Ecological Modelling, 20 (1983) 1–19
Elsevier Science Publishers B.V., Amsterdam – Printed in The Netherlands

MULTISEASONAL MANAGEMENT OF AN AGRICULTURAL PEST. * I: DEVELOPMENT OF THE THEORY

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(Accepted for publication 28 February 1983)

ABSTRACT

Mangel, M. and Plant, R.E., 1983. Multiseasonal management of an agricultural pest. I: Development of the theory. *Ecol. Modelling*, 20: 1–19.

A framework for analyzing the trade-off between economic yield from a crop and buildup of resistance to pesticide caused by repeated applications of pesticide is developed. The analysis begins with the case of age-independent pest dynamics, in which pests infest a field by arriving from an external pool. Initially, it is assumed that the pest genetics of interest are single locus, two allele, with resistance to pesticide dominant and susceptible pests more fit in the absence of spraying. The pesticide is applied only once during the season, with timing and intensity of the application as control variables. Interseasonal pest and crop dynamics are studied by solving appropriate ordinary differential equations. Intraseasonal pest dynamics are assumed to follow the Hardy–Weinberg formula. It is shown that the three class diploid model can be replaced by a two class haploid model with essentially no change in the results. A model based on partial differential equations is developed, for the case in which pest dynamics depend upon age, and it is shown that the partial differential equation model can be replaced by a pair of coupled ordinary differential equations. The main operational conclusion in this paper is that the timing of the application of pesticide can be used to control buildup of resistance and that the intensity of the application can be used to control the crop yield.

INTRODUCTION: THE AGRICULTURAL DECISION PROBLEM

It is common in many agricultural enterprises to use pesticides to control pests. It is also commonly observed that as pesticides are applied, resistance to the pesticide builds up as susceptible pests are removed from the population. Thus, an agricultural manager is faced with a decision problem of the following kind: By spraying his field this year, to increase the present year's economic yield, he increases resistance to the pesticide, which, in principle,

* Giannini Foundation Paper.

0304-3800/83/$03.00

reduces future yields. It is this decision problem that is studied here and in an accompanying paper by Plant et al. (1983). The problem is of sufficient complexity that the two papers use only deterministic approaches. A third paper will include stochastic effects in our models.

The goals in these papers are to develop a framework in which questions about the trade-off between resistance and yield can be posed and then to answer some of the more important questions. The models are formulated in a way that is sufficiently realistic to provide useful results, but, at the same time, is sufficiently simple to understand the behavior of the model. Many of the ideas used here have previously appeared in the literature in some modified form, see for example the papers by Hall and Norgaard (1973); Hueth and Regev (1974); Gutierrez et al. (1975); Regev, Gutierrez and Feder (1976); Georghiou and Taylor (1976; 1977a, b); Georghiou (1980); Headley (1981); and Shoemaker (1982). The approach in Shoemaker's paper is conceptually similar to this one, but the operational formulation and analytical tools are quite different.

One agricultural system that examplifies the kind of problem of interest is the cotton-spider mite (Aracina: Tetranychidae) system in the San Joaquin valley in California. Some of the pest dynamics were examined by Carey and Bradley (1981). This system has the following features. The cotton is an annual crop grown every other year, frequently in rotation with wheat, a crop that does not support the mites. Although some mites over-winter in the field, mites also immigrate to the field at some rate throughout the entire season, coming from external sources such as fruit orchards and weed patches. These external sources of the mites appear to be much more important than the over-wintering mites already in the field. It is also known that in greenhouses, where there is no alternation of crops and no external source of non-resistant mites, resistance to the commonly used pesticides builds up quickly.

The modelling approach is based on a submodel for the pest dynamics and a submodel for the crop dynamics. The pest submodel has the following goal: given the fractions of resistant and susceptible pests in the population at year n and the spraying strategy in year n, how does one find the respective fractions in year $n + 1$? In order to answer this question, one must include population dynamics and relatively simple genetics in the pest submodel.

The submodel for the crop dynamics has the following goal: given the pest populations at the start of year n, and a spraying strategy in year n, what is the yield of crop in year n? Using this submodel one can also determine the optimal spraying strategy within a single season.

The models do not include the effect of biological controls such as predators. In the case of the spider mite–cotton system in the San Joaquin

valley, there is some question as to whether natural predators play an important role in controlling outbreaks, but in any case the inclusion of the effects of predators in the model at this time would not provide useful information.

The three primary parameters in the application of a pesticide in a given season are the number of applications, the timing of the applications, and the intensity of the applications. In the next paper (Plant et al. 1983) the first parameter is considered; for the present it is assumed that a single pesticide application occurs each year. This allows one to study the effects of timing and intensity. In Sections 2 and 3, a simple age-independent model of the pest-crop interaction is given and the effects of varying the timing and intensity of pesticide application are examined. Since the accumulation of pesticide resistance in the population is important, the effect of assumptions about the genetics on the behavior of the model is studied.

Previous simulation studies (e.g. Regev et al., 1976), have shown that the age structure of the pest population plays an important role in determining pest control strategies. Dependence on age structure occurs in two aspects of the model: susceptibility to the pesticide, and consumption of the crop. The introduction of age structure greatly complicates any model. This complication is minimized by assuming that pests inhabit colonies founded by a pregnant female. Spider mites behave this way (Carey and Bradley, 1981). The colonies have an age structure that depends on the age of the colony; for example, younger colonies have more eggs and larvae, and older colonies have more adults. Carey and Bradley (1981) have studied the demography of these mite colonies.

The model is formulated in terms of age structure of colonies; it is assumed that from this the age structure of the population may be inferred. This greatly simplifies the formulation. It does, however, have one serious consequence. If the pest population has an age-specific susceptibility to the pesticide, then each application of the pesticide will change the age structure of the already established pest colonies. This is of no consequence if crop consumption is not age specific and if only one application of the pesticide is made. For the present, the change in age structure due to pesticide application is neglected. In the study of the age structured model, a simplified formulation that adequately represents the age-dependent effects is presented. This model does not require the assumption of a single pesticide application per season.

The unit of time in the model is the physiological time, degree–days. It is assumed that the crop and pests are affected by physical temperature in the same way. This assumption could be dropped in a simulation. The utility of the models, when compared to simulations, is that because of the simplifying assumptions it is much easier to understand the crop, pest, and resistance dynamics than it is in simulations.

THE AGE-INDEPENDENT MODEL

To start, consider a model for the case in which pesticide susceptibility and crop consumption are independent of age. Let $X(t)$ be the total number of pests in the field at time t, and let $C(t)$ be a measure of the value of the crop. The most commonly used such measure is the leaf mass (Gutierrez et al., 1975). Assume that the crop is harvested before density dependent effects become important. The equation for the crop dynamics is therefore

$$\frac{dC}{dt} = \begin{cases} r_c C - vX & : \quad C(t) > 0 \\ 0 & : \quad C(t) = 0 \end{cases} \tag{1}$$

$$C(0) = C_0, \; 0 \le t \le T$$

where C_0 is a positive constant. The parameter r_c is the intrinsic growth rate of the crop, v is a measure of the pest's unit rate of consumption of the crop, and T is the length of the season.

According to eq. 1, the crop decreases as soon as $r_c C < vX$. This is the simplest possible assumption about the crop–pest interactions. Other assumptions, such as replacing vX by vXC give similar qualitative results. T is fixed and finite; for the parameters used here, $C(T)$ is always bounded away from zero.

Consider now the model for the pest dynamics, starting with the simplest model and then increasing in complexity. All models are based on the following operational picture. At the start of season n the agricultural field has negligibly few pests in it. There is an external source of pests, which we call a pool, and pests leave the pool and arrive at the field at a rate $I(t)$, where t denotes time within a season. The length of the agricultural season is T; at the end of the season the remaining pests in the external pool and pests in the agricultural field are mixed together for the overwintering process.

To simplify the derivation of the pest dynamics, begin by considering a model with only one class; thus, effectively, assume that all pests are susceptible to the pesticide. When it is important to distinguish the pest population in year n, it will be denoted by $X(t; n)$. When there is no possibility for confusion, $X(t)$ will be used.

Within a season, the intensity of spraying in year n is denoted by $s(t; n)$. In most cases, assume that the pesticide is applied only once and that $s(t; n)$ takes the form

$$s(t; n) = \begin{cases} 0: & 0 \le t < t_s(n) \\ \eta(n): & t_s(n) \le t < t_s(n) + \delta(n) \\ 0: & t_s(n) + \delta(n) \le t \le T \end{cases} \tag{2}$$

Here $\eta(n)$ is the intensity of the spraying in year n and $\delta(n)$ is the length of

time that the pesticide is active in year n. As before, the argument n is dropped when no confusion results. As with the crop component, it is assumed that the pest population does not reach its carrying capacity before the end of the season. The model is therefore linear. A consequence of this assumption is that the growth rate of the pest population is independent of the value of C, the crop. Therefore, the differential equation for the pest component is

$$\frac{dX}{dt} = r(t; n)X + I(t; n) \tag{3}$$

$$X(0; n) = 0, 0 \leq t \leq T$$

Here, $I(t; n)$ is the rate of immigration from the pool at time t in year n and $r(t; n)$ is the intrinsic rate of increase of the population at time t in year n. It depends upon n and t through the spraying function; i.e., when pesticide is applied, $X(t)$ does not instantaneously decrease, but decreases over some finite period of time. This decrease can be modeled by making $r(t; n)$ negative. In particular, the following form is chosen

$$r(t; n) = r_0\left\{1 - \frac{\omega s(t; n)}{e + s(t; n)}\right\} \tag{4}$$

In the formula, r_0 is the intrinsic growth rate in the absence of spraying, and ω and e measure the effects of the spraying. The parameter ω measures the maximal pesticide effect in the sense that as $s(t)$ approaches infinity, $r(t; n)$ approaches $r_0(1 - \omega)$. The parameter e is related to LD_{50} (the intensity required to kill 50% of the pests) *. If e is small (as would be the case with susceptible pests) then the LD_{50} is reached at low levels of $s(t)$; if e is large (as would be the case with resistant pests) then the required value of $s(t)$ is large.

The next step is to incorporate genetics into the models. The philosophy here is that in dealing with the genetics of the system, one should use the simplest assumptions possible, even at the cost of sacrificing some accuracy in the model. Hence, no effect is made to take into account the particular genetic characteristics of a pest species (such as the haplo-diploidy of mites, or the ratio of inbreeding to outcrossing). Rather, it is assumed that whatever these characteristics are, their effects may be accounted for by simple, empirical parameters.

The simplest point of departure is a single locus, two allele, genetic model,

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* If η_{50} is the intensity corresponding to LD_{50}, and the pesticide is active for δ units of time, then $e = \omega\eta[1 - 1/\delta r_0 \ln(0.5)]^{-1} - \eta$.

with resistance to pesticide dominant. Let

$$\left.\begin{aligned} X_1(t;n) &= \text{number of homozygous resistant (RR) pests in the field at time } t \text{ in season } n \\ X_2(t;n) &= \text{number of heterozygous resistant (RS) pests in the field at time } t \text{ in season } n \\ X_3(t;n) &= \text{number of homozygous susceptible (SS) pests in the field at time } t \text{ in season } n \end{aligned}\right\} \tag{5}$$

In analogy to eq. 3, assume that these variables satisfy the equations

$$\left.\begin{aligned} \frac{dX_1}{dt} &= r_1(t;n)X_1 + \mu_1(n)I + \tfrac{1}{2}\alpha X_2 \\ \frac{dX_2}{dt} &= r_2(t;n)X_2 + \mu_2(n)I - \alpha X_2 \\ \frac{dX_3}{dt} &= r_3(t;n)X_3 + \mu_3(n)I + \tfrac{1}{2}\alpha X_2 \\ X_i(0;n) &= 0,\ i = 1, 2, 3;\ 0 \le t \le T \end{aligned}\right\} \tag{6}$$

In these equations the $r_i(t;n)$ are the growth rates of pests of class i; assume that they take the form

$$\left.\begin{aligned} r_1(t;n) &= r_{10}\left\{1 - \frac{\omega s}{e_1 + s}\right\} \\ r_2(t;n) &= r_{20}\left\{1 - \frac{\omega s}{e_1 + s}\right\} \\ r_3(t;n) &= r_{30}\left\{1 - \frac{\omega s}{e_2 + s}\right\} \end{aligned}\right\} \tag{7}$$

where $s = s(t;n)$.

In eqs. 7, assume that $r_{30} = r_{20} > r_{10}$, so that susceptible pests and heterozygotes are more fit, in the absence of spraying. It it also assumed that $0 < e_2 \ll e_1$, so that a given intensity of spraying affects the susceptible pests much more than the resistant ones. Note that heterozygotes have the best of both worlds in that they have resistance and a high growth rate. This appears to be the case with the spider mite (J. Carey, personal communication). As before, $I(t;n)$ is the rate at which pests arrive from the pool at the field in year n. In eqs. 6, $\mu_i(n)$ is the fraction of pest class i in the pool during year n.

In eqs. 6, α characterizes the rate at which heterozygosity is lost. For a particular genetic model, α can be found according to an algorithm relating genetic to physiological time (Georghiou and Taylor (1977a, b), Haldane (1937)). The parameter α depends on the rate of outcrossing between colonies, the number of generations per season, and the genetics of the pest.

The model relates $\mu_i(n+1)$ to $\mu_i(n)$ as follows. Set

$$p_i(n) = \frac{X_i(T; n)}{\Sigma X_i(T; n)} \tag{8}$$

so that $p_i(n)$ is the fraction of the population in the ith class at the end of the season. Assume that the fractions of the ith class at the start of year $n+1$ are given by the Hardy–Weinberg relationship

$$\left.\begin{aligned} \mu_1(n+1) &= \left(p_1(n) + \tfrac{1}{2}p_2(n)\right)^2 \\ \mu_2(n+1) &= 2\left(p_1(n) + \tfrac{1}{2}p_2(n)\right)\left(p_3(n) + \tfrac{1}{2}p_2(n)\right) \\ \mu_3(n+1) &= \left(p_3(n) + \tfrac{1}{2}p_2(n)\right)^2 \end{aligned}\right\} \tag{9}$$

In order to write eqs. 9, it is assumed that there are no pests in the pool at the end of the season, that there is no change in the ratios of the classes during the wintering period, and that the Hardy–Weinberg formula is applicable. Since the ultimate goal is to use these models for control of resistance, this assumption will imply that the strategy is conservative (since there would be a higher fraction of susceptible pests in the pool and, in general, in the absence of spraying, the susceptibles would grow at a faster rate). In the light of the many uncertainties in a real agricultural system, and of the stochastic effects, such a conservative approach seems reasonable.

To complete the specification of the model, one must specify the immigration rate $I(t;n)$. A very simple form is chosen; namely

$$I(t; n) = I_0(1 - I_c/T) \tag{10}$$

In the next section, the solutions of the model given by eqs. 1–10 are studied.

SOLUTIONS OF THE AGE-INDEPENDENT MODEL

This section contains some results obtained using the model. The economic quantity of interest is the total discounted yield of crop over the economic horizon. Suppose that the horizon is M years. Then the total yield is

$$J = \sum_{n=1}^{M} \rho^{n-1} C(T, n) \tag{11}$$

TABLE I

Parameter	Value	Parameter	Value	Parameter	Value
I_0	1	e_1	35	ρ	0.9
I_c	1	e_2	5	$\mu_1(0)$	10^{-8}
r_c	0.1	r_{10}	0.2	M	10
v	0.005	r_{20}, r_{30}	0.21	C_0	1
T	20	δ	2		
α	1	η	2		
t_s	6	ω	20		

where ρ is a discount factor and $C(T, n)$ is the yield of crop at the end of the season in year n. Another useful quantity for comparisons is

$$f(n) = C(T, n)/C_0 \, e^{r_c T} \tag{12}$$

which is the fraction of potential crop yield in year n.

Since the models consist of linear differential equations, the solutions may be determined analytically. However, the complexity of these solutions eliminates their usefulness, so the solutions are obtained numerically. Parameter values chosen for the standard case are given in Table I.

These values were chosen so that an application of the pesticide causes an 85% reduction in the susceptible population and a 5% reduction in the resistant population.

The two control parameters are η, the intensity of spraying, and t_s, the initiation time of spraying. Table II shows the effect of η and t_s on the total yield J of eq. 11.

For these parameter values, an increase in η leads to an increase in discounted total yield, while an increase in t_s leads to an initial increase followed by a decrease in total yield. These results may be explained by examining Figs. 1 and 2. Figure 1 shows the effects of varying t_s. Figure 1a gives the relative yield $f(n)$ of eq. 3.2, and Fig. 1b gives the fraction $\mu_3(n)$ of susceptibles. Both $f(n)$ and $\mu_3(n)$ are functions of a discrete variable; however, to aid the eye they are plotted as continuous curves. When t_s is

TABLE II

η	t_s	J
2	6	32.1
1.5	6	30.4
2.5	6	33.2
2	4	31.2
2	8	29.0

increased, initial yield increases up to an optimal point at $t_s = 6$. Yield in later years declines dramatically; the effect of the discount rate, however, makes these crop yields less valuable than those in the present and near future. The portion $\mu_3(n)$ of susceptibles declines more rapidly as t_s is increased, because the susceptible population has less remaining time to recover from the effects of the pesticide. When t_s is very early (e.g. $t_s = 2$), the slightly higher growth rate of the susceptible population allows it to recover almost completely.

Figure 2 shows the dependence of $f(n)$ (Fig. 2a) and $\mu_3(n)$ (Fig. 2b) on the spray intensity η. The values $\eta = 1.5$, 2, and 2.5, are regarded as represensative of a "moderate" intensity of spraying. As shown in Fig. 2a, the yield ratio $f(n)$ increases over most of the ten seasons with increasing η. Figure 2b shows that the susceptible ratio $\mu_3(n)$ does not strongly depend on η. The curves for the high intensity $\eta = 5$ deserve special comment. Note in

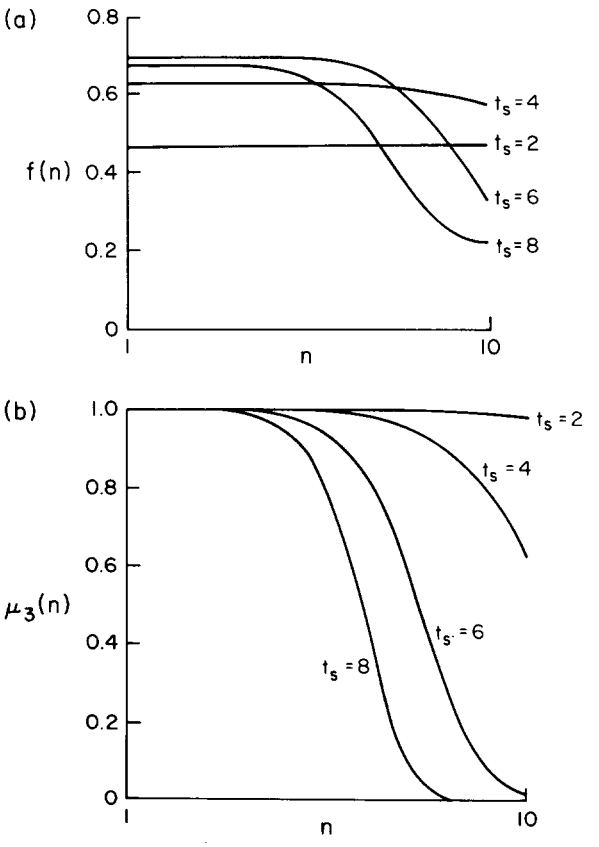

Fig. 1. (a) Crop yield in year n plotted against year for varying timings of pesticide application. (b) Fraction of susceptibles in the population.

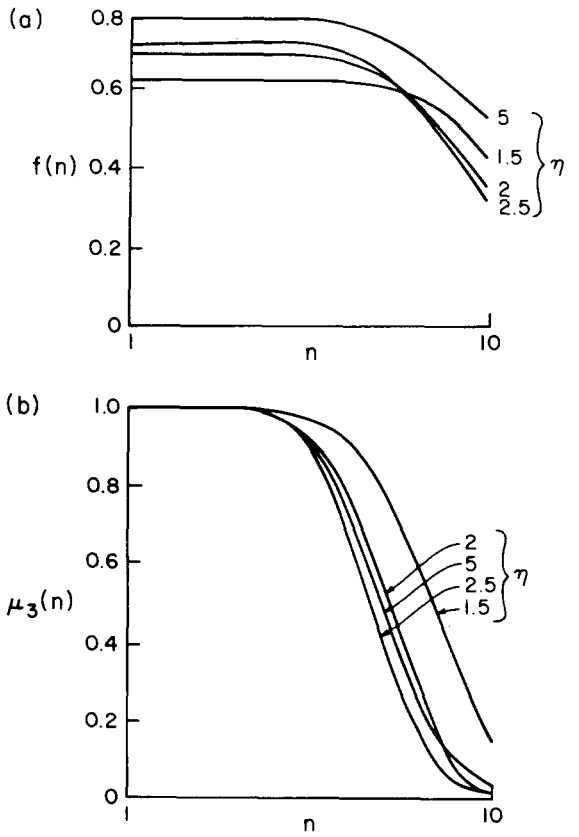

Fig. 2. (a) Crop yield in year n plotted against year for varying intensities of pesticides. (b) Fraction of susceptibles in the population.

Fig. 2b that the fraction of susceptibles actually *increases* as a result of this very high intensity spraying. This seemingly paradoxical result is amplified in Fig. 3. This figure shows $\Delta\mu_3(1)$, which is defined as $\mu_3(2) - \mu_3(1)$, as a function of η. Note that $\Delta\mu_3(1)$ declines steadily, as expected, through the moderate region, but then begins to rise at $\eta = 3.25$.

To help clarify this phenomenon, define the quantity $g_i(\eta)$ by

$$g_i = e^{r_i \delta} \tag{12}$$

where the r_i are as defined in eq. 7 with $s = \eta$. The quantity g_i is the ratio $x_i(t+\delta)/x_i(t)$ if there were no immigration during the period of length δ. Figure 4 shows plots of $g_i(\eta)$ for the resistant (R, $i = 1$) and susceptible (S, $i = 3$) cases. The plot of $g_2(\eta)$ is virtually identical with that of g_1. Note from this figure that the effect of pesticide on the susceptible population has

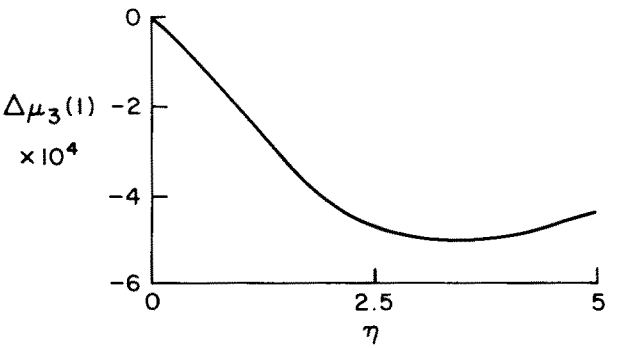

Fig. 3. Single year change in the fraction of susceptibles as a function of the intensity of pesticide application.

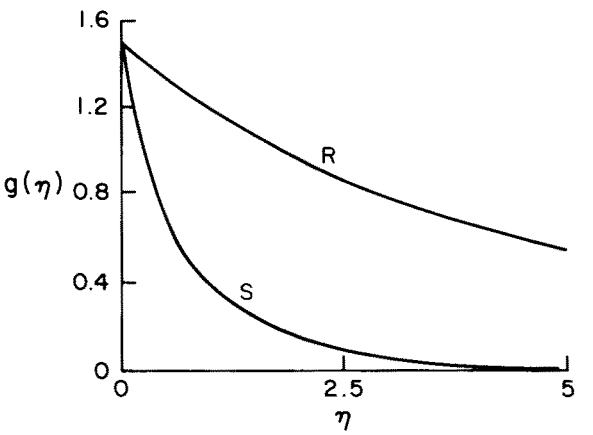

Fig. 4. The function $g_i(\eta)$ for resistant (R) and susceptible (S) pests.

virtually saturated at $\eta = 2.5$, while increasing η continues to have an increasing effect on the resistants. Thus, very high spray intensities, if they kill a significant portion of the resistant population as well as the susceptibles, may actually decrease the resistance for the population. This phenomenon was also noted by Georghiou and Taylor (1977a, b) and Taylor and Georghiou (1982).

Figure 5 shows the effect of changes in assumptions on the genetics of the model. The standard case is that in which the parameter α is 1. The size of α reflects the degree of outcrossing between colonies and the number of pest generations during the season. When $\alpha = 0$ there is either random mating or only one generation per season. As shown in Fig. 5, the value of α makes virtually no difference in the model. The third curve in Fig. 5 corresponds to the case in which there are only two classes, resistants and susceptibles.

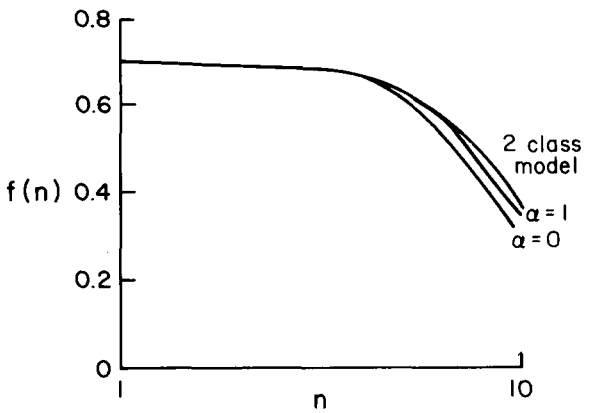

Fig. 5. Comparison of yields predicted by the three pest class ($\alpha = 0$ and $\alpha = 1$) and two pest class models.

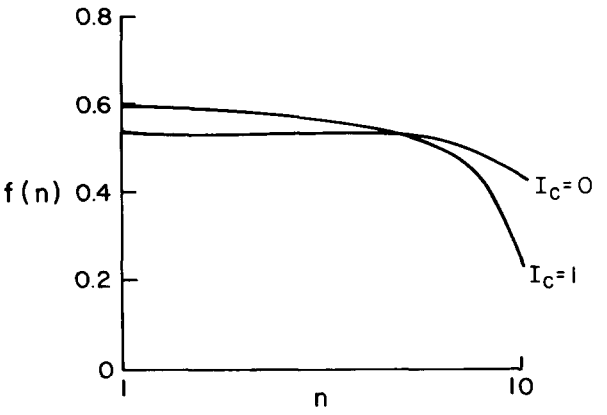

Fig. 6. Comparison of yields for decreasing ($I_c = 1$) and constant ($I_c = 0$) influxes of pests from the pool.

Equations 9, which define the $\mu_i(n+1)$, are replaced by

$$\mu_i(n+1) = \frac{X_i(T; n)}{X_1(T; n) + X_2(T; n)}, \quad i = 1, 2 \tag{13}$$

As shown in Fig. 5, the results with this model are almost identical with those of the three-class model.

Figure 6 shows the effect of changing the parameter I_c in eq. 10 from $I_c = 1$ to $I_c = 0$. The lower value of I_c results in increased immigration late in the season, which reduces the yield when n is small. The increased immigration, however, brings in more susceptibles, which help maintain the susceptibility of the population, raising the yield in later years.

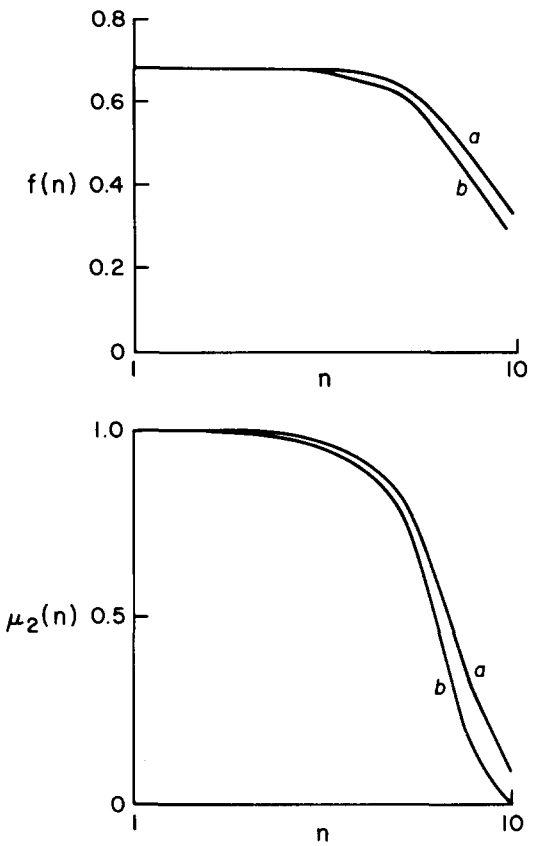

Fig. 7. Comparison of yields for models with differential fitness in the absence of spraying (a) and identical fitness in the absence of spraying (b, with $r_{i0} = 0.2$).

Figure 7 shows the effect of dropping the assumption that susceptible and intermediate pests have a competitive advantage. As expected, if the intrinsic growth rate of the resistants is equal to that of the other classes, then the resistants dominate the population more quickly. The difference in the two cases, however, is surprisingly small. The reason for this is that during the early seasons the population of resistants and intermediates is very small. Therefore, even though the population of susceptibles is reduced by 80–90% after the pesticide application, it is still several orders of magnitude larger than the resistant and intermediate populations.

THE EFFECT OF AGE STRUCTURE ON THE MODEL

In this section the effect of the age structure of the pest component in the dynamics of the model is studied. The formulation of the model incorpo-

rated age dependence in both susceptibility to the pesticide and consumption of the crop. Numerical results are presented only for age dependent susceptibility, however, since crop consumption plays no role in accumulation of resistance by the pest component of the model.

As mentioned in the introduction, age dependence is initially built into the model in the form of a von Foerster-like equation in which the age of the pest *colonies*, rather than that of individual pests, is measured. This is useful since it greatly simplifies the model, but it has the disadvantage of only allowing one application of the pesticide per season. Therefore, an approximate model which divides the pest population into the categories young and old is developed. This has the advantages of replacing the partial differential equations of the model with ordinary differential equations, and eliminating the need to assume only one pesticide application per year.

To begin, let $x(t, a)\mathrm{d}a$ be the number of pests in the field at time t in colonies of age a to $a + \mathrm{d}a$. Assume that the pests in the field grow at a rate $r(t, a)$; we will specify $r(t, a)$ below. With these assumptions, compare $x(t + h, a + h)$ and $x(t, a)$:

$$x(t + h, a + h) - x(t, a) = r(t, a)x(t, a)h + \mathrm{o}(h) \tag{14}$$

Adding and subtracting $x(t, a + h)$ to the left hand side, dividing by h and letting $h \to 0$ gives the equation

$$\frac{\partial x}{\partial t} + \frac{\partial x}{\partial a} = r(t, a)x(t, a) \tag{15}$$

Next, consider boundary conditions for this equation. By assumption, there are no pests present at time 0. Thus $x(0, a) = 0$ for all a. In fact, it must be that $x(t, a) = 0$ if $a > t$. Now consider $x(t, 0)$. Assume that pests arrive at a rate $I(t)$. Thus

$x(t, 0)\Delta t$ = number of pests in colonies of age $(0, \Delta t)$ at time t

$$= \int_{t-\Delta t}^{t} I(s)\mathrm{d}s + \mathrm{o}(\Delta t) \tag{16}$$

Using a Taylor expansion in eq. 16, dividing by Δt, and letting $\Delta \mathrm{t} \to 0$ gives the condition

$$x(t, 0) = I(t) \tag{17}$$

The intrinsic growth rate $r(t, a)$ (or, more correctly, $r(t, a; n)$) is specified by an equation similar to eq. 4 except with an age dependent ω. If, as is the case of Kelthane acting on mites, the eggs and larvae are primarily affected, then one would expect $\omega(a)$ to decline with a. This is because older colonies in general may be expected to contain a lower number of eggs and larvae. Conversely, if a given pesticide were more effective against adults, one would expect $\omega(a)$ to increase with a.

If ω is independent of a, one may convert eq. 15 to an ordinary differential equation by direct integration. Let

$$X(t) = \int_0^t x(t, a)\mathrm{d}a \tag{18}$$

Integrating eq. 15 yields

$$\int_0^t \frac{\partial x}{\partial t}\mathrm{d}a + \int_0^t \frac{\partial x}{\partial a}\mathrm{d}a = r(t)\int_0^t x(t, a)\mathrm{d}a \tag{19}$$

Exchanging the order of integration and differentiation, applying the boundary condition (17) and noting that $x(t, t) = 0$, yields

$$\frac{\mathrm{d}X}{\mathrm{d}t} = r(t)X + I \tag{20}$$

which is the same as eq. 3.

To incorporate genetics, the result of Section 3—that the behavior of the two-class model is essentially the same as that of the three-class one (cf. Fig. 5)—is used. Therefore, one can use the two-class model and let $x_1(t, a)\mathrm{d}a$ denote number of resistant pests in colonies of ages a to $a + \mathrm{d}a$, and similarly define $x_2(t, a)$ for susceptibles. The equations of the pest submodel then become

$$\begin{aligned}
\frac{\partial x_1}{\partial t} + \frac{\partial x_1}{\partial a} &= r_{10}\left\{1 - \frac{\omega(a)s(t; n)}{e_1 + s(t; n)}\right\}x_1 \\
\frac{\partial x_2}{\partial t} + \frac{\partial x_2}{\partial a} &= r_{20}\left\{1 - \frac{\omega(a)s(t; n)}{e_2 + s(t; n)}\right\}x_2
\end{aligned} \tag{21}$$

together with the boundary conditions

$$x_i(0, a) = 0,\ x_i(t, 0) = \mu_i(n)I(t). \tag{22}$$

For the crop submodel, replace eq. 1 with

$$\frac{\mathrm{d}C}{\mathrm{d}t} = r_c C - \sum_{i=1}^{2}\int_0^t v(a)x_i(t, a)\mathrm{d}a \tag{23}$$

$$C(0) = C_0$$

All other equations remain unchanged. In eq. 13, the value of $X_i(t; n)$ is obtained from

$$X_i(T; n) = \int_0^T x_i(T, a; n)\mathrm{d}a \tag{24}$$

To complete the specification of the model one must give the form of $\omega(a)$. For the case in which $\omega(a)$ declines with a, a useful model is

$$\omega(a) = \omega_0 \mathrm{e}^{-ma} \tag{25}$$

and for the case in which $\omega(a)$ increases with a, a useful model is

$$\omega(a) = \omega_0(1 - e^{-ma}) \tag{26}$$

As with the age-independent model, the equations of the age-dependent model may be solved analytically, but here they are solved numerically. The difference scheme for eqs. 4.2 is taken from John (1978) and is

$$x_i(t + h, a + h) = r_i(t, a)x_i(t, a) + x_i(t, a), \; i = 1, 2 \tag{27}$$

The parameter values used in the numerical solution were those given in Table I. The parameter m in eqs. 25 and 26 was fixed at 0.1.

Figure 8 shows the results. Curves "a" correspond to the age-independent case ($m = 0$). These are not quite identical to the "standard" case curves of Section 3 because the curves of Fig. 8 were generated using the partial

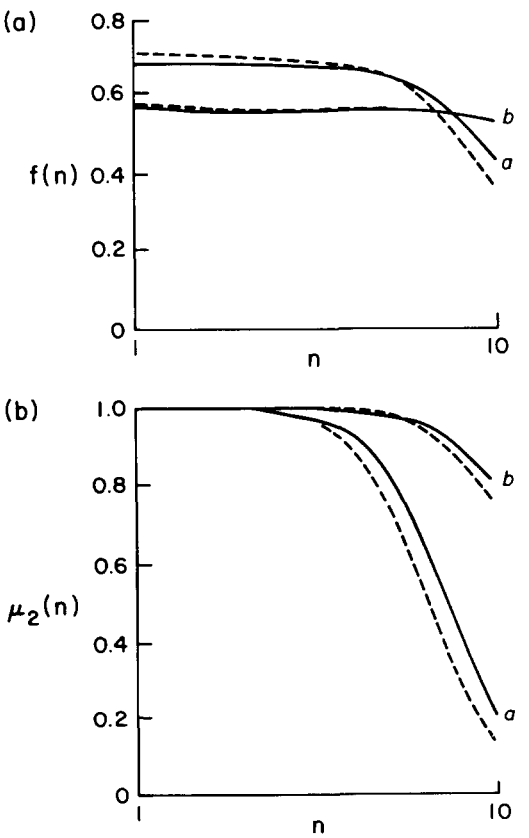

Fig. 8. Comparison of two-pest class model with age dependence and approximating ordinary differential equations. Panel (a) shows crop yields, panel (b) shows fraction of susceptible pests. Curves (a) correspond to the age independent case (———) and fitted ODE Model (- - - - - -). Curves (b) correspond to $\omega(a) = \omega_0 e^{-ma}$ (———) and fitted ODE model (- - - - - -).

differential equation solver. Curves "b" correspond to the case of $\omega(a)$ decreasing with a (eq. 25); the curves for the case of $\omega(a)$ increasing with a (eq. 26) are qualitatively similar to curves "b" and are not shown.

As may be expected, the initial yield in the age-dependent case is lower than that of the age-independent case. This is because if a portion of the genetically susceptible population is effectively resistant, then the effective intensity of the pesticide is reduced. Corresponding to this effective reduction in intensity is an increase in crop yield in later years. A more interesting observation may be made by comparing curve b in Fig. 8 with the curve for $\eta = 1.5$ in Fig. 2. The reduction in the initial season's crop yield in both cases is roughly the same. The increase in resistance is much slower, however, in the age-dependent case with $\eta = 2$ (Fig. 8). The reason is that in the latter case a portion of the genetically susceptible population is effectively resistant, so that the selection pressure on the genetically susceptible population is reduced.

In the preceding paragraph it was seen that each class may be effectively subdivided into two groups, young and old, corresponding to the age of the colony. This motivates a simple approximation for the case of age-dependent susceptibility. The total population of each class is divided into two categories: young and adult. The differential equations for the dynamics of the pest population are assumed to be

$$\begin{aligned}
\frac{dy_i}{dt} &= \rho_i a_i - \varphi y_i - \sigma_i(t) y_i + \mu_i I \\
\frac{da_i}{dt} &= \varphi y_i - \nu_i(t) a_i \\
y_i(0) &= a_i(0) = 0, \; i = 1, 2
\end{aligned} \tag{28}$$

The essence of this approximation is that young are generated at a rate proportional to the population of adults, and become adults at a rate proportional to the population of young. Young and adults die at rates $\sigma_i(t)$ and $\nu_i(t)$, respectively. Equations 28 may be derived rigorously from eqs. 21 by assuming that young are converted to adults at an age that follows a Poisson distribution (MacDonald, 1978). Another view is to regard eqs. 28 as an ad hoc approximation to eq. 21 and choose the parameters of (28) to make the solutions of this system behave similarly to those of (21).

The death rate functions in eqs. 28 were chosen to resemble the functions of eqs. 21. Specifically,

$$\begin{aligned}
\sigma_i(t) &= \left[\frac{\omega_y s(t)}{e_i + s(t)} + \bar{\sigma}_i \right] \\
\nu_i &= \left[\frac{\omega_a s(t)}{e_i + s(t)} + \bar{\nu}_i \right]
\end{aligned} \tag{29}$$

where the e_i and $s(t)$ are as in eq. 4.8, and $\bar{\sigma}_i$, $\bar{\nu}_i$ are the death rates due to natural mortality. The dashed lines in Fig. 8 show the solutions of the approximate equations. The parameter values are $\rho_1 = 0.22$, $\rho_2 = 0.231$ (i.e., $1.05 \times \rho_1$), $\varphi = 3$, $\omega_y = 4.2$, $\omega_a = 4.2$ for curve a, $\omega_a = 2.6$ for curve b, $\bar{\sigma}_i = 0$, and $\bar{\nu}_i = 0$. The agreement between solutions of eqs. 28 and 21 is clearly satisfactory.

In addition to its obvious simplicity, the model of eq. 28 has an important conceptual advantage over that of eq. 21. This is that the variables $y_i(t)$ and $\mu_i(t)$ measure the total numbers of young and adult pests of class i in the field, rather than the pests in young and old colonies. Therefore one may drop the restriction of a single pesticide application per season.

CONCLUSIONS AND DISCUSSION

The main conclusions of the paper are these. First consider the model itself. There are a number of simplifying assumptions, such as spatial independence, linear dynamics, simplified temperature effects, genetics, and age structure, that we do not consider crucial for our purpose. While such assumptions would be important for a simulation model, the goal here is to develop models that will provide qualitative predictions and interpretations.

The first conclusion is that while increasing the intensity of pesticide application can increase the short term crop yields, proper timing of pesticide application is necessary to ensure maintenance of a susceptible population. As shown in Fig. 2a, increased intensity of application leads to uniformly higher yields over most of the time span. The declining yields near the time horizon are rendered less important by the effect of the discount rate. Even at very higher spray intensities, in which virtually all of the susceptibles in the field are killed, the combination of immigration of susceptibles after the spraying period and their slightly higher growth rate are sufficient to ensure that their population recovers. The importance of proper timing is apparent from the result shown in Fig. 4. Spraying too early allows the susceptibles sufficient time to recover, but sacrifices crop yield. Spraying too late is even worse since it, too, sacrifices yield, but also prevents susceptibles from recovering. Therefore, the manager should employ a timing strategy which uses proper timing to maximize crop yield, while minimizing buildup of susceptibility. In summary, one would introduce an ad hoc rule that "proper timing controls resistance buildup and proper intensity controls yield".

In Plant et al. (1983) dynamic programming is used to achieve an optimal timing strategy. The result displayed in Fig. 5, that the genetic model does not significantly affect the results, is useful in reducing the dimensionality of the dynamic programming problem. It is also noteworthy because it allows

an extreme simplification (from diploid to haploid genetics) without a serious modification of results. The main cause is the explosive growth of the resistant population during the later seasons. This growth is so rapid that the other two classes are swamped as the susceptibles decline to a level that cannot maintain a significant heterozygous population.

Figure 6 shows that changing the functional form of the immigration function $I(t)$ affects the behavior of the model quantitatively but not qualitatively. Because of the importance of proper timing of pesticide application however, the form of $I(t)$ becomes a factor in management decisions. All the parameters of the model have a stochastic component, but the form of $I(t)$ is in practice known with the least certainty.

The main operational conclusion of Section 4 is that the use of a pesticide which primarily affects a single age class of the pest population can significantly reduce the buildup of resistance in the pest population. From the perspective of development of the model, the principal conclusion is that a simple two-age class model can effectively represent the more complex model that uses a continuous age dependence. This representation will be used in subsequent papers.

ACKNOWLEDGEMENTS

For motivation and helpful comments we thank J. Carey. For other comments we thank G.P. Georghiou, T. Prout, M. Turelli, and C. Taylor. This research is supported in part by ONR contract NK00014-81-K-0030, NSF grant MCS-81-21659 to MM, NSF grant MCS-81-21413 to REP, and U.C. Davis OPER grant 4817 to both authors.

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