Predator-Prey Systems in Pest Management

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The use of chemical pesticides frequently causes minor pests to become serious problems by disturbing the natural controls that keep them in check. As a result, it is possible to suffer heavier crop losses after pesticides are introduced than before their introduction. Efficient use of pesticides requires complete biological modeling that takes the appropriate predator-prey relationships into account. A bioeconomic model is introduced involving three key species: a primary target pest, a secondary pest, and a natural enemy of the secondary pest. Optimal decision rules are derived and contrasted with myopic decision making, which treats the predator-prey system as an externality. The issue of resistance in the secondary pest is examined briefly.

Chemical pest-control programs directed at a target pest species often create new economic pests out of other pest species that had previously been of minor or sporadic importance. These species, known as secondary pests, are those which in "normal" growing seasons do not inflict major crop damage, although significant outbreaks may sometimes be stimulated by natural causes such as unusual weather. Secondary-pest outbreaks are known as "induced" when they are brought about by human activities that disrupt the agricultural ecosystem.

Induced secondary-pest problems often occur because most available pesticides have broad-spectrum toxicity, rather than being narrowly targeted to a particular species, so that various species, including natural predators, are destroyed along with target pests. When their natural predators are reduced in numbers, secondary pests may proliferate to a point where they pose a serious economic threat to agricultural producers. Not infrequently, secondary-pest damage may come to exceed damage from the original target pest. Moreover, such problems may emerge gradually over time because of "the remarkable ability of pests, but the infrequent ability of natural enemies of pests, to develop resistance to pesticides" (Prokopy, p. 2).

A striking example comes from the Rio Grande Valley of northeastern Mexico where DDT was highly successful in controlling the boll weevil, the region's primary cotton pest, throughout the 1940s. By the 1950s, boll weevil numbers were still basically under control, but the population of a formerly minor pest, the cotton bollworm, was exploding and could not be controlled even with eighteen pesticide applications per year. As a result, cotton production in the region declined from 700,000 acres in 1960 to fewer than 1,200 in 1970, marking the end of a multimillion dollar industry (Prokopy).

The problem of induced secondary pests has been almost entirely ignored in the economics literature, even though it is acknowledged to be of worldwide economic importance (Getz and Gutierrez). Harper and Zilberman have approached the problem using static optimization techniques exclusively. Related work by Feder and Regev described how the balance of a single pest system, consisting of a primary target pest and its natural enemy, may be disrupted when broad-spectrum chemical pesticides are introduced.

Optimal management of predator-prey systems has been described in detail for cases in which one or both species have positive economic value. Relatively recent explorations include those by Ragozin and Brown, and by Mesterton-Gibbons. Predator-prey relationships are equally important in the world of pest control, but there they play an entirely different economic role. Typically, in this setting, the "prey" is a pest (i.e., a damage-inflicting agent that causes economic losses), while the "predator" functions as a natural form of damage control.

This paper introduces the optimal management of a simple multiple-pest system in a dynamic framework, emphasizing the key role that predator-prey relationships need to play in multispecies modeling. The structure of the paper is as follows.

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The biology of a primary-secondary pest system, incorporating three key species, is first introduced. An optimal-control framework is then applied to determine the optimal intensity of pesticide use under myopic single-pest management and under comprehensive multiple-pest management, respectively. Finally, the issue of resistance in the secondary pest is discussed briefly.

It will be shown that, as in Feder and Regev, once the original predator-prey equilibrium is disturbed by chemical intervention, an economically and environmentally inferior equilibrium is likely to be approached over time in which profit is reduced in spite of increasing dependence on toxic chemicals. Moreover, a lengthy and costly adjustment process with high interim levels of crop damage must often be endured if an attempt is made to reduce chemical use and return to the original equilibrium. In the absence of complete biological modeling, even the existence of the potential secondary-pest problem and the economic importance of the predator may go unrecognized until extensive damage has already been incurred.

The Biological Model

This paper models a situation in which pesticide applied to control a target pest in a particular crop incidentally reduces populations of coexisting species as well, in particular a secondary pest and its natural predator. The natural predator tends to keep the secondary pest in check and is therefore known as a "beneficial" species. The primary pest is assumed to have no important natural predators. The biology of this system is the following:

$$\text{Primary Pest: } \dot{X} = \frac{dX_t}{dt} = F(X_t),$$

$$\text{Secondary Pest: } \dot{Y} = \frac{dY_t}{dt} = G(Y_t, Z_t),$$

$$\text{Predator: } \dot{Z} = \frac{dZ_t}{dt} = H(Y_t, Z_t),$$

where X_t is the primary-pest population, Y_t the secondary-pest population, and Z_t the natural-predator population. Net growth in the primary-pest population, $\dot{X}$, changes at a rate that is a function of the current population. Typically as X_t increases beyond a certain level, $\dot{X}$ decreases and eventually falls toward zero as the environmental carrying capacity of the species is approached.

Similarly, net growth in the secondary-pest and predator populations, $\dot{Y}$ and $\dot{Z}$, depends on their current populations, Y_t and Z_t, respectively. In addition, however, net growth in the prey and predator populations is influenced by their interaction. Secondary-pest population growth falls as predators become more numerous ($G_z < 0$), while predator population growth increases as the number of secondary pests that constitute its food supply increases ($H_Y > 0$). In the absence of human intervention, the secondary-pest and natural-predator populations develop according to their mutual biology.

When chemical pesticide is applied, it is assumed that all three species are affected, but to varying degrees. Letting A_t represent pesticide use at time t, human intervention transforms the biological system as follows:

$$\dot{X} = \frac{dX_t}{dt} = F(X_t) - K(A_t, X_t),$$

$$\dot{Y} = \frac{dY_t}{dt} = G(Y_t, Z_t) - L(A_t, Y_t),$$

$$\dot{Z} = \frac{dZ_t}{dt} = H(Y_t, Z_t) - M(A_t, Z_t),$$

where K, L, and M are the respective pesticide kill functions. It is assumed that more pesticide kills more pests, and that as a given population increases, the number of individuals killed by a given dose of pesticide also increases: K_A, K_X, L_A, L_Y, M_A, $M_Z > 0$.

Both the primary and secondary pests reduce crop yield below its maximum potential level. Therefore, the revenue function, $R_t = R(X_t, Y_t)$, which represents the increment to crop value at time t, is a decreasing function of both primary- and secondary-pest numbers: R_X, $R_Y < 0$. The relevant cost function is the cost of pest control, $C_t = C(A_t)$, which depends on the amount of pesticide applied, with $C_A > 0$.

Single-Pest Management

Potential secondary-pest problems may be overlooked because of incomplete biological modeling, or they may be beyond the reach of the individual decision maker because of common property effects such as pest mobility. In either case, the pest-control problem facing the individual producer is simply to choose a time path for applying pesticide to control the primary-pest population. The single-pest management problem is to maximize discounted net revenues from the crop subject to the natural biology of the primary pest:

$$\max \int_0^T e^{-\delta t} [R(X_t) - C(A_t)]\, dt$$

$$\text{subject to } \dot{X} = F(X_t) - K(A_t, X_t),$$

where δ is the rate of discount.

The current-value Hamiltonian function is

$$\tilde{H} = R(X_t) - C(A_t) + \mu_t [F(X_t) - K(A_t, X_t)],$$

with a solution described by the following necessary conditions:

$$(1) \qquad 0 = \frac{\partial \tilde{H}}{\partial A_t} = - C_A - \mu_t K_A$$

(or more generally, choose A_t to maximize $\tilde{H}$);

$$(2) \quad \dot{\mu} = \delta \mu_t - \frac{\partial \tilde{H}}{\partial X_t}$$
$$= \delta \mu_t - R_X - \mu_t [F_X - K_X(A_t, X_t)];$$

and

$$(3) \qquad \dot{X} = \frac{\partial \tilde{H}}{\partial \mu_t} = F(X_t) - K(A_t, X_t).$$

Expressions (1) through (3) can be solved for the time path of optimal pesticide use (see appendix), but the decision rule for the general case is too complex to be very informative. For practical application, it is necessary to specify a biological model. The model that will be considered here is analogous to the well-known Schaefer fisheries model. The biological growth function is logistic

$$F(X_t) = q X_t \left(1 - \frac{X_t}{U}\right),$$

and the effectiveness of human intervention (the pesticide kill function) is proportional to both human effort (pesticide-application rate) and the current population level,

$$K(A_t, X_t) = k A_t X_t,$$

where q, U, and k are constants. Assuming that the marginal cost of applying pesticide is constant, $C(A_t) = c A_t$, we have

$$\tilde{H} = R(X_t) - cA_t + \mu_t [qX_t(1 - \frac{X_t}{U}) - k A_t X_t].$$

The difference between this model and the Schaefer model is that here revenue does not depend positively on a resource *flow*, such as fish harvest, but instead depends negatively on the *stock* of pests.

Then equation (1) becomes

$$c = - \mu_t k X_t.$$

The result is a singular solution that implies the following decision rule: If the marginal benefit from killing a pest exceeds the cost of pesticide ($- \mu_t k X_t > c$), apply at the maximum permissible rate; if the reverse, apply no pesticide. If the equality just holds, maintain the economic-threshold pest population, $\hat{X}_t$, defined by

$$\hat{X}_t = - \frac{c}{\mu_t} k.$$

In particular it can be shown that the economic-threshold pest population for this model is given by (see appendix)

$$(4) \qquad \delta = - \frac{R_X(\hat{X}_t)\, k\, \hat{X}_t}{c} - \frac{q\hat{X}_t}{U}.$$

To solve explicitly for $\hat{X}$, the only remaining information requirement is specification of the marginal revenue function, R_X. No effort has been made here to give a general characterization of $R(X)$ because the relationship between crop value and pest numbers varies intrinsically from crop to crop and pest to pest.

Equation (4) indicates that the opportunity cost of capital, δ, should equal the marginal net rate of return from pesticide use. This rate of return has two components: (1) the marginal increase in revenue from reduced pest damage per dollar spent on pesticide and (2) the decrease in future pest pressure due to a lower pest growth rate. The second effect will not be taken into account in static pest management models.

Since $R()$ is a single-valued function of X_t, expression (5) has a unique solution, implying that $\dot{X} = 0$ and $\hat{X}_t = \hat{X}$ along the singular solution path. Rearranging (5), we have

$$(5) \qquad \hat{X} = \frac{- \delta c U}{R_X k U + qc}, \text{ or}$$

$$(6) \qquad c = - \frac{R_X\, k\, \hat{X}}{\delta + q\hat{X}/U}.$$

The optimal decision rule is therefore to apply pesticide at the maximum or minimum rate possible until the pest population reaches the economic threshold $\hat{X}$. Thereafter, optimal pesticide use, $\hat{A}$, is selected to maintain the pest population at $\hat{X}$. Since $q\hat{X}(1 - \hat{X}/U) = k\hat{A}\hat{X}$, this means

$$(7) \qquad \hat{A} = \frac{q(1 - \hat{X}/U)}{k}.$$

The solution is to apply a bang-bang control until the economic threshold is reached, followed by a steady-state solution with constant pesticide use at level $\hat{A}$. Given a specific revenue function, $R(X)$, the threshold pest population, $\hat{X}$, and the optimal level of pesticide use, $\hat{A}$, can be derived explicitly.

The Secondary-Pest Externality

When an amount of pesticide, A_t, is applied to combat the primary pest, unintended reductions of

the secondary-pest and predator populations also occur, given by the kill functions L and M. The destruction of secondary pests is a positive externality, but because the natural predator is useful in controlling the secondary pest, its destruction is a negative externality of pesticide use. The net consequence depends on the biological relationships that relate the predator, the pest, and the chemical pesticide.

The natural extension of the logistic growth model to a predator-prey system is the Larkin model. This is perhaps the simplest dynamic model which satisfies basic conditions for structural stability.[1] For both the prey (secondary-pest) population, Y_t, and predator (natural-enemy) population, Z_t, the Larkin model combines the basic logistic growth function with a predator-prey response. The natural biology is described by

$$\dot{Y} = G(Y_t, Z_t) = rY_t(1 - \frac{Y_t}{V}) - \alpha Y_t Z_t$$

$$\dot{Z} = H(Y_t, Z_t) = sZ_t(1 - \frac{Z_t}{W}) + \beta Y_t Z_t,$$

where r, V, α, s, W, and β are nonnegative parameters.

In the absence of human intervention, this system is characterized by two linear isoclines, one upward sloping and one downward sloping (Figure 1):

$$\dot{Y} = Y_t [r (1 - \frac{Y_t}{V}) - \alpha Z_t] = 0, \text{ and}$$

$$\dot{Z} = Z_t [s(1 - \frac{Z_t}{W}) + \beta Y_t] = 0.$$

There are three nontrivial equilibria: $\{Y = 0, Z = W\}$; $\{Y = V, Z = 0\}$; and $\{Y = Y_0, Z = Z_0\}$, where

$$Y_0 = \frac{sV(r - \alpha W)}{\alpha\beta VW + sr},$$

$$Z_0 = \frac{rW(s + \beta V)}{\alpha\beta VW + sr}.$$

[1] A simpler model is the Lotka-Volterra specification:

$$\dot{Y} = G(Y_t, Z_t) = rY_t - \alpha Y_t Z_t$$

$$\dot{Z} = H(Y_t, Z_t) = -\alpha Z_t + \beta Y_t Z_t.$$

This system generates closed-orbit population cycles. It is not structurally stable, however. Small changes in the structure of these functional forms may lead to great changes in the nature of the solution, resulting in converging or diverging spirals. The model cannot, therefore, be considered a valid description of natural biological systems (May; Clark, p. 183).

It can be shown that if the equilibrium solution $\{Y_0, Z_0\}$ exists in the positive quadrant, then that solution is a stable node or focus toward which the system will tend from any nontrivial point of origin (Clark). In the absence of external shocks to the system, the secondary-pest and natural-predator populations approach these levels over time.

When chemical pesticide is introduced, the relevant model of predator-prey biology plus human intervention becomes the following:

$$\dot{Y} = G(Y_t, Z_t) - L(A_t, Y_t)$$
$$= rY_t(1 - \frac{Y_t}{V}) - \alpha Y_t Z_t - lA_t Y_t,$$

$$\dot{Z} = H(Y_t, Z_t) - M(A_t, Z_t)$$
$$= sZ_t(1 - \frac{Z_t}{W}) + \beta Y_t Z_t - mA_t Z_t.$$

The essential properties of the model are unchanged by the introduction of pesticide. The equilibrium shifts but remains stable so long as it still occurs in the first quadrant. The intercepts, but not the slopes, of each isocline are affected. The $\dot{Y} = 0$ isocline shifts downward, and the $\dot{Z} = 0$ isocline shifts to the right. The new equilibrium secondary-pest and predator-populations are

$$Y'_0 = Y_0 + \frac{A_t V(\alpha Wm - sl)}{\alpha\beta VW + sr},$$

$$Z'_0 = Z_0 + \frac{A_t W(-\beta VL - rm)}{\alpha\beta VW + sr}.$$

Since parameters are nonnegative, $Z'_0 < Z_0$, meaning that in equilibrium there are unambiguously fewer natural predators when pesticide is used. The secondary-pest population, however, may be larger or smaller with pesticide use; that is, Y'_0 may be greater or less than Y_0, depending on the biological-parameter values.

Specifically, the effect of an increase in the rate of pesticide use on the secondary pest is given by

$$\frac{dY'_0}{dA_t} = \frac{V(\alpha Wm - sl)}{\alpha\beta VW + sr}.$$

The equilibrium secondary-pest population increases if $\alpha W m > s l$ and decreases if the reverse. Not surprisingly, it is the relative toxicity of the pesticide to the secondary pest and to the predator, l and m respectively, that determines whether the positive externality outweighs the negative externality, or the reverse. The appropriate weights in making this comparison are seen here to be s and αW, where s and W are logistic growth parameters for the predator, and α is the rate of predation.

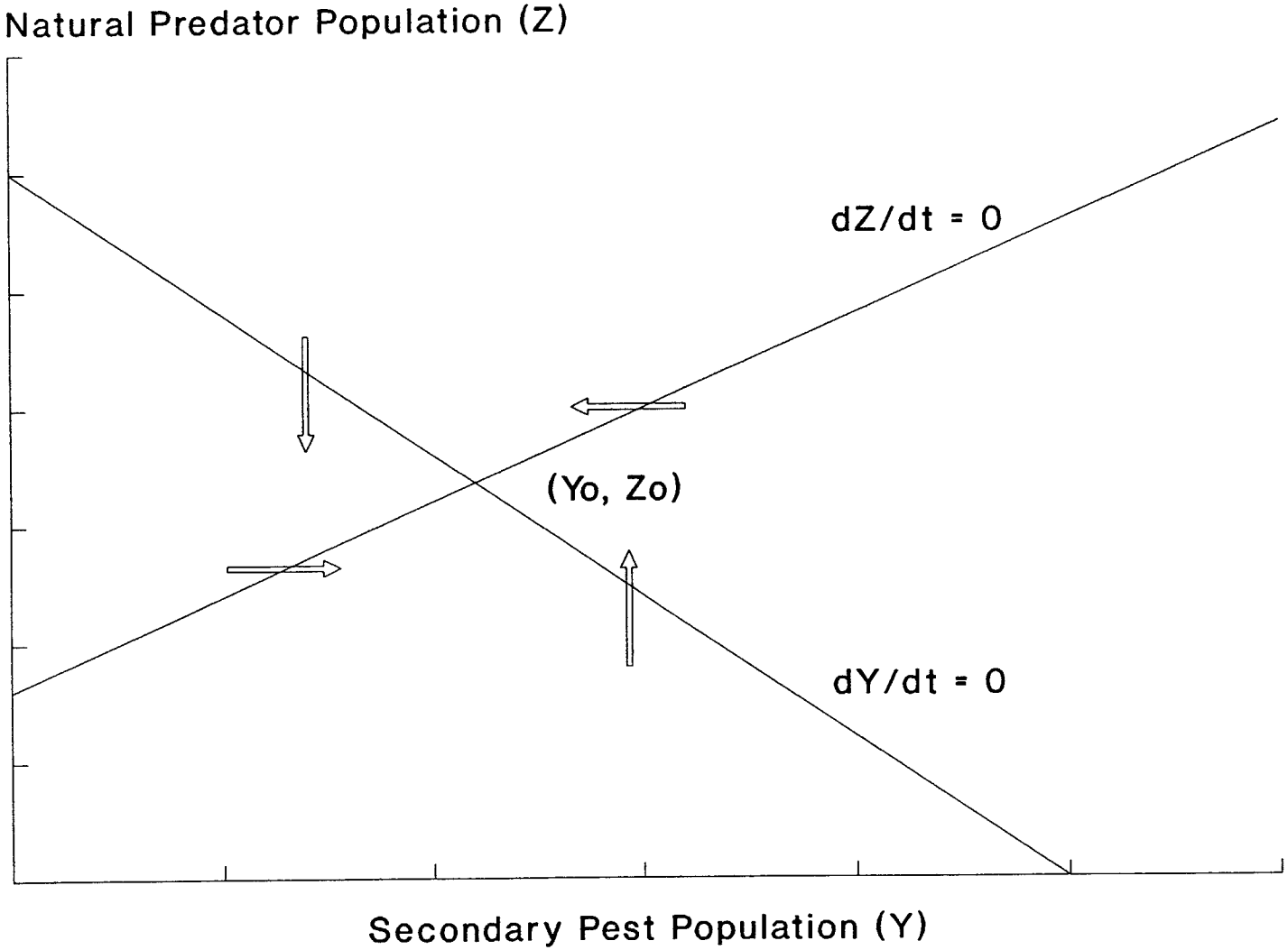

Figure 1. Predator-Prey Model of Secondary Pests

Multiple-Pest Management

In contrast to myopic single-pest management, which considers only the effect of pesticide on the target pest X_t, optimal management takes into account its effects on the complete system. Potential crop damage from secondary as well as primary pests is incorporated, and the rate of pesticide use, A_t, is adjusted to allow for undesirable or desirable effects of the pesticide on the secondary-pest population.

The complete optimization problem is to maximize discounted net revenue over time, subject to all biological interactions:

$$\max \int_0^T e^{-\delta t} [R(X_t, Y_t) - C(A_t)]\, dt,$$

$$\text{subject to } \dot{X} = F(X_t) - K(A_t, X_t)$$

$$\dot{Y} = G(Y_t, Z_t) - L(A_t, Y_t)$$

$$\dot{Z} = H(y_t, Z_t) - M(A_t, Z_t).$$

The Hamiltonian function for the complete problem is therefore a function of the primary- and secondary-pest populations, the predator population, and the amount of pesticide applied:

$$\tilde{H}(X_t, Y_t, Z_t, A_t) = R(X_t, Y_t) - C(A_t) + \mu_X [F(X_t) - K(A_t, X_t)] + \mu_Y [G(Y_t, Z_t) - L(A_t, Y_t)] + \mu_Z [H(Y_t, Z_t) - M(A_t, Z_t)].$$

Because X and Y are pests while Z is beneficial, we expect to find $\mu_X < 0$, $\mu_Y < 0$, and $\mu_Z > 0$.

Necessary conditions for an optimum are

$$(1.1) \qquad 0 = \frac{\partial \tilde{H}}{\partial A_t} = - C_A - \mu_X K_A - \mu_Y L_A - \mu_Z M_A$$

$$(2.1) \qquad \dot{\mu}_X = \delta\mu_X - \frac{\partial \tilde{H}}{\partial X_t} = \mu_X [\delta - F_X + K_X] - R_X;$$

$$\dot{\mu}_Y = \delta\mu_Y - \frac{\partial \tilde{H}}{\partial Y_t} = \mu_Y [\delta - G_Y + L_Y] - \mu_Z H_Y - R_Y;$$

$$\dot{\mu}_Z = \delta\mu_Z - \frac{\partial \tilde{H}}{\partial Z_t} = \mu_Z [\delta - H_Z + M_Z] - \mu_Y G_Z$$

$$(3.1) \qquad \dot{X} = \frac{\partial \tilde{H}}{\partial \mu_X}; \dot{Y} = \frac{\partial \tilde{H}}{\partial \mu_Y}; \dot{Z} = \frac{\partial \tilde{H}}{\partial \mu_Z}$$

(Kamien and Schwartz, p. 132).

The maximum principle, as reflected in condition (1.1), indicates that pesticide should be applied at a rate such that its marginal cost equals the value of its total effect, which includes the current and future impacts of reducing the primary-pest, secondary-pest, and natural-predator populations.

For the Larkin-type model, conditions (1.1) and (2.1) become the following (see appendix):

$$(4.1) \qquad 0 = - c - \mu_X kX - \mu_Y lY - \mu_Z mZ;$$

$$\dot{\mu}_X = \mu_X[\delta - q + 2q\frac{X}{U} + kA] - R_X;$$

$$\dot{\mu}_Y = \mu_Y [\delta - r + 2r\frac{Y}{V} + \alpha X - lA] - \mu_Z \beta Z - R_Y;$$

$$\dot{\mu}_Z = \mu_Z [\delta - s + 2s\frac{Z}{W} - \beta Y - mA] + \mu_Y \alpha Y.$$

The optimal solution to the myopic single pest management problem was seen to approach a steady-state solution ($\dot{A} = \dot{X} = 0$). In addition, the secondary-pest population tends toward a stable equilibrium with its natural predator for any constant rate of pesticide use. It follows that the solution to the complete optimization problem will also approach a steady-state solution ($\dot{A} = \dot{X} = \dot{Y} = \dot{Z} = 0$). An optimal constant rate of pesticide use, A^*, will be chosen that takes into account the effect of the chemical in reducing all three species through the respective pesticide kill functions, K, L, and M.

The equilibrium thus described, if it occurs in the positive quadrant, is a stable node or focus since it results from linear shifts in each isocline. In addition, there are no limit cycles in this model, as can be confirmed using du Lac's test (Clark, pp. 195, 324).

To see how the economic-threshold population, X^*, and the optimal rate of pesticide use, A^*, are influenced by secondary-pest considerations, note that for any pesticide use pattern which is constant over time ($A_t = \bar{A}$), the primary- and secondary-pest populations are also constant:

$$\bar{X} = X(\bar{A}) = U\,(1 - k\bar{A}/q), \text{ and}$$
$$\bar{Y} = y(\bar{A}) = \frac{sV\,(r - \alpha\,W) + V\,(\alpha Wm - sl)\,A}{\alpha\beta VW + sr}.$$

Then discounted profit is simply

$$\int_0^T e^{-\delta t}\,(R\,\{X(\bar{A});\,Y\,(\bar{A})\} - C\,(\bar{A}))\,dt = \int_0^T e^{-\delta t}\,(R\,(\bar{A}) - c\bar{A})\,dt.$$

The optimal solution for the Larkin-type multiple-pest problem is the following:

$$c = -\mu_X kX - \mu_Y lY - \mu_Z mZ;$$
$$0 = \mu_X\,(\delta + q\frac{X}{U}) - R_X;$$
$$0 = \mu_Y\,(\delta + r\frac{Y}{V} + \alpha Z) - \mu_Z\,\beta Z - R_Y;$$
$$0 = \mu_Z\,(\delta + s\frac{Z}{W} - \beta Y) + \mu_Y \alpha Y,$$

which can be solved to obtain

$$c = -R_X \frac{kX}{(\delta + qX/U)} - R_Y \frac{lY\,(\delta + sZ/W - \beta\,Y)}{(\delta + sZ/W - \beta\,Y)\,(\delta + rY/r + \alpha Z) + \beta Z \alpha Y} + R_Y \frac{\alpha\,YmZ}{(\delta + sZ/W - \beta Y)\,(\delta + rY/V + \alpha Z) + \beta Z \alpha Y} \tag{5.1}$$

This expression indicates that marginal cost, c, should equal the sum of marginal benefits, which consist of three types. The first right-hand term is identical to that in equation (6) for the single pest management problem, reflecting the pesticide's marginal contribution to crop value by reducing primary-pest damage. The second term, which has the same sign as the first, reflects the additional economic benefit resulting from the destruction of secondary pests. The third term, opposite in sign to the other two, reflects the *loss* in benefits resulting from the destruction of the natural predator.

In principle, it is possible for net economic benefits from pesticide to increase when secondary-pest effects are taken into account if the second right-hand term outweighs the third. One might conclude that a prescription for heavier use of pesticides would be a common result of complete biological modeling. Historical experience suggests, however, that in many settings the harm done by destruction of natural pest predators outweighs the immediate benefit that results from toxicity to secondary pests, particularly when resistance effects are taken into account. In U.S. cotton production alone, severe secondary-pest outbreaks have included, besides cotton bollworm, the tobacco budworm and cotton leafperforator. In situations such as these, the optimal level of pesticide use therefore tends to be reduced when the secondary pest and its predator are taken into account.

In evaluating optimal pesticide use in particular cases, correct specification of the revenue function, $R[X;Y]$, representing biological interactions between pests and plants, plays just as important a role as correct specification of the interactions among pests and predators (Lichtenberg and Zilberman). It is necessary to know something about not only the toxicity of the pesticide to all three species, but also the relative marginal crop damage that can be expected from primary and secondary pests. If the impact of the secondary pest is large when its population increases, and if the pesticide is more toxic to the natural predator than to the secondary pest itself, it is very possible for pesticide use to increase net pest damage, even when it is effective in reducing the primary-pest population.

Resistance in the Secondary Pest

The predator-prey model implies that if either the pest or its predator should develop resistance to the pesticide in the future, the pesticide kill functions

and, hence, the equilibrium secondary-pest population will change, even if pesticide use is continued at the same level. Historically, it has often been secondary pests, rather than primary pests or natural predators, that have developed resistance to pesticides, in some cases to one chemical after another. "For a variety of genetic, behavioral, and ecological reasons, predators and parasites of pests are much less able than pests themselves to build into pesticide-resistant populations" (Prokopy, p. 11).

In the present model, the effect of secondary-pest resistance is to decrease kill effectiveness, l, and increase the equilibrium secondary-pest population. One familiar scenario for resistance is that initially the pesticide is a good control for secondary as well as primary pests. Even though the pesticide reduces the number of predators, the secondary pest is kept in check better by the combined effect of predators and pesticide than it was by predators alone (i.e., $Y'_0 < Y_0$). As resistance develops in the secondary pest, however, the pesticide effect becomes weaker, and the reduced number of predators permits the equilibrium secondary-pest population, Y'_0, to rise above the prepesticide level, resulting in heavier and heavier crop damage.

Another scenario is that the use of pesticide drives the natural-predator population to extinction in the local geographical area, leaving the secondary pest to be controlled by pesticide alone. This state of affairs may initially be satisfactory for the producer. As the secondary pest becomes resistant to the pesticide over time, however, the absence of natural biological controls becomes evident. In this case, an additional pesticide must be used to control the secondary pest, or else natural predators must be reintroduced along with a less intensive chemical pesticide regime.

Conclusions

Although it is often acknowledged in principle that pest managers need to address multispecies interactions, this is seldom done in practice. This paper has attempted to identify an extremely common, but easily overlooked, situation: that in which a pest of secondary economic importance becomes transformed into a major threat as a result of the system of chemical control used against a target pest. For many crops, secondary pests have the potential to inflict crop damage that is at least as devastating as that due to primary pests if the secondary-pest population is permitted to increase to a sufficient level.

The effects of chemical pesticide on a predator-prey system consisting of the secondary pest and its natural enemy often function as a pest-control externality. Ignoring secondary pests can lead to devastating crop damage that may continue over a considerable period of time. Induced secondary-pest infestations, once they arise, may prove difficult to control by chemical means. Many secondary pests have rapidly developed resistance to one chemical after another. Some highly successful integrated pest management strategies, such as the one now used in high plains cotton, originated largely out of failure to achieve chemical control of secondary pests.

Crop ecosystem models that incorporate all relevant pests and natural predators are required to determine economically optimal pest management. Chemical control of a primary pest should ideally be used with knowledge of its likely effects on not only natural predators of the target pest, but also natural predators of relevant secondary-pest species. In addition, complete modeling is needed in the regulatory environment, when costs and benefits of various pest controls are evaluated, to avoid overestimating economic benefits attributable to chemical controls.

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Appendix

A general expression for the optimal time path of pesticide use can be derived as follows. From (1),

$$\mu_t = -C_A/K_A$$

and hence

$$\dot{\mu} = -\frac{C_{AA}}{K_A}\dot{A} + \frac{C_A}{K^2_A}[K_{AA}\dot{A} + K_{AX}\dot{X}],$$

which with (2) can be solved to get a general optimal solution:

$$\dot{A} = \frac{-R_X K^2_A - C_A K_A[\delta - F_X + K_X] - C_A K_{AX}[F(X_t) - K(A_t, X_t)]}{-C_{AA}K_A + C_A K_{AA}}.$$

For the Schaefer-type model with constant marginal costs of pesticide, (1) and (3) give

$$\dot{\mu} = \frac{c\dot{X}}{kX_t^2} = \frac{c(qX_t - qX_t^2/U - kA_t X_t)}{kX_t^2}$$

and (2) gives

$$\dot{\mu} = -R_X + \mu_t\frac{\delta - q + 2qX_t/U + kA_t}{kX_t}.$$

These two expressions can be solved to obtain expression (4).

For the multiple pest management problem, conditions (4.1) are derived as follows. For the Larkin-type model, (1.1) through (3.1) give

$$c = -\mu_X kX - \mu_Y lY - \mu_Z mZ;$$

$$\mu_X\left[\delta - q + \frac{2qX}{U} + kA\right] - R_X = 0;$$

$$\mu_Y\left[\delta - r + \frac{2rY}{V} + \alpha Z + lA\right] - \mu_Z \beta Z - R_Y = 0;$$

$$\mu_Z\left[\delta - s + \frac{2sZ}{W} - \beta Y + mA\right] + \mu_Y \alpha Y = 0.$$

However, in a steady state, $qX(1 - \frac{X}{U}) = kAX$, $rY(1 - \frac{Y}{V}) = lAY$, and $sZ(1 - \frac{Z}{W}) = mAZ$. This gives condition (4.1).