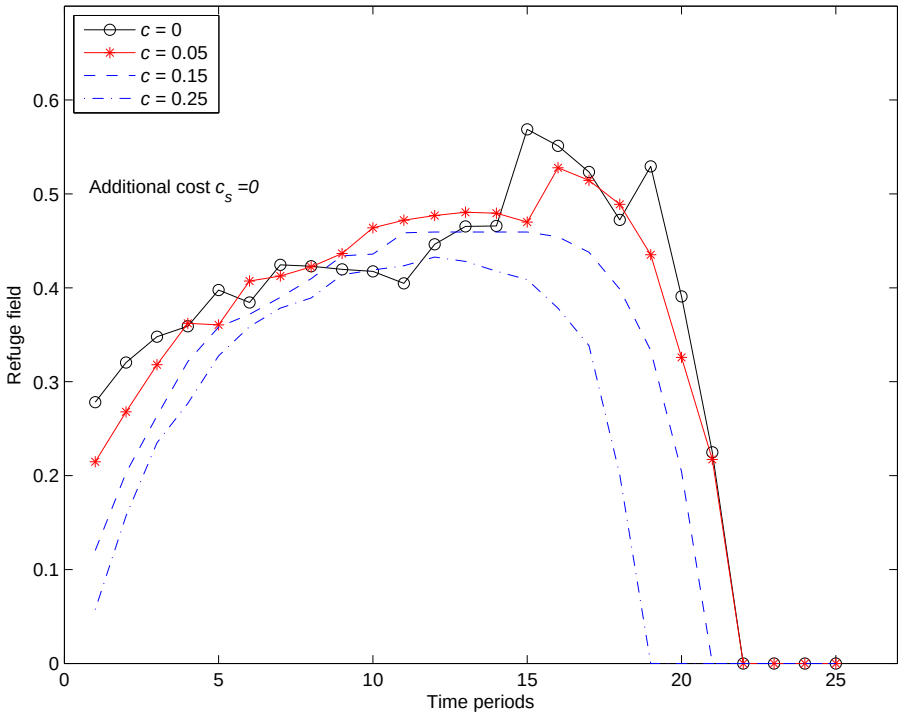

Figure 4: Comparative dynamics of the refuge policy with $c_s = 0$

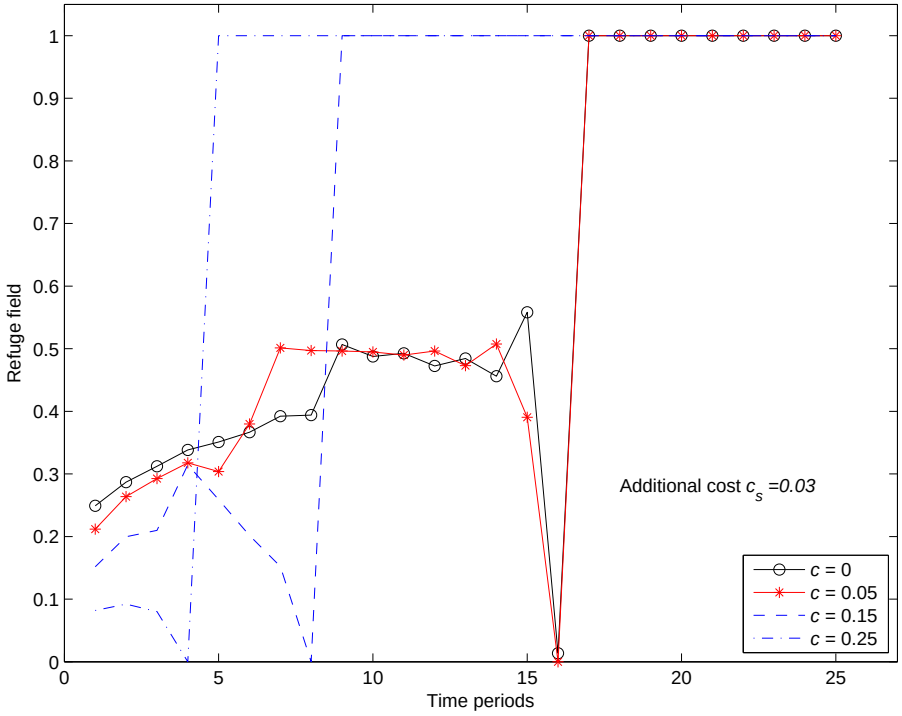

Figure 5: Comparative dynamics of the refuge policy with $c_s > 0$

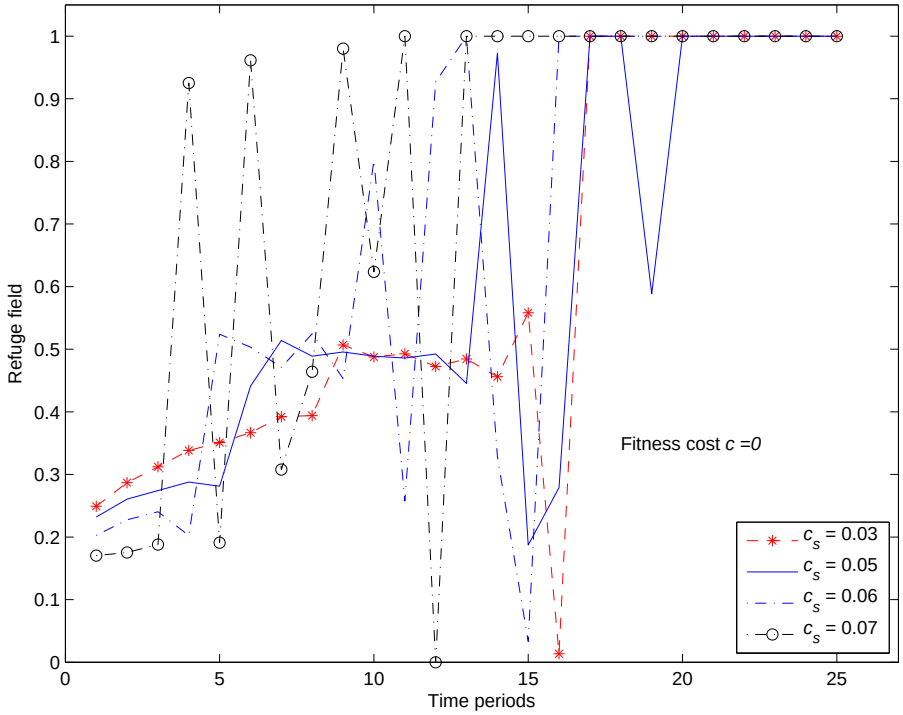

Figure 6: Comparative dynamics of the refuge policy and the cost premium (c_s) when $c = 0$

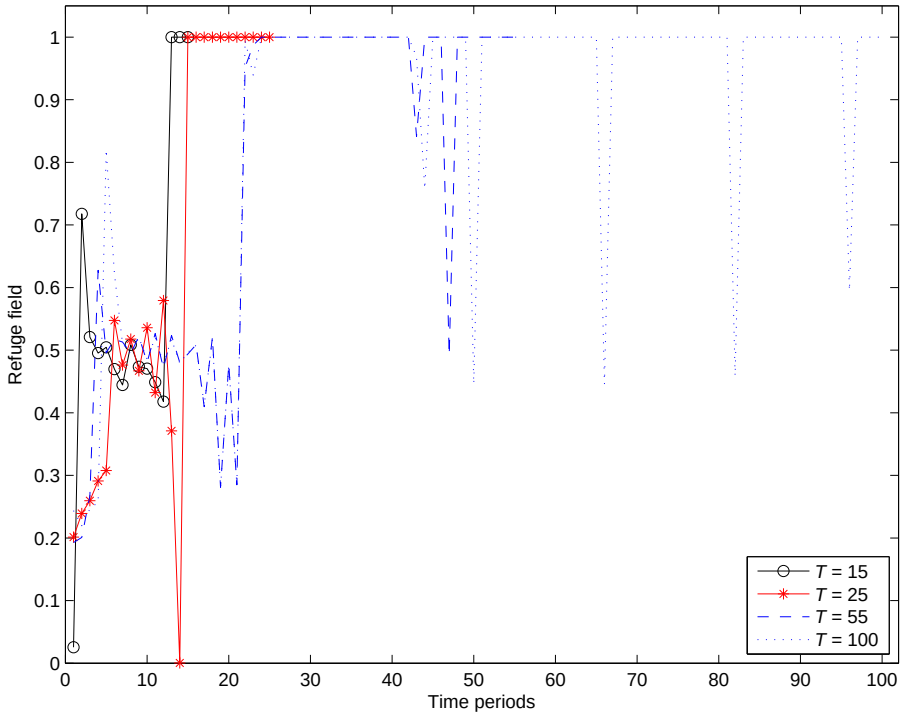

Figure 7: Comparative dynamics of the refuge policy and the time horizon (T) when $c_s = 0.05$

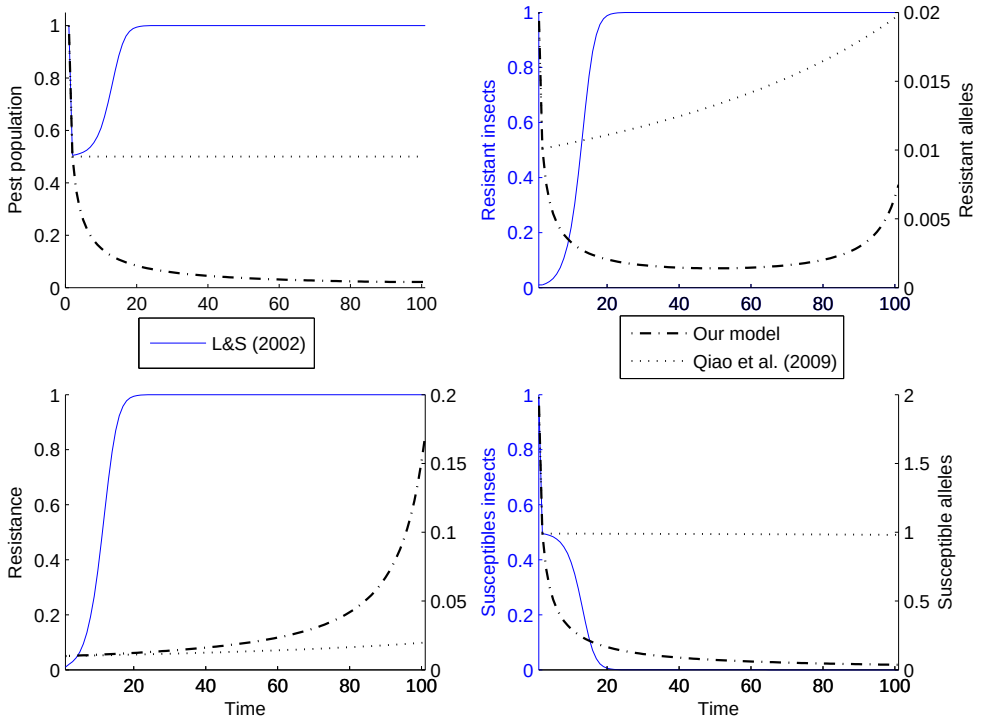

Figure 8: Comparison with L&S (2002) and Qiao *et al.* (2009)

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).

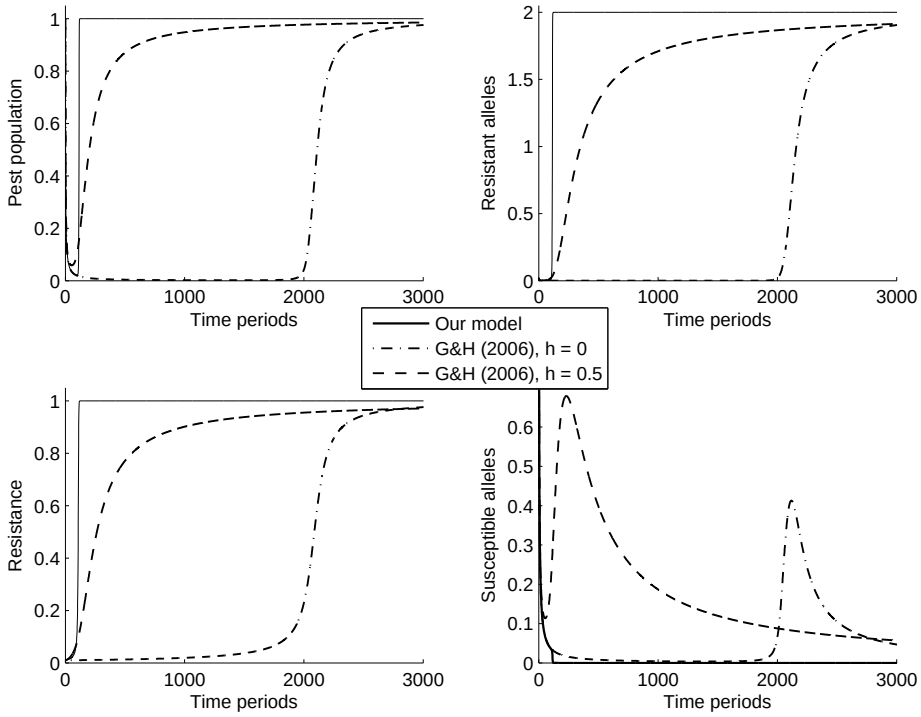

Figure 9: Comparison with Grimsrud and Huffaker (G&H, 2006)