Biological Limits on Agricultural Intensification: An Example from Resistance Management

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Abstract

When the application of pesticides places selective evolutionary pressure on pest populations, it can be useful to plant refuge areas—crop areas intended to encourage the breeding of pests that are susceptible to the pesticide. Renewed interest in refuge areas has arisen with recent advances in biotechnology and genetically modified (GM) crops. In this paper, we use a simple model of the evolution of pest resistance to characterize the socially optimal refuge strategy for managing pest resistance. We demonstrate some interesting analogies with other models of renewable resource management, such as those of fisheries. Among the analogous results are findings that maintaining what we might call "maximal sustainable susceptibility" is typically not economically optimal and that the stock of pesticide effectiveness maintained is a declining function of the discount rate. The former result is in contrast to some existing studies based solely on biological considerations. We also examine the land use consequences of the enhanced agricultural productivity that results from the use of GM crops. Arguments are frequently encountered to the effect that GM crops could reduce the total area required for agriculture and thereby increase the quantity of land conserved for natural habitat. We show that the situation may not be as simple as standard arguments portray it. If refuge areas are used to manage resistance, then more land will be devoted to agriculture than would be the case were it simply a matter of adopting a technology that offered the same yield per hectare without requiring the management of a biological stock such as pest susceptibility.

Key Words: Pest resistance, biotechnology, optimization.

JEL Classification Numbers: Q1, Q2.

Contents

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1. Introduction

Progress in agricultural technology has resulted in spectacular increases in yields over the last century.[1] The "green revolution" transformed agriculture in many countries through the use of inputs such as pesticides, herbicides, fertilizers, and hybrid seeds. Biotechnology now represents the cutting edge of efforts to increase agricultural yields even more.

Agricultural intensification may be the only way of mitigating the threat posed by the increasing food needs of larger and more prosperous human populations to the natural habitats on which much of the world's biological diversity depends. Many authors have argued that growing more food on the same area of land can reduce the pressure on natural habitats (Leisinger, 1999; Pagiola et al., 1998; Southgate, 1997). This argument is not complete, however. Increases in agricultural productivity per unit of land could, by lowering the price of food, increase the quantity demanded and increase the area of land devoted to agriculture.[2]

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[1] Some aggregate statistics are illustrative. In U.S. wheat production, for example, yields per acre averaged 12.3 bushels between 1866 and 1875, 13.6 bushels between 1926 and 1935, and 36.8 bushels between 1988 and 1997 (USDA, 1936; 1998). Whereas these statistics reflect some differences in the location of farming over the years (although there has not been much change in total area harvested in the past 80 years), the difference is largely due to changes in technology. Similarly spectacular increases can be seen in other crops and in other countries employing modern agricultural technologies and practices.

[2] Conventional wisdom has it that the demand for food is inelastic, hence, this scenario is unrealistic. Two counterarguments can be made. First, improvements in agricultural technology that make farming more productive in tropical regions could result in shifts in the location of production from the relatively biologically poor North to the more biodiverse South. Second, whereas the demand for calories and basic nutrients may be inelastic, tastes for the form in which they are packaged depend on prices and wealth. When grain is expensive, people eat it. As it becomes cheaper, grain is fed to cattle, and people eat beef. Cattle are notoriously poor converters of the products of photosynthesis into body mass and require much more land area per calorie delivered than would the grain on which they feed.

Another strain of research concerns the sustainability of agricultural systems. Some researchers are concerned that high yields do not represent so much a triumph of technology as a squandering of natural capital. Barrett (1991) and Krautkraemer (1994) consider the effects of agricultural practices on soil depletion. Pimental et al. (1995) present an alarming report on soil erosion (but see also Trimble and Crosson, 2000). Naylor and Ehrlich (1997) argue that the negative effects of pesticides on beneficial organisms may outweigh their positive effects in controlling their intended targets. Perrings and Walker (1995) and Albers and Goldbach (2000) consider situations in which intensive agriculture can induce irreversible changes in ecosystem characteristics. All of these papers offer examples of what we call "biological limits on agricultural intensification"—constraints on technological possibilities imposed by the physical and biological environment.

In this paper, we consider another example of such a constraint: that imposed by pest resistance to pesticides. The application of any pesticide will exert evolutionary pressure in favor of organisms resistant to its toxin. An effective pesticide is one that kills the great majority of its target pests. In almost any population, however, some organisms will be blessed with a fortuitous combination of genetic attributes that enable them to survive. Subsequent reproduction will then result in a greater frequency of genetically resistant pests and, consequently, reduced effectiveness for the pesticide. If agricultural yields are to be sustained in the face of growing resistance, one of two things must happen: either new and better pesticides must be developed as old ones become ineffective, or strategies must be undertaken to manage resistance.

With advances in biotechnology, pesticides are increasingly bred *into* as opposed to applied *onto* crop plants. Therefore, resistance management is accomplished not by the timing of pesticide application, as was the case in the past, but by putting aside "refuges" to encourage the breeding of pests in a pesticide-free environment. It may seem strange to set aside an area of crops for the express purpose of feeding the insects one is trying to eliminate. However, the argument is that a population of "susceptible" organisms from the refuge areas will interbreed with organisms that are genetically resistant to the pesticide. A crucial consideration is that resistance typically comes with an evolutionary "fitness cost" (Anderson and May, 1991). In the absence of the pesticide, mortality is higher among resistant than among susceptible pest organisms. To give a simple example, an insect with a thicker shell may be less affected by a toxin than one with a thinner shell. The thicker shell could, however, also make its bearer

ungainly and therefore easier prey for predators.[3] Hence, reserve areas reduce the selection pressure on susceptible pests and maintain their predominance.

We will return to a more detailed discussion of resistance and its management in the following section. But first we summarize our approach, our findings, and our interpretation of those findings. The model we work with is stripped down to the basics to highlight results concerning agricultural intensification, resistance management, and land use. A social planner is assumed to care about two things: the production of food and the preservation of natural habitat for maintaining biodiversity. We abstract from most real-world aspects of agricultural production and suppose that land is the *only* costly input employed in growing a single crop. Implicitly, then, we are treating pesticides as free inputs. This may not be as unrealistic as it seems. Because the toxin is expressed in the genetically modified plant, we might suppose that the social planner is deciding how much use to make of a resource that can be acquired at negligible marginal cost: genetically modified seed. We also suppose—again somewhat unrealistically, but in the interest of presenting clear results—that the conversion of land from natural habitat to agricultural use and back is costless.

Two interesting insights emerge from our simple model. The first is simply that we can compare and contrast our results with those arising from models that describe the management of other renewable resources over time, such as models of fisheries or forests. In our model, higher discount rates also result in less environmentally friendly outcomes: more land is devoted to agriculture, at the expense of habitat retained for biodiversity. The reasoning is somewhat different here than in other biological resource models, however. In models of fisheries, for example, higher discount rates imply that the stock of fish—the biological resource of interest in that context—is deemed less valuable, and thus lower stocks will be maintained. Higher discount rates also motivate less conservation in our model, even though habitat supporting biological diversity is an argument of the period-by-period objective function.[4]

The reasoning behind our results is as follows. The choice of how much land to plant at any time is determined by balancing the short-term marginal product of additional acreage against future increased losses to pests as a result of decreased pest susceptibility to pesticides.

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[3] Resistance is also often a recessive genetic trait, making the event that it is expressed in offspring resulting from the random mating of two parent organisms less likely.

[4] In this respect, our model is similar to that of Hartman (1976) on optimal forest rotation when standing forests have value.

At higher discount rates, the losses become less significant and short-term production becomes more desirable. More output requires more land in production, and consequently, less habitat is maintained for biodiversity. A sort of "exhaustion" result also is obtained: when the discount rate is high enough, the optimal policy allows a fully resistant pest population to evolve.

Our second insight may be less intuitive. In the steady state of the model, yield per hectare planted is constant. We might regard the combination of pesticide use and resistance management as an agricultural "technology" that affords a certain yield per hectare. At steady state, *more* land would be devoted to agriculture using this "technology" than under one that afforded the same yield per hectare but required no resistance management. The result is subtle, so we should clarify what we are *not* saying. The model is, by construction, one in which social welfare is maximized. Thus, we are not saying that the externality under consideration generates suboptimal performance. We are also not saying that the improvements afforded by the intelligent combination of biotechnology and resistance management are a bad thing. The point is simply that enthusiasm for these improvements should be tempered. Biotechnological improvements are clearly welfare-enhancing and may well be land-saving. *However, the amount of land saved may be less than one might initially suppose.*

In the next section of the paper, we discuss resistance management strategies in somewhat more detail. We introduce our model in the third section. We derive its steady state and summarize its implications in the fourth section. The fifth section is a brief conclusion.

2. Resistance Management

Earlier studies have considered the economics of resistance management. Hueth and Regev (1974) consider the timing of pesticide applications and its effect on resistance (see also Regev et al., 1983). Laxminarayan (1999), Laxminarayan and Brown (forthcoming), and Göschl and Swanson (2000) consider analogous issues in the management of antibiotic resistance.

Recent developments in biotechnology have spurred a renewed interest in the topic. A gene from the bacterium *Bacillus thuringiensis* (frequently abbreviated as Bt) has been inserted in cotton, tobacco, corn, and soybean varieties. This gene codes for the production of a protein that is highly toxic to many insect pests. In 1990, no genetically modified organisms (GMOs)

were under commercial cultivation in the United States. By 1999, nearly 100 million acres were planted with GMOs.[5]

In the United States, the Environmental Protection Agency (EPA) is responsible for the regulation of pesticides and, hence, of Bt GMOs. EPA has mandated that refuge areas be grown in conjunction with all transgenic crops (EPA, 1998). Farmers have been offered two refuge strategies from which to choose. They may either set aside fields on which they plant non-Bt varieties and use other pesticides on them, or they may set aside a considerably smaller area for non-Bt plantings and use no pesticides.[6]

It is interesting that EPA regulations regarding GMOs are the first from any agency in the United States that treat pest susceptibility as a public good (Livingston et al., 2000), even though resistance issues arose with more traditional pesticides as well.[7] Several economic analyses have now been conducted of EPA's refuge policy (Hurley et al., 1997; Hyde et al., 1999; Livingston et al., 2000). Such analyses—and indeed, EPA's policy itself—point to an interesting issue in our modeling. One might reasonably wonder whether a refuge strategy is ever really necessary. To borrow a term from the literature on nonrenewable resources, resistance management might be obviated, or would at least take a different form, if there were a "backstop technology" waiting to be substituted for pesticides whose targets had developed resistance.

Two arguments defend our assumption that the model reaches a steady state and our analysis of conditions in that steady state. The first argument is biological: different poisons have unique or specific mechanisms of imparting toxicity. However, the manner in which resistant pests survive the effects of different toxins can be fairly similar. Therefore, a pest population that

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[5] Total cropland area planted in the United States is some 334 million acres (USDA, 1998).

[6] Under the first option, a reserve 20% of the size of the area of crops planted in the Bt variety is typically required. When no pesticides are used, the reserve requirement is 4%.

[7] We might speculate as to why GMOs triggered policy action when traditional pesticides did not. In both cases, an externality is present: any farmer applying a pesticide of any form is helping to create a resistant pest population that can affect other farmers. GMOs are perhaps somewhat more conducive to the development of resistance, however. Traditional pesticides could be blown, washed, or carried away (by organisms unfortunate enough to have come in contact with it). Susceptible organisms might then reestablish themselves between pesticide applications. Bt toxin, on the other hand, is always present in the plant and thus provides no respite for susceptible pests.

We suspect that the reasons EPA has chosen to institute refuge requirements now has more to do with politics and public perception, however. GMOs have given rise to a number of public concerns regarding human health and safety, ecological effects, and the potential spread of engineered genes to wild organisms. Refuge requirements might have been instituted in part to create the impression that the agency was "doing something" about these other concerns.

has become resistant to one compound may also have acquired resistance to others. It may then prove impossible to generate an indefinite series of effective pesticides. The constraints imposed by environmental, human health, production cost, and research and development issues make the generation of such an indefinite series of compounds even less likely.

The second argument is simply that EPA's policy is formulated *as if* the system were in a steady state without a technology backstop.[8] If an existing regulatory agency is behaving this way, then it seems reasonable to ask how a hypothetical social planner would set policy. We should point out, however, that we are not invested in this position. The results we develop would hold in the absence of sufficiently rapid technological progress. In fact, it remains an open question as to whether that progress will occur in the long run.[9]

3. The Model

We develop a simple model of the evolution of pest resistance. The setting is a large area in which a single crop can be planted. The pest population is assumed to be local; both in-migration and out-migration are ruled out. Other conditions implicit in deriving the Hardy–Weinberg principle, such as random mating between resistant and susceptible pests, negligible mutation, non-overlapping pest generations, and sexual reproduction of pests, are all assumed to hold.[10]

The pest population is denoted by D. The proportion of susceptible pests in the population is denoted by a fraction w. Susceptible pests are those that have not developed genetic resistance to the toxin. Put in another way, w may be thought of as the stock of effectiveness of the pesticide; it is the proportion of the pest population to which the toxin is lethal.

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[8] We might speculate as to whether this is due in part to the influence of natural scientists in the policy formation process. Biologists' writings on the subject (e.g., Tabashnik, 1997) make it appear that some see maintaining negligible resistance levels as analogous to maximum sustainable yield in fisheries, that is, a seemingly "natural" objective that does not necessarily withstand economic scrutiny.

[9] On a technical note, the effects of anticipated technological progress are analogous to those of a higher discount rate: future resistance is less important if improved pesticides obviate concern with it.

[10] A *genotype* is a particular genetic configuration. An *allele* is any one of the two or more forms that may compose a gene; for example, alleles for blue or brown eyes are common in many human populations. The Hardy–Weinberg principle of quantitative genetics holds that, for a population satisfying the assumptions we have stated and in which expected mortality is the same across different genotypes, the expected proportion of alleles and of genotypes remains constant from generation to generation.

The pest population is assumed to grow logistically with an intrinsic growth rate of g and a carrying capacity of K per unit of land planted in the crop. Total land is assumed to be fixed and is normalized to 1. The fraction of total available land area devoted to agriculture is denoted by Q. The total number of new pest organisms hatched (presuming they are the offspring of egg-bearing insects) in every period, then, is $gD(1 - D/KQ)$. From this gross increase, we must subtract mortality among both susceptible and nonsusceptible pests.

A refuge strategy calls for planting a fraction q of the total land devoted to agriculture Q in a crop to which pesticide is applied (or in this case, with GMOs implanted). Hence, a fraction $1 - q$ of agricultural land is not treated with pesticide. Recall that a fraction w of all pests is susceptible. We suppose that a fraction h of those susceptible pests that light in crops treated with pesticide die after exposure to the toxin.[11] A fraction r of nonsusceptible pests die, regardless of whether they are exposed to the pesticide. It is easily demonstrated that h and r can be regarded as the difference among mortality rates relative to the mortality of susceptible pests that are not exposed to the pesticide. This "baseline" mortality can be subsumed in the constants g and K of the population growth equation. With regard to this last observation, it is important to note that both h and r are positive. Susceptible pests are, of course, more likely to die in the presence of the toxin. Resistant pests are also more likely to die than are susceptible ones when the toxin is not present. This is the biological notion of fitness cost, to which we referred above. As we show momentarily, the relationship of h to r is important; we generally assume that the former is greater than the latter.[12]

We assume that pests distribute themselves evenly over the area planted with crops. A fraction q of the pest population D will light in the area treated with pesticide. Of these, a fraction h of the fraction w that are susceptible will die. A fraction r of the fraction $1 - w$ of resistant pests will also die.

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[11] This parameter could be conveniently normalized to 1 in the subsequent analysis, but we consider it explicitly to show that our model is somewhat more general than it might appear if we simply assumed that all susceptible organisms died on exposure to the pesticide. One might explain mortality being less than 1 by supposing that the toxin weakens pests rather than killing them outright, thus making them easier prey for predators, or by motivating our model as a heuristic depiction of a more complicated reality in which susceptible pests may be exposed to the pesticide in different concentrations.

[12] If this relationship did not hold, all resistant alleles would eventually be eradicated. The continued survival of "unfit" genes is problematic but might be explained by the infrequent occurrence of random events that temporarily favor otherwise "unfit" organisms.

Combining our assumptions, the growth of the pest population can be modeled as

$$(1) \qquad \dot{D} \;=\; gD\!\left(1-\frac{D}{KQ}\right) - wqhD - (1-w)rD.$$

It can be shown that the proportion of resistant pests in the population also follows a logistic equation, with the growth parameter equal to the difference in relative mortality rates between genotypes (e.g., Bonhoeffer et al., 1997). Thus

$$(2) \qquad \dot{w} \;=\; (qh-r)w(w-1).$$

Because $w < 1$, the larger the fraction of crop land treated with pesticide, the greater the decline in the effectiveness of the pesticide.

4. Optimal Refuge Areas and Steady State Results

We now characterize the optimal refuge strategy. The proportion of agricultural land set aside as refuge area in each period determines the net crop yield in that period as well as the effectiveness of the pesticide in succeeding periods. There is, then, an intertemporal trade-off between increasing refuge size (and losing less agricultural yield to pests today) and more rapidly eroding pest susceptibility to the toxin.

Suppose that each surviving pest eats an amount α. Normalize gross output per unit area planted to 1. If a fraction q of the area Q devoted to agriculture is treated with pesticide, then gross production in this area will be qQ. A fraction q of the pest population D will light in the area treated with pesticide. Of these, the fraction $1 - w$ that are resistant will survive, as will a fraction $1 - h$ of the susceptible fraction w. Each of these surviving organisms will consume α units. Thus, the net yield from the area treated with pesticide is given by $qQ - [(1-w)q+wq(1-h)]D\alpha = qQ - (1 - wh)qD\alpha$. Similarly, the gross yield from the area without pesticide is $(1 - q)Q$. A fraction $1 - q$ of pests will light in the untreated area, where they will consume $(1 - q)D\alpha$ units of the crop.[13] Net yield in the untreated area is, then, $(1-q)Q - (1-q)D\alpha$. Net yield from agriculture, Y, is given by the sum of net yields from the area treated with pesticide and the refuge area that is not treated:

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[13] Implicit in our assumptions are the notions that susceptible pests die so quickly on exposure to the pesticide as to consume only a negligible amount of the crop before expiring and that mortality to other causes occurs after consumption. Both assumptions could be relaxed, but neither is important for establishing our general results.

(3) $Y(q, Q, D, w) = qQ - (1 - wh)qD\alpha + (1 - q)Q - (1 - q)D\alpha = Q - (1 - whq)D\alpha$

For the purposes of our very simple example, we suppose that social welfare depends on two goods: the net yield from agriculture [Y from equation (3)] and the total quantity of land conserved as natural habitat, $1 - Q$. To facilitate the derivation of tractable results, we suppose that preferences are of the Cobb–Douglas form $U(Y, 1 - Q) = Y^{\beta}(1 - Q)^{1-\beta}$.[14] Let the social discount rate be ρ. Then, a social planner would choose q and Q so as to maximize

(4) $\int_0^{\infty} (Q - [1 - wqh]D\alpha)^{\beta} (1 - Q)^{1-\beta} e^{-\rho t} dt$

subject to equations (1) and (2) for the evolution of pest populations and resistance.

The current-value Hamiltonian for our problem is

(5) $H = (Q - [1 - wqh]D\alpha)^{\beta} (1 - Q)^{1-\beta} + \lambda_1 D\left[g\left(1 - \dfrac{D}{KQ}\right) - qhw - r(1 - w)\right]$

$+ \lambda_2 (qh - r)w(w - 1)$

where λ_1 and λ_2 are the co-state variables associated with the stock of pests D and the proportion of susceptible pests w, respectively. Necessary conditions for an optimum are given by equations (5.1) through (5.4):

(5.1) $\beta \dfrac{U}{Y} whD\alpha - \lambda_1 whD - \lambda_2 wh(1 - w) \begin{pmatrix} < \\ = \\ > \end{pmatrix} 0 \ as \ q \begin{pmatrix} = 0 \\ \in (0,1) \\ = 1 \end{pmatrix};$

(5.2) $\beta \dfrac{U}{Y} - (1 - \beta)\dfrac{U}{1 - Q} + \lambda_1 g \dfrac{D^2}{KQ^2} = 0;$

(5.3) $\rho\lambda_1 - \dot{\lambda}_1 = \beta \dfrac{U}{Y}(wqh - 1)\alpha + \lambda_1\left[g\left(1 - \dfrac{2D}{KQ}\right) - qhw - r(1 - w)\right]$

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[14] Our basic results also obtain under a CES utility function of the form $U = \left(\beta Y^{\eta} + (1 - \beta)(1 - Q)^{\eta}\right)^{1/\eta}$; see footnote 17.

$$(5.4) \quad \rho\lambda_2 - \dot{\lambda}_2 = \beta\frac{U}{Y}qhD\alpha + \lambda_1(r - qh)D + \lambda_2(r - qh)(1 - 2w).$$

Let us consider results in a steady state. It is obvious from equation (2) that, if (5.1) holds in the steady state (i.e., if $0 < q < 1$), then $q = r/h$. If $r = 0$, then there would be no fitness cost of resistance, and refuge areas would serve no purpose. If $r > h$ and if the discount rate were not too high, then the optimal strategy would involve planting refuges until all organisms that contained resistant alleles were eradicated.

If $D \neq 0$, then from equations (1), (2), (5.3), and (5.4), we have

$$(6) \quad D = \frac{g - r}{g}KQ,$$

$$(7.1) \quad \lambda_1 = \beta\frac{U}{Y}\frac{(wr - 1)\alpha}{\rho + g - r},$$

and

$$(7.2) \quad \lambda_2 = \beta\frac{U}{Y}\frac{rD\alpha}{\rho}.$$

Note that equation (6) requires that $g > r$ if the pest population is to be positive in steady state. If this were not the case, then it would be optimal to eradicate the pest population.

From equation (7.1), we note that $wr - 1$ is non-positive because both w and r are fractions. It follows that $\lambda_1 < 0$; λ_1 represents the shadow price of a "bad," the pest population. Similarly, from equation (7.2), $\lambda_2 > 0$, because it is the shadow price of a "good" stock, susceptibility to the pesticide. Substituting for λ_1 and λ_2 from equations (7.1) and (7.2) and for D from equation (6), the steady-state level of w (w^{SS}) can be derived from equation (5.1):

$$(8) \quad w^{SS} = \frac{(r - \rho)(\rho + g - r) - \rho}{r(g - r)}.$$

Our results echo findings from other renewable resource contexts. Current thinking in GMO crop management seems to be that refuge areas should be established to manage resistance. Equation (8) demonstrates that such a strategy is not always optimal, however. If the discount rate, ρ, is large enough—a sufficient condition is that it be greater than the fitness cost of resistance, r—it is optimal to exhaust the stock of effectiveness at steady state. This

conclusion is analogous to a finding from the fisheries literature: it may be optimal[15] to harvest a species to extinction if its growth rate is less than the discount rate (e.g., Clark, 1973).

Next, consider the steady-state level of agricultural land use. Using the expressions derived thus far, we can solve for Q in the steady state (Q^{SS}):

$$(9) \qquad Q^{SS} = \frac{\beta}{1 - \dfrac{(1-\beta)\rho(1+\rho-r)\alpha K}{g - (1+\rho-r)(g-r)\alpha K}} \quad .$$

Consider the fraction in the denominator of equation (9):

$$\frac{(1-\beta)\rho(1+\rho-r)\alpha K}{g - (1+\rho-r)(g-r)\alpha K} \quad .$$

The numerator of this expression is positive, because $r < 1$ and $\rho > 0$. To evaluate the denominator, equation (5.2) can be rearranged using equations (6), (7.1), and (8) to show that in the steady state,

$$(10) \qquad g - (1+\rho-r)(g-r)\alpha K = g\frac{Y}{1-Q}\frac{1-\beta}{\beta} > 0,$$

The inequality results because Cobb–Douglas preferences imply that both Y and $1 - Q$ are essential to welfare and, hence, are strictly positive. Because the denominator of equation (9) must be positive for Q^{SS} to be positive, we have established that $Q^{SS} > \beta$.[16]

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[15] This condition holds when harvesting costs are independent of population, and of course, we should note that it is "optimal" in the narrow sense of profit maximizing in the absence of ethical concerns, ecological externalities, etc.

[16] We have assumed Cobb–Douglas preferences because they make the modeling somewhat more compact and present an especially clear illustration of the phenomenon we wish to highlight. In a static model with Cobb–Douglas preferences, the amount of land devoted to agriculture is independent of the productivity of land in agriculture. Thus, because we find that $Q^{SS} > \beta$, the requirements of resistance management clearly drive the result.

We have also derived results for the case of CES preferences (see footnote 15). In this case we find

$$Q^{SS} = \frac{1}{1 + \left(\dfrac{\beta}{1-\beta}\right)^{\frac{1}{\eta-1}}\left(\kappa^{SS}\right)^{\frac{1}{\eta-1}}\left(\kappa^{SS} - \dfrac{\rho(1+\rho-r)\alpha K}{g}\right)},$$

where

The finding that land devoted to agriculture in the steady state is greater than the parameter β has an interesting interpretation. Crop losses in a steady state would be $\alpha wKQr(g - r)/g$, so net yields in steady state would be $\left[1 - GwKr\dfrac{g-r}{r}\right]Q$. As equation (9) makes clear, however, a pesticide for which resistance is optimally managed *cannot* be regarded as if it simply enables an agricultural technology for which yield is a constant $1 - \alpha wKr\dfrac{(g-r)}{g}$ units of output per hectare planted. If steady state yield per hectare is regarded as a constant technological parameter, then it is easily demonstrated that with Cobb–Douglas preferences the amount of land devoted to agriculture is independent of the productivity of land in agriculture: $Q = \beta$ [17]. However, when yield per hectare is dependent on a biological stock that is depleted with use, $Q^{SS} > \beta$.

Differentiating equation (9) with respect to the social discount rate, ρ, it can be shown that the amount of land devoted to agriculture in the steady state is an increasing function of the discount rate.[18] It is also interesting to consider the prescription of the model in the limit as the

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$$\kappa^{SS} = \frac{g - (1 + \rho - r)(g - r)\alpha K}{g}$$

is the yield per hectare planted (averaged over areas treated with pesticide and those not treated). It can be shown that maximization of the CES objective in footnote 16 with a productivity of κ^{SS} per hectare would result in a choice of land allocation

$$Q = \frac{1}{1 + \left(\dfrac{\beta}{1-\beta}\right)^{\frac{1}{\eta-1}}\left(\kappa^{SS}\right)^{\frac{\eta}{\eta-1}}}.$$

Note that this expression reduces to $Q = \beta$ in the Cobb–Douglas case, $\eta = 0$. The demonstration is somewhat laborious, but it can be shown that $Q^{SS} > Q$ in the more general CES case as well.

[17] If we consider a pest management technology that kills a constant fraction of all pests is available, then we can easily show that $Q^{SS} < \beta$. The underlying economic intuition is that every additional unit of land devoted to agriculture increases current yield but increases future pest population. The intertemporal externality imposed by future pests forces the optimizing planner to devote less land to agriculture than if no such externality were present.

[18] The details of this calculation are more tedious than enlightening. Land use in the steady state is increasing in ρ if the denominator of equation (9) is increasing in ρ. Differentiating that denominator using the quotient rule, we find that the sign of the derivative is the sign of

$$[g - (1 + \rho - r)(g - r)\alpha K](1 + 2\rho - r) + \rho(1 + \rho - r)(g - r)\alpha K.$$

discount rate approaches 0. Inspection of equations (8) and (9) reveal that w^{ss} and Q^{ss} would approach 1 and β, respectively. So long as there remain any resistant alleles in the population, welfare in the indefinitely long run would be increased by eradicating those alleles.[19]

We close this section by reviewing the economic intuition that we offered in the introduction. In the short run, the social planner values both agricultural output and natural habitat and resolves the trade-off between these two goods by weighing a third consideration: the long-term effects of pests on agricultural output. This third factor declines in importance as the discount rate increases. Hence, the social planner sacrifices more habitat in favor of agricultural production at higher discount rates.

In the steady state, the social planner treats some of the planted land with pesticide and maintains some as a refuge. Pests consume a constant proportion of output. A planner who treats all generations equally—who does not discount at all—would behave as if net production were a constant fraction of total area planted. A planner who discounts, however, will recognize that some short-term increase in production should be traded against long-term reductions in susceptibility in steady state. The principle is analogous to the reasons for which maintaining maximum sustainable yield is not optimal in fisheries management with a positive discount rate.[20]

5. Conclusion

We employed a simple model to demonstrate some implications of pesticide use and resistance for land use. It is probably unnecessary to caution readers that the model is intended

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We have signed the expression in square brackets in equation (10). As noted above, $g > r$. Since ρ and r are necessarily positive and <1, this expression is positive.

[19] This extreme result is an artifact of a specification of the evolution of resistance [equation (2)] in which resistant alleles can be reduced to arbitrarily small proportions without ever being entirely eliminated. The fundamentally discrete character of alleles and the possibilities of mutation preclude interpreting the model literally. It is, nonetheless, a useful way of thinking about these issues.

[20] The argument implied is that a social planner *should* discount in making choices. As the discount rate goes to 0, the "optimal" policy calls for not using pesticides at all. Resistance would then be driven to an infinitesimal level so that when pesticides finally were used, they would be most effective. Would it be fair, however, to demand that all foreseeable generations make such sacrifices? This interpretation is analogous to Tjalling Koopmans's argument for a positive discount rate in macroeconomic growth models (e.g., Arrow, 1999). So long as additional capital is productive, all generations would be called on to make sacrifices in the interest of providing an infinitely distant posterity with a higher level of steady-state consumption.

be illustrative rather than realistic. Despite the fact that we have chosen assumptions to facilitate analytical solutions, we believe that our conclusions generalize.[21]

We return to our main result. Adopting biotechnology increases the yield per hectare, as does a neutral technology that involves no biological stock. However, agricultural intensification through these two modes has sharply different consequences for the quantity of land devoted to habitat. Under the former case, the social planner trades off current yield for future pest susceptibility and devotes more land to agriculture to ensure that sufficiently large refuge areas are available to manage pest susceptibility in a sustainable manner. In the latter case, there is no biological stock that needs management; hence more land is devoted to habitat.

The conclusion that we emphasize is that biological constraints limit the intensification of agriculture in ways that may not be immediately apparent. Whereas other examples of this phenomenon have been developed, we find the case of resistance to biotechnology particularly interesting in that the interactions are complicated and the implications subtle. Our results do not hinge on externalities, nor do they suggest that technological advances are not desirable. They only point to the wisdom of measured expectations concerning the potential of agricultural intensification to solve the problem of habitat and biodiversity loss.

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[21] Two items in particular might be worth mentioning in this respect. First, entomologists and agricultural experts note that sudden and rapid expansions in pest populations may be a greater concern than the gradual increases postulated by the logistic specification of population growth that we have employed. A more realistic setup might then be achieved by incorporating stochastic growth rates in that specification, but it would not necessarily change our qualitative results. Second, our assumption that the amount of consumed per pest is independent of the number of pests is also not strictly true. It seems a reasonable approximation for pest populations contained at manageable levels, however.

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