MODELLING INSECT POPULATIONS AFFECTED BY PESTICIDES WITH APPLICATION TO PESTICIDE EFFICACY TRIALS

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ABSTRACT


Few attempts have been made to model the dynamics of insect populations as affected by pesticides. We attempt to develop such a model by incorporating a pesticide effectiveness model into a sojourn time model for population dynamics. However, obtaining a workable model requires simplifying assumptions such as that insects rid themselves of the pesticide as they mature from one growth stage to the next. Using this assumption, a simple pesticide–population model is fitted to data from a pesticide efficacy trial. This fit is good, and agreement between parameter estimates from the two blocks in the study as well as agreement between some parameter estimates and external estimates of the same parameter suggest that the model may be valid. A pesticide–population model utilizing some of these parameter estimates is used to investigate the estimand of the modified Abbott’s formula. Because of the dependence of this formula on the phenological status of the populations, the routine use of a simple pesticide–population model in analysis of data from pesticide efficacy trials is discussed.

INTRODUCTION

With the recent development of procedures for modelling pesticide effectiveness (Schaalje, 1988), it is desirable to attempt to incorporate such
models into insect population models in order to study the dynamics of insect populations as affected by pesticide applications. Such composite models would be useful in developing optimal strategies for timing and selection of pesticide applications as well as in interpreting field experiments and monitoring studies involving pesticide applications. They might also be useful in evaluating and developing hypotheses regarding migration of insects into sprayed fields after some lag time and the appearance of post-spray peaks in insect numbers at times not predicted by phenological models in the absence of pesticides. Several models of populations affected by pesticides have been developed. Usually the assumption in such models is that the pesticide effect is a single instantaneous reduction in numbers (Curry and Feldman, 1987, p. 79), but some researchers have included effects lasting a few or several days. Wilkerson et al. (1983) used a table look-up procedure to supply daily mortalities due to pesticides. In all cases, the pesticide effects were set to zero on the 3rd day after pesticide application. Shaffer and Gold (1985) calculated the daily mortality due to pesticide in their model by using the predicted pesticide residue at that time. They evaluated dose–mortality relationships developed in the laboratory at the predicted residue levels to compute the daily mortalities due to the pesticide.

The purpose of this paper is to investigate theoretical and numerical issues associated with the linking of a pesticide effectiveness model as discussed by Schaalje (1988) to an insect population model. Data from a pesticide efficacy trial are used in model evaluation, and the model is then used to investigate properties of statistics commonly computed in the analysis of pesticide efficacy trials.

**POPULATION MODEL**

Models for insect populations have been reviewed by Curry and Feldman (1987) and Schaalje and van der Vaart (1989). For the purposes of this paper we employ “sojourn time” models (Gurney et al., 1986; Schaalje and van der Vaart, 1989) in which, given initial conditions $n_h(s, 0)$, the number of individuals in stage $h (h = 1, 2, \ldots, f)$ of sojourn time between $s$ and $s + ds$ at time $t$ is given by:

$$n_h(s, t) \, ds$$

where

$$\frac{\partial n_h(s, t)}{\partial t} + \frac{\partial n_h(s, t)}{\partial s} = v_h(s, t) - q_h(s, t) n_h(s, t) - w_h(s, t) n_h(s, t)$$

(1)
and

\[ v_{h+1}(0, t) = \begin{cases} \Phi_{h+1}(t) \int_0^\infty q_h(s, t) n_h(s, t) \, ds, & 0 < h < f \\ \int_0^\infty b(s, t) n_f(s, t) \, ds, & h = 0 \end{cases} \]  

in which \( v_h(s, t) \) is the rate of recruitment of individuals into stage \( h \), \( q_h(s, t) \) is the 'force of maturation' of individuals or the per-capita rate at which individuals become candidates for transition into stage \( h + 1 \) from stage \( h \) (for \( h = f \), \( q_f(s, t) = 0 \)), \( w_h(s, t) \) is the 'force of mortality' or hazard function (Curry and Feldman, 1987, p. 86), \( \Phi_{h+1}(t) \) is the probability of successfully making the transition from stage \( h \) to stage \( h + 1 \), and \( b(s, t) \) is the per-capita fecundity of individuals in stage \( f \). We have assumed that only those in stage \( f \) can reproduce but this can be modified easily.

The hazard function in (1) also appears in other types of models for insect populations such as the distributed delay model (Vansickle, 1977) and the forward Kolmogorov model (Weiss, 1968), both of which have been classified as development index models by Schaalje and van der Vaart (1989).

Note that in the above model, \( q_h(s, t) \) and \( w_h(s, t) \) are the "cause specific" (Prentice et al., 1978) or "crude" (Elandt-Johnson and Johnson, 1980) exit rates due to the competing exit processes of maturation to stage \( h + 1 \) and mortality.

The common use, in modelling insect development, of a time-invariant distribution of physiological ages (Gurney et al., 1986) at which maturation to the next stage takes place (Stinner et al., 1975; Curry and Feldman, 1987, p. 49–54) is covered by this model. If: (a) \( z \) denotes physiological age, (b) \( r(t) \) gives the strictly positive time-dependent (and thus possibly temperature-dependent) rate function, (c) \( g_h(z) \) and \( G_h(z) \) are the density and distribution functions, respectively, of the distribution of maturation ages on the physiological age scale, and (d) \( z = 0 \) when \( s = 0 \), then:

\[ q_h(s, t) = \frac{g_h[z(s, t)] r(t)}{1 - G_h[z(s, t)]} \]  

where

\[ z(s, t) = \int_{t-s}^t r(x) \, dx \]

In this paper we will assume that recruitment into stage \( h \) is comprised of advancement from lower stages or reproduction, but not immigration. Hence \( v_h(s, t) = 0 \) unless \( s = 0 \). Under these conditions, the solution to (1)
is given by:

\[
 n_h(s, t) = \begin{cases} 
 n_h(s - t, 0) \exp \left\{ - \int_0^t [q_h(s - t + \tau, \tau) + w_h(s - t + \tau, \tau)] \, d\tau \right\} & s > t \\
 v_h(0, t - s) \exp \left\{ - \int_0^s [q_h(\sigma, t - s + \sigma) + w_h(\sigma, t - s + \sigma)] \, d\sigma \right\} & s < t 
\end{cases}
\]  

(5)

and the total number of individuals in growth stage \( h \) at time \( t \) is:

\[
 N_h(t) = \int_0^\infty n_h(s, t) \, ds
\]  

(6)

Note that sojourn time models are population models and do not directly address properties of individuals.

PESTICIDE EFFECTIVENESS MODEL

We will use the pesticide effectiveness model formulation discussed by Schaalje (1988). This formulation in general consists of the differential equations:

\[
 \frac{dc}{dt} = f(c, t)
\]  

(7)

and

\[
 \frac{dx}{dt} = g(c, x, t) - h(x, t)
\]  

(8)

and the functional equation:

\[
 \lambda(t) = K \left\{ [x(\tau), \tau \in (0, t)], t \right\}
\]  

(9)

where \( c \) is the concentration of pesticide in the environment, \( x \) is the amount of pesticide retained by an individual, \( t \) is time, \( \lambda \) is the hazard function which, when multiplied by \( dt \), gives the conditional probability of dying in \( (t, t + dt) \) given that the individual has survived to time \( t \), \( f \), \( g \), and \( h \) are appropriate deposition and degradation, intake, and clearance functions, and \( K \) is a functional relating the hazard function to the pattern of pesticide retention by the individual.

For example, in the simple case of first-order degradation of the pesticide in the environment, intake proportional to the current environmental concentration, first-order clearance of the pesticide from the individual, and a
linear relationship between the amount of pesticide currently retained by the individual and the current hazard, the hazard function at time $t$ is:

$$
\lambda(t) = \lambda_0(t) + \lambda_1 \left( x_0 \exp(-\beta t) + \frac{\mu c_0}{\beta - \alpha} \left[ \exp(-\alpha t) - \exp(-\beta t) \right] \right)
$$

(10)

where $\lambda_0(t)$ is the hazard function corresponding to mortality due to causes other than the pesticide, $\lambda_1$ is the coefficient of a linear term relating the hazard of dying at a specific time to the amount of pesticide retained by the insect at that time, $x_0$ is the amount of pesticide taken in by the insect at the time of application, $c_0$ is the initial concentration of pesticide in the environment, and $\mu$, $\alpha$, and $\beta$ are instantaneous rate coefficients for degradation of the pesticide in the environment, intake of the pesticide by the insect, and clearance of the pesticide from the insect, respectively. The two terms of equation (10) will be referred to as the non-pesticide-specific hazard rate and the pesticide-specific hazard rate, respectively. Schaalje (1988) noted that the non-pesticide-specific hazard rate is not necessarily equal to the hazard rate that would apply in the absence of the pesticide. Often $\lambda_0(t)$ is taken to be constant throughout an insect’s lifetime (Kemp-ton, 1979) and if so, the cumulative distribution function for the death times is given by:

$$
P(t) = 1 - \exp\left\{ -\lambda_0 t - \lambda_1 x_0 \left[ \frac{1 - \exp(-\beta t)}{\beta} \right] \right\} \\
- \frac{\lambda_1 \mu c_0}{\beta - \alpha} \left[ \frac{1 - \exp(-\alpha t)}{\alpha} - \frac{1 - \exp(-\beta t)}{\beta} \right]
$$

(11)

Although the environmental deposition and degradation parameters are not stage-specific, it is known that the toxicities of many chemical pesticides vary among growth stages (Wells et al., 1983). Hence in the above example, the parameters $\lambda_0$, $x_0$, $\lambda_1$, $\mu$ and $\beta$ may be stage-specific. We will express stage specificity by attaching subscripts, e.g., $\lambda_{h,0}$.

### LINKING THE MODELS

Because one component of the population model (1) is the hazard function $w_h(s, t)$, and the pesticide effectiveness model (9) is explicitly constructed as a model for the hazard function $\lambda_h(t)$, it would seem that all we need to do to link the models is replace $w_h(s, t)$ with $\lambda_h(t)$. On closer examination, however, several issues have to be dealt with in relating $w_h(s, t)$ to $\lambda_h(t)$.

First, an apparently convenient feature of $\lambda_n(t)$ is that it is the sum of terms for both mortality due to the pesticide and mortality due to other causes. However, it must be noted that the rate of mortality due to other
causes may itself be different in the present of a pesticide than in its absence. Even the rate of maturation may change in the presence of a pesticide. Hence maturation- and non-pesticide-specific hazard rates which are known to be appropriate in the absence of a pesticide may not apply when a pesticide is used. In addition to killing insects, the pesticide may directly slow their rate of development (Johnson and Pavlikova, 1986) or weaken them and make them more susceptible to death due to disease or predators. Indirect effects of the pesticide on the maturation- and non-pesticide-specific hazard rates of the population may also occur. For example, the weaker members of the population, which might be characterized by, say, long development periods and high susceptibility to death from sources other than pesticide, may also be selectively killed by the pesticide (Kuenen, 1957). With a lower proportion of these weak individuals left in the population, the maturation- and non-pesticide-specific hazard rates would be altered. In earlier models, most researchers simply added the mortality rate due to the pesticide to the ‘natural’ mortality and the ‘natural’ maturation rate, and implicitly made the assumption of mutual independence of the latent exit times due to maturation, non-pesticide mortality, and pesticide-induced mortality (Wilkerson et al., 1983; Shaffer and Gold, 1985; Curry and Feldman, 1987, p. 96). Ideas from proportional hazards models (Kalbfleisch and Prentice, 1980) might be useful in “fixing up” the maturation- and non-pesticide-specific hazard rates to accommodate pesticide effects.

Second, it must be noted that whereas the sojourn time model is a population model with no facility for dealing with individual characteristics, the pesticide effectiveness model \( \lambda_h(t) \) as formulated by Schaalje (1988) applies to one individual or possibly a cohort of (assumed) identical individuals. It has not been formulated to allow for dependence on the varying sojourn times of the individuals in a growth stage, nor has it been formulated to allow for random variability between individuals in the deposition amounts or rates of degradation, intake, or clearance. Because a population usually consists of individuals of varying sojourn times existing in a nonhomogeneous environment to which the pesticide has been applied nonuniformly, either the pesticide effectiveness model has to be modified or some assumptions have to be made. We could rely on the fact that insect are mobile to partially justify assumptions that all individuals in a growth stage receive the same initial dose of pesticide, and thereafter all take in and clear the pesticide at the same rates. Also, because the hazard function for the population is, in some sense, the average of the individual hazard functions, we may have some reason to hope that it would be approximately equal to the individual hazard function evaluated at the averages of the individual parameter values.
As for variability in sojourn times, it would be a simple matter to allow dependence on sojourn time for the non-pesticide-specific term of the hazard function, $\lambda_{h,0}(t)$. Natural mortality in populations is often modelled using hazard rates corresponding to Weibull or gamma distributions of (sojourn) times before death, both of which imply hazard rates dependent upon sojourn time. Even if we assume the parameters of the pesticide-specific term of the hazard function, $\lambda_{h,1}(t)$, to be independent of sojourn time, the term will still have to be made dependent on sojourn time to account for the fact that insects which are recruited into the growth stage after application of the pesticide are exposed to the pesticide (as members of that growth stage) for a shorter time than those that were in the growth stage at the time of application. Also, for the new recruits, such things as the initial concentration of pesticide in the environment ($c_0$ in equation 10) and the initial dose of pesticide received by the insects ($x_0$ in equation 10) will have to be changed in $\lambda_{h,1}(t)$ because the pesticide will have degraded somewhat before their first exposure to it in their new growth stage. Thus, for individuals of sojourn time $s$ at the current time $t$, who were present in stage $h$ at the time of application $t_a$, we have:

$$s > t - t_a$$

and

$$w_h(s, t) = \lambda_{h,0}(s, t) + \lambda_{h,1}(t - t_a)$$

(12)

where $\lambda_{h,0}$ gives the non-pesticide-specific hazard rate, and $\lambda_{h,1}(t - t_a)$ is given by equation (9) or any of its specializations. Adapting equation (10), this becomes:

$$w_h(s, t) = \lambda_{h,0}(s, t) + \lambda_{h,1}\left[x_0 \exp[-\beta(t-t_a)]ight]$$

$$+ \frac{\mu c_0}{\beta - \alpha} \left[\exp[-\alpha(t-t_a)] - \exp[-\beta(t-t_a)]\right]$$

(13)

For individuals who enter stage $h$ after application of the pesticide, if we can, for example, assume that they do not carry any pesticide with them as they mature from the previous stage, we have:

$$s < t - t_a$$

and

$$w_h(s, t) = \lambda_{h,0}(s, t) + \lambda_{h,1}'(s)$$

(14)

where $\lambda_{h,1}'(.)$ is equal to $\lambda_{h,1}(.)$ in equation (12) except that it has been modified to eliminate the effects of initial absorption amounts – e.g., $x_0$ in equation (10) is set to zero – and to allow for some degradation of the
pesticide before the individual's first exposure to it. Applying these ideas to equation (10) we obtain:

\[ w_h(s, t) = \lambda_{h,0}(s, t) + \lambda_{h,1}\left( \frac{\mu c_0}{\beta - \alpha} \exp[-\alpha(t - t_a)]\left[\exp(-\alpha s) - \exp(-\beta s)\right]\right) \]  

(15)

For individuals who mature from one stage to the next during the period when the pesticide is effective, and possibly carry some of the pesticide with them as they make the stage transition, several additional difficulties are encountered in relating \( w_h(s, t) \) to \( \lambda_h(t) \). These difficulties stem from the fact that \( w_h(s, t) \) is the hazard rate for the cohort of individuals who enter stage \( h \) at time \( t - s \). Because the population model allows variability in the lengths of time individuals spend in any growth stage, the cohort entering stage \( h \) at time \( t - s \) includes individuals who have been exposed to the pesticide in the previous stage(s) for varying lengths of time. Therefore, even if we can assume that all individuals have the same absorption and clearance rates, we still cannot assume that those entering a growth stage at one point in time have the same levels of retained pesticide. The appropriate pesticide-specific hazard rate will be a complicated weighted average (with weights that vary in time as the number of individuals in the cohort changes) of the hazard rates appropriate for the various amounts of pesticide carried over from the previous stage by members of the cohort. We could possibly work with subcohorts instead of cohorts, but in the continuous time situation, we are immediately faced with an infinite number of subcohorts, all of which have their own hazard function.

Thus, in practice, the sojourn time model will only be useful in connection with a pesticide effectiveness model if special conditions are met. Two such circumstances that lead to particularly simple formulations are:

1. That in which insects purge themselves of all pesticide as they mature from one growth stage to the next. Hence, no matter how long they have been exposed to the pesticide in the previous stage, all insects entering any growth stage at a particular time would be identical with respect to their response to the pesticide effects in the current stage. In some insect species, this conditions may be approximately met. As insects of some species approach their moult to a new stage, they stop feeding (hence also ingesting pesticide) for 12 or more hours prior to the moult. Yet during this period, metabolic clearance processes continue to operate. Also, the new cuticle develops for some period prior to the moult and there is thus a double barrier to surface absorption of the pesticide. Any pesticide residing in the old cuticle will obviously be eliminated during the moult.
(2) That in which all insects within a growth stage, even the most recent recruits, are subject to the same pesticide effect regardless of their sojourn time in the stage. This condition would be met for a population in which all growth stages are affected equally by the pesticide, in which there is no recruitment into the population after application of the pesticide, and in which the insects carry their full load of pesticide with them as they mature from one stage to the next. The condition may be approximately met for situations in which the half-life of the pesticide is short relative to the sojourn times in the various stages, and the variability in stage-specific pesticide effects is small. A variant of this situation is that in which insects do not lose any of the pesticide as they mature from one stage to the next, and they all were in the previous stage from the time of application of the pesticide up to the time of their maturation. This situation results in the relevant equations being much more complex (compare equation 10 with equation 16 to follow).

Even if neither of the above circumstances are appropriate, models developed assuming them may still be useful. In a sense, the two conditions can be viewed as providing upper and lower bounds on $w_h(s, t)$. At any particular time, the true time- and sojourn time-specific hazard rate will be greater than that predicted by circumstance (1) and, although not guaranteed, is likely to be less than that predicted by circumstance (2) above.

There will be some populations for which neither of the circumstances under which the sojourn time model has a simple form is appropriate, and for which more than just boundary information is desired. In such situations, the use of the sojourn time formulation of the population model would require one to keep track of individual life histories of members of the population in order to compute correct values of the hazard functions. However, if all individual life histories were recorded, no model would be needed for population dynamics because all information on the population would be known.

Although the computing requirements would be extensive, the strategy of modelling a population by keeping track of individual life histories is in principle possible. As pointed out above, the sojourn time formulation would be superfluous in such a procedure, but the pesticide effectiveness model might still be useful in implementing the strategy. If we had a rule for how the pesticide carried over as an individual insect matured from one growth stage to the next, and if we could specify the time at which it entered the stage, the model could be used to predict the hazard function for the insect at all times. For example, if equation (10) applied for every growth stage and if insects were known to carry all of the pesticide with them as they matured from one growth stage to the next. then for an insect that was
in stage $h$ at the time of application of the pesticide ($t_a$) but matured to stage $h+1$ at $t_m$ and remained alive in stage $h$ at least until $t$:

$$
\lambda_{h+1}(t) = \lambda_{h+1,0}(t) + \lambda_{h+1,1} \left\{ x_0 \exp\left[ -\beta_h(t_m - t_a) \right] \exp\left[ -\beta_{h+1}(t - t_m) \right] 
+ \frac{\mu_h c_0}{\beta_h - \alpha} \left( \exp\left[ -\alpha(t_m - t_a) \right] - \exp\left[ -\beta_h(t_m - t_a) \right] \right) 
\times \exp\left[ -\beta_{h+1}(t - t_m) \right] + \frac{\mu_{h+1} c_0}{\beta_{h+1} - \alpha} \left( \exp\left[ -\alpha(t - t_a) \right] \right) 
\times \left( \exp\left[ -\alpha(t_m - t_m) \right] - \exp\left[ -\beta_{h+1}(t - t_m) \right] \right) \right\}
$$

Equation (16) could be readily extended to individuals which had changed stages more than once between $t_a$ and $t$. From these equations for the hazard functions, the distribution functions for death times in each stage could be derived. Assuming that we also knew the maturation-specific hazard rates which applied in the presence of the pesticide, we could derive the probability laws associated with the stochastic process of entering the population and advancing through the growth stages or dying because of natural or pesticide-related effects. These probability laws could be used to generate the individual life histories.

We will concern ourselves herein only with those simple situations in which a sojourn time population model is appropriate for use with a pesticide effectiveness model.

**NUMERICAL CONSIDERATIONS**

It is well known that the Leslie (1945) model of dynamics of an age-structured population is the discrete version of the Von Foerster (1959) equation both in the sense that the latter is the limit of the former as the length of the time step goes to zero (Schaalje and van der Vaart, 1989), and in that the former can be obtained from the latter by replacing the derivatives with finite differences (Oster, 1978; McFarland and van der Vaart, 1985). The close relationship between discrete and continuous population models has also been investigated by Goodman (1967) and Keyfitz (1967), who demonstrated that the discrete formulation would give approximately the same results as the continuous formulation if the age steps were small enough. In related work, Mode and Pickens (1988) used discrete formulations of semi-Markov processes to approximate those in continuous time. Since the sojourn time model considered in this paper is of the form of a Von Foerster equation (Schaalje and van der Vaart, 1989) for each growth stage, it seems reasonable to numerically approximate our models of insect populations affected by pesticide by a sequence of generalized Leslie models with a small
The discrete analog of equation (1) is:
\[
n_h(s + 1, t + 1) = n_h(s, t)[1 - p_h(s, t)][1 - m_h(s, t)] + u_h(s + 1, t + 1)
\]  
(17)

where \( n_h(s, t) \) is the number of individuals in stage \( h \) of sojourn time \( s \) at time \( t \), \( p_h(s, t) \) is the proportion of individuals of sojourn time \( s \) at time \( t \) who mature to stage \( h + 1 \) before time \( t + 1 \) given that they survive from \( t \) to \( t + 1 \), \( m_h(s, t) \) is the proportion of individuals in stage \( h \) of sojourn time \( s \) at time \( t \) who die before \( t + 1 \), and \( u_h(s, t) \) is the number of individuals of sojourn time \( s \) at time \( t \) who enter stage \( h \) (via birth, maturation, or immigration) between \( t - 1 \) and \( t \).

We can approximate equation (1) with equation (17) by setting:
\[
\begin{align*}
    p_h(s, t) &= 1 - \exp\left[-\int_t^{t+k} q_h(s - t - x, x) \, dx\right] \\
    m_h(s, t) &= 1 - \exp\left[-\int_t^{t+k} w_h(s - t - x, x) \, dx\right] \\
    u_h(s, t) &= \int_t^{t+k} v_h(s - t - x, x) \, dx
\end{align*}
\]  
(18) (19) (20)

where \( k \) is the size of the time step. Plant (1986) argued that if the continuous model were correct, the above formulae would lead to inaccuracies in predictions of population dynamics. He suggested other formulae which he claimed would be better than equations (18), (19) and (20) when \( k \) had to be large. For our purposes, it is simpler and more straightforward to make the time step small enough that the above formulae are adequate.

For illustration, we use the discrete approximation to model a hypothetical grasshopper population affected by a hypothetical pesticide. The grasshopper is assumed to have two immature growth stages plus an adult growth stage. The force of maturation, \( q_h(s, t) \) is assumed to be of the form \( g_h(s)/[1 - G_h(s)] \), where \( g_h(s) \) is a gamma density. Parameter values for the gamma distributions are obtained from Kempton (1979), except that our first growth stage corresponds to Kempton’s first and second growth stages, our second to his third and fourth, etc. The non-pesticide-specific hazards are taken as constant over time and sojourn time for each stage and are also
Fig. 1. Numbers of insects in growth stages 1 (solid line), 2 (dotted line), and 3 (dashes) versus time for a hypothetical population of insects affected by a pesticide. The model has been discretized for computer implementation using different sizes of time steps. Figs. 1a, b, c, d, and e give the results for time steps of size 6, 3, 2, 1, and 0.5 days, respectively. Fig. 1f uses a time step of 1 day to show the dynamics of the population without the pesticide effects.

Based on those of Kempton (1979) except that natural mortality rates in the last two growth stages are assumed to be slightly lower than that in the first stage. The pesticide-specific hazard for each stage is formed using equations (13) and (15), with parameter values chosen in rough accordance with experimental indications. We assume the pesticide to have a 5-day half-life in the environment, and we assume the half-life of metabolic clearance to be 1 day in adult and 2 days in the immature stages. We assume that insects enter each growth stage free of residual pesticide from the previous growth stages.

As \( k \) (the size of the time-step) becomes smaller, the curves giving the number of individuals in each of the growth stages converge (Fig. 1a–1e). If the size of the time step is too small, roundoff errors become a problem as can be seen in the apparent (but impossible) increase in the number of
Fitting the model to pesticide efficacy trial data

In order to demonstrate that the models developed in this paper are at least consistent with real data, we fitted a simple model of this type to data from an efficacy trial of the pesticide deltamethrin, aerially applied to rangeland for control of grasshoppers. The trial was carried out near Claresholm, Alberta, Canada in 1983, and some of the results as well as details of the experiment have been reported elsewhere (Johnson et al., 1986). The experiment consisted of two blocks, each of which was composed

Table 1

Observed and predicted numbers of grasshoppers (*Melanoplus infantilis*) taken in sweep-net samples during a test of the insecticide deltamethrin on rangeland in southern Alberta in 1983

<table>
<thead>
<tr>
<th>Block</th>
<th>Plot&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Days&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Number in instars</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1–2</td>
<td>3</td>
</tr>
<tr>
<td>1</td>
<td>C</td>
<td>-1</td>
<td>41 (34.81) 92 (66.72) 42 (51.29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>10 (23.68) 48 (55.04) 74 (68.60)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>23 (12.95) 26 (36.70) 105 (88.76)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>5 (5.61) 10 (18.62) 90 (100.51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td>3 (1.22) 6 (4.70) 95 (89.99)</td>
</tr>
<tr>
<td></td>
<td>T</td>
<td>-1</td>
<td>120 (95.51) 177 (183.06) 116 (140.74)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>8 (7.59) 19 (16.98) 17 (16.05)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>3 (1.93) 4 (8.67) 7 (14.98)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td>1 (0.33) 2 (1.46) 23 (16.77)</td>
</tr>
<tr>
<td>2</td>
<td>C</td>
<td>-1</td>
<td>15 (15.88) 27 (22.59) 33 (27.23)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>9 (12.15) 14 (20.53) 27 (30.85)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>7 (7.89) 18 (16.92) 34 (35.40)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>3 (4.19) 8 (12.02) 52 (39.25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td>4 (1.21) 11 (5.42) 31 (39.15)</td>
</tr>
<tr>
<td></td>
<td>T</td>
<td>-1</td>
<td>12 (19.80) 39 (28.41) 34 (34.64)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>1 (4.80) 3 (5.90) 15 (13.17)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>4 (2.20) 1 (5.04) 9 (11.67)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td>2 (0.60) 3 (2.67) 22 (12.06)</td>
</tr>
</tbody>
</table>

Predicted numbers (in parentheses) are based on a pesticide–population model developed in the present paper and fitted using a modified form of maximum likelihood

<sup>a</sup> C, untreated control; T, treated plot.

<sup>b</sup> Number of days after application of deltamethrin.
of a sprayed field and a control field. We used the numbers of individuals of the dominant species *Melanoplus infantilis* Scudder taken in sweep-net samples from the control and sprayed fields 1 day before, and 1, 4, 8, and 15 days after, application of the pesticide (Table 1). In order for the numbers of insects to be large enough to analyse, and to reduce the number of parameters to be fitted in the model, the numbers of grasshoppers in instars 1 and 2 were pooled as were the numbers in instars 4 and 5. Only a very few adult grasshoppers were observed during the course of the experiment, and these were ignored. We did not use the data from the treated plots 1 day after application of the pesticide because Johnson et al. (1986), comparing the results from two methods of sampling, concluded that sweep net samples are negatively biased immediately after pesticide application because of transient sublethal effects of the pesticide on insect behavior.

The insect population model is developed under the assumptions of:
- Constant 'natural' death rates for all individuals, i.e., in equation (1), \( w_h(s, t) = c \), say, for all \( h \), \( s \), and \( t \).
- No mortality during stage transitions, i.e., in equation (2), \( \Phi_h = 1 \) for all \( h \).
- Maturation rates which were time-invariant but sojourn time-dependent and given by hazard functions corresponding to independent gamma distributions, i.e., for all \( t \) and each \( h \):

\[
q_h(s, t) = \frac{g_h(s; r_h, k_h)}{1 - G_h(s; r_h, k_h)}
\]

where \( g_h(s; r_h, k_h) \) is the density function for a gamma distribution over \( s \) with parameters \( r_h \) and \( k_h \).
- Gamma-distributed recruitment times for insects entering the population, i.e., \( v_1(0, t) = N g_0(t; r_0, k_0) \) where \( g_0 \) is a gamma density as above, and \( N \) is the total number of individuals entering the first growth stage of the population.

The pesticide effectiveness model used is that described by equations (10) and (11). To reduce the number of parameters to be fitted in the model, we assume that all parameters except \( \lambda_1 \) are constant among the growth stages. In linking the population model and the pesticide effectiveness model, we assume that all insects become free of the pesticide as they mature from one stage to the next. We also assume that the rates of maturation and natural mortality are unaffected by the pesticide. We have no way of knowing how long after the grasshoppers began emerging the pesticide was applied, so we arbitrarily choose the ballpark figure of 48 days after the beginning of emergence as the date of application of the pesticide. Similarly, Read and
Fig. 2. Observed (growth stage number as symbol) and predicted numbers of grasshoppers (*Melanoplus infantilis*) of growth stages 1 (solid line), 2 (dashes), and 3 (dotted line) versus time for (a) the control and (b) treated populations of block 1 of a pesticide efficacy trial of deltamethrin conducted near Claresholm, Alberta in 1983. The pesticide was applied on day 48 of the time scale. See text for a description of the model and the fitting procedure.

Ashford (1968) and Kempton (1979) arbitrarily chose reasonable starting dates in fitting their population models to data.

The fitting of the model to the data (Figs. 2 and 3) is done using a slightly modified version of maximum likelihood. Even though the instar counts at a single point in time were taken from the same sweep net sample, we assume that all insect counts are independently distributed as Poisson random variables with mean given by the pesticide–population model, and that the various instars have the same catchability. The maximum likelihood estimates of the parameters of the model are then those which maximize the log-likelihood:

\[
L = \sum_i \sum_j \sum_k N_{ijk} \log(\hat{N}_{ijk} - \tilde{N}_{ijk}) - \text{constant}
\]  

(21)

where \(N_{ijk}\) is the observed number of individuals in the \(i\)th growth stage at the \(j\)th observation time for the \(k\)th plot, and \(\hat{N}_{ijk}\) is the corresponding...
Fig. 3. Observed (growth stage number as symbol) and predicted numbers of grasshoppers (*Melanoplus infantilis*) of growth stages 1 (solid line), 2 (dashes), and 3 (dotted line) versus time for (a) the control and (b) treated populations of block 2 of a pesticide efficacy trial of deltamethrin conducted near Claresholm, Alberta in 1983. The pesticide was applied on day 48 of the time scale. See text for a description of the model and the fitting procedure.

predicted value based on the pesticide-population model. The procedure is modified in that the likelihood is maximized first for each control plot separately to get estimates of the parameters of the population model for each block. Then, taking these parameter estimates as truth, the parameters of the pesticide model are fit for each treated plot. This procedure is adopted for computational efficiency, but it makes sense for two other reasons:

(1) There is no information about the pesticide model parameters in the control plot data, and there is very little information about the population model parameters in the treated plot data.

(2) The underlying rationale for the experimental design is that the control plot should tell what the population would have done in the absence of the pesticide treatment. The sequential method of analysis described above conforms to this rationale.
TABLE 2
Parameter estimates and goodness-of-fit statistics for the insect population model fitted to the control plot data of Table 1

<table>
<thead>
<tr>
<th>Population model parameter</th>
<th>Estimates</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>185.749</td>
<td>129.645</td>
<td></td>
</tr>
<tr>
<td>$r_0$</td>
<td>4.322</td>
<td>0.417</td>
<td></td>
</tr>
<tr>
<td>$r_1$</td>
<td>0.232</td>
<td>0.395</td>
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<tr>
<td>$r_2$</td>
<td>1.648</td>
<td>0.493</td>
<td></td>
</tr>
<tr>
<td>$r_3$</td>
<td>0.143</td>
<td>0.413</td>
<td></td>
</tr>
<tr>
<td>$k_0$</td>
<td>158.189</td>
<td>1.613</td>
<td></td>
</tr>
<tr>
<td>$k_1$</td>
<td>1.809</td>
<td>14.875</td>
<td></td>
</tr>
<tr>
<td>$k_2$</td>
<td>12.979</td>
<td>5.090</td>
<td></td>
</tr>
<tr>
<td>$k_3$</td>
<td>3.784</td>
<td>12.339</td>
<td></td>
</tr>
<tr>
<td>Mean_0</td>
<td>36.600</td>
<td>3.870</td>
<td></td>
</tr>
<tr>
<td>Mean_1</td>
<td>7.787</td>
<td>37.661</td>
<td></td>
</tr>
<tr>
<td>Mean_2</td>
<td>7.874</td>
<td>10.333</td>
<td></td>
</tr>
<tr>
<td>Mean_3</td>
<td>26.452</td>
<td>29.893</td>
<td></td>
</tr>
<tr>
<td>SD_0</td>
<td>2.910</td>
<td>3.048</td>
<td></td>
</tr>
<tr>
<td>SD_1</td>
<td>5.789</td>
<td>9.765</td>
<td></td>
</tr>
<tr>
<td>SD_2</td>
<td>2.186</td>
<td>4.580</td>
<td></td>
</tr>
<tr>
<td>SD_3</td>
<td>13.599</td>
<td>8.510</td>
<td></td>
</tr>
<tr>
<td>$\lambda_0$</td>
<td>0.016185</td>
<td>0.015198</td>
<td></td>
</tr>
<tr>
<td>$\chi^2$</td>
<td>43.876</td>
<td>25.427</td>
<td></td>
</tr>
</tbody>
</table>

$^a$ $r_i$ and $k_j$ are parameters corresponding to the development time distributions in the various stages. $\lambda_0$ is the mortality parameter for all growth stages, $\text{mean}_i = k_i / r_i$, $\text{sd}_j = (k_j)^{1/2} / r_j$, and $\chi^2$ is the Pearson goodness-of-fit statistic. See text for details.

Maximization is carried out using the IMSL subroutine BCONF (IMSL, 1987) which uses a modified Newton iteration method, based on finite differences instead of analytical derivatives. The surface of the likelihood function must have several local maxima because, in practice, a combination of BCONF runs and graphical assessment of the fit are required to arrive at the approximate global maxima and parameter estimates.

The population model parameter estimates from each block do not agree well (Table 2) but even without a model, the data seem to indicate that at the start of the experiment, the grasshopper populations in block 2 were further advanced phenologically than those in block 1 (Figs. 2a, 3a). Because most of the recruitment and first instar development had taken place before the start of the experiment, the problem of estimating recruitment and first growth stage parameters is related to the notoriously difficult problem of backward projection. Hence, discrepancies in these parameters are not surprising. However, the estimated means and standard deviations of the
gamma distributions of the latent insect development times for instars 3 and 4–5 are somewhat similar between blocks.

Except for the parameter relating to deposition and uptake of the pesticide ($\mu c_0$) and the proportionality constant for the pesticide-specific hazard in the third growth stage ($\lambda_{1,3}$), there is good agreement between blocks in the pesticide model parameters (Table 3, Figs. 2b, 3b). Residue studies (Johnson et al., 1986) indicated that a higher concentration of pesticide was deposited on both plots of block 1 than on block 2 (78.6% or target amount for block 1 vs. 67.8% for block 2), and our estimates of $\mu c_0$ for the blocks agree with this ranking. The time required for 50% of deposited deltamethrin to degrade on forage in southern Alberta is 5.9 days for the isomer reported to be most active against insects (Hill and Johnson, 1987). If our pesticide-population model is approximately correct, the degradation half-life in the current study is between 2 and 3 days (Table 3). The lower half-life estimate obtained here may be due to random error, may be an indication of a problem in the model, or may suggest the intriguing possibility that from the insect's point of view, there are non-chemical (possibly behavioral) components also causing a decline in the efficacy of deltamethrin.

We would like to somehow investigate the difference between the two blocks in estimated pesticide effectiveness due to the combination of all of the pesticide effectiveness parameters. To carry out this comparison, we insert the parameter estimates for each growth stage of each block into
Fig. 4. Adjusted cumulative pesticide efficacy \( (P) \) curves (see text for definition) for growth stages 1 (solid line), 2 (dashes) and 3 (dotted line) computed using parameter estimates from block 1 (Fig. 4a) and block 2 (Fig. 4b) of a pesticide efficacy trial of deltamethrin conducted near Claresholm, Alberta in 1983.

equation (11), but to free the comparison of differences not related to the pesticide we set \( \lambda_0 \) equal to zero for each block (Fig. 4). We refer to the resulting functions as the adjusted cumulative pesticide effectiveness curves. An examination of the two sets of curves (Fig. 4) suggests that insects in the lowest instars (1–2) are less affected by the pesticide than those in the higher instars. Also, in block 1, insects of instar 3 appear to be subject to approximately the same pesticide effects at those of instars 4–5, with instars 4–5 possibly affected the most. In block 2, insects of instar 3 appear to be subject to a much greater effect than those in instars 4–5.

One would like to evaluate the fit of the models to the data, possibly by goodness-of-fit statistical tests. Because we assumed the observed counts of insects to be independent Poisson random variables in fitting the models, it would seem appropriate to use Pearson chi-square goodness-of-fit tests. These tests are significant for all plots (Tables 2 and 3), indicating significant disagreement between the model and the data. However, we are not discouraged because: (a) it is hard to see how the data could be fitted much
better by any other smooth model (Figs. 2 and 3), and (b) the assumed error structure may well be wrong. The counts taken from the same sweep-net samples are certainly correlated, extra-Poisson variability is quite likely, Taylor, 1987; Braner and Hairston, 1988), catchability may vary between instars, and errors arising from the fact that population growth is a stochastic process are ignored. In addition, the population and pesticide effectiveness models employed are relatively simple, and the dependence of insect phenology on temperature (Curry and Feldman, 1987, p. 38–47) is ignored. It is quite possible that the model is, in fact, adequate for many purposes (for example, to supply a model with realistic behavior for our simulations in a subsequent section) and the incorrect assumptions about the error structure may still allow good parameter estimates even though they may overwhelm the goodness-of-fit tests.

ESTIMAND OF THE MODIFIED ABBOTT'S FORMULA

Now that we have a model for an insect population affected by a pesticide (referred to hereafter as the pesticide–population model), some possible applications of the model become attractive. For example, we could now begin a study of the properties of some statistics often computed in connection with pesticide efficacy trials. Because of troubles they were having estimating the effects of pesticides on rapidly reproducing laboratory aphid populations, Sun and Shepard (1947) developed an “index of population reduction”:

\[ P_r = 1 - \left( \frac{N_{t,2}}{N_{t,1}} \right) / \left( \frac{N_{c,2}}{N_{c,1}} \right) \]  (22)

where \( N_{t,1} \) is the number of insects in the treated population at time 1 (before application of the pesticide), \( N_{t,2} \) is the number of insects in the treated population at time 2 (after application of the pesticide), \( N_{c,1} \) is the number of insects in the control (untreated) population at time 1, and \( N_{c,2} \) is the number of insects in the control population at time 2. They claimed that this index would give information on the simultaneous effects of the pesticide on mortality and reproduction while adjusting for initial differences in the sizes of the two populations.

Henderson and Tilton (1955) developed the same formula, calling it a “modified version” of the Abbott (1925) formula which adjusts for natural mortality in laboratory studies. The modification involved using numbers of insects in pre- and post-treatment samples in the formula as opposed to actual numbers of insects tested and surviving in a laboratory study.

Schaalje et al. (1986) discussed optimal estimation of \( p_r \) using data from field experiments arranged in randomized blocks. They also noted that analysis of numbers of insects in samples from field trials on insect popula-
tions by linear statistical models often leads to hypothesis testing and estimation of quantities closely related to $p_r$. For example, if the number of insects in the samples is log-transformed prior to linear model analysis, then a test of whether the drop in the logarithm of the population size, from the pretreatment sampling period to a posttreatment sampling period, is greater in the treated populations than in the control populations, is equivalent to a test of the hypothesis that $p_r$ is equal to 0 versus the alternative, that it is greater than zero. Thus an investigation of the properties of $p_r$ will also help in evaluating the usefulness of the above-discussed linear models for analyzing pesticide efficacy trials. Taylor (1987) argued for a need to examine current methods of analyzing data from pesticide efficacy trials.

Some (e.g., Kuenen, 1957) have questioned the validity of adjusting the pattern of mortality in a treated population which has two sources of death (pesticide-caused and other) using mortality observed in a control population with only one source of death (‘natural’ death). We will not discuss these questions further here except to note that these are important issues that need to be addressed. They are related to the general ideas of competing risks theory (Prentice et al., 1978; Gail, 1982). However, there is much to investigate about $p_r$ without getting into the issues of competing risks theory. For now we accept, and build into our models, the assumption that the probabilities of dying due to natural (non-pesticide) causes or of maturing from one stage to the next are the same in both the treated and untreated populations.

It is fairly clear how to estimate $p_r$ from pesticide efficacy trial data, but what is not clear is the nature of $p_r$ itself or in other words its “estimand” (Mosteller and Tukey, 1977). If we knew more about the estimand, we would know better whether or not and how to use $p_r$. We will use the pesticide-population model, both analytically and in a simulation study, to investigate this estimand.

In our analytical work, we employ equations (1) through (10) of this paper and use the symbols defined for those equations except that in our notation here we use the asterisk (*) superscript for symbols relating to the population to receive the treatment and use unsuperscripted symbols for the untreated (control) population. If the dynamics of growth stage ($h$) of each of two populations, with initial conditions of:

$$n_h(s, 0) = 0$$

and

$$n_h^*(s, 0) = 0 \text{ for all } s$$

are so similar that they involve identical rates of mortality and maturation,
and recruitment rates differ only by a constant \( c \), say:

\[
v_h^*(0, t) = cv_h(0, t) \quad \text{for all } t
\]

then at the very instant of pesticide application \( t_1 \), before the pesticide has had any effect, we have:

\[
n_h^*(s, t_1) = cn_h(s, t_1) \quad \text{for all } s
\]

and so (using equation 6):

\[
N_h^*(t_1) = cN_h(t_1)
\]

A short time later (at \( t_2 \), say), according to equations (5) and (6),

\[
N_h(t_2) = \int_{t_2}^{t_2} n_h(s, t_2, t_1) \exp\left\{ -\int_{t_2}^{t_1} q_h(s - t_2 + \tau, \tau) \\
+ w_h(s - t_2 + \tau, \tau) \right\} d\tau \right\} ds
\]

\[
+ \int_0^{t_2 - t_1} v_h(0, t_2 - s) \exp\left\{ -\int_0^s q_h(\sigma, t_2 - s + \sigma) \\
+ w_h(\sigma, t_2 - s + \sigma) \right\} d\sigma \right\} ds
\]  

The equation for \( N_h^*(t_2) \) has exactly the same form as equation (23) except that \( n_h(s, t_1) \) and \( v_h(0, t) \) are replaced by \( n_h^*(s, t_1) \) and \( v_h^*(0, t) \), respectively, and, on the assumption that only the rate of mortality is affected by the pesticide, \( w_h(s, t) \) will be replaced by a quantity \( w_h^*(s, t) \). The assumption that the non-pesticide-specific hazard rate is not affected by the pesticide leads to the relation:

\[
w_h^*(s, t) = w_h(s, t) + y_h^*(s, t)
\]  

where \( y_h^*(s, t) \) is the pesticide-specific hazard rate.

Now, we can write:

\[
p_r = 1 - \left[ \frac{N_h^*(t_2)}{N_h^*(t_1)} \right] / \left[ \frac{N_h(t_2)}{N_h(t_1)} \right]
\]

which under the conditions described above becomes:

\[
p_r = 1 - \left[ \frac{1}{c} \right] \left[ \frac{N_h^*(t_2)}{N_h(t_2)} \right]
\]  

The substitution of equation (23) and its counterpart for the treated population into equation (25) will result in a very complicated expression involving a ratio of sums of integrals. But it reduces to a particularly simple form when: (1) \( v_h(0, t) \) is so close to 0 for \( t_1 \leq t \leq t_2 \) that the second term of equation (23) can be neglected (which may happen either in the case in
which \( t_2 \) is very close to \( t_1 \), or recruitment into stage \( h \) has virtually stopped), and (2) when \( y_h^*(s, t) \) of equation (24) is not dependent on sojourn time. This latter condition will apply when the same pesticide-specific hazard rate applies to all insects in the growth stage, regardless of their sojourn time in the stage. Here equation (25) reduces to:

\[
p_r = 1 - \exp\left[-\int_{t_1}^{t_2} y_h^*(\tau) \, d\tau\right]
\]

A special case of this equation, using the pesticide-specific hazard rate of equation (10), has been referred to in the previous section as an ‘adjusted cumulative pesticide efficacy curve’ and we will now use this term (referred to hereafter as ACPEC) for general expressions of the form of equation (26). Thus it is clear that the estimand of \( p_r \) is dependent on the nature of the pesticide effect and on the rate of recruitment into the growth stage. If the pesticide affects all insects in a growth stage equally, regardless of their sojourn time in the stage, \( p_r \) gives values of the ACPEC whenever there is no recruitment into the stage. It will probably give values close to those of the ACPEC if the rate of recruitment is very low relative to the number of insects in the stage or if \( t_2 \) is close to \( t_1 \). Because of the complexity of equation (25) when the above-mentioned conditions are not met, we turn to simulation to get a feel for the estimand of \( p_r \) in other circumstances. We also use this simulation to investigate the estimand of the ‘pooled \( p_r \)’, the \( p_r \) value computed using the total numbers of insects in all of the growth stages.

We simulate two populations with identical dynamics (although different total population sizes) except that one is a control population and the other will be subject to pesticide effects after a certain date. We want to investigate the estimand of \( p_r \) under conditions of varying levels of recruitment into the populations and varying relative numbers of insects in the growth stages of the populations, and we would like the model populations to be plausible. We can accomplish these objectives by using the fitted parameter values for both the population model and the pesticide effectiveness model from block 1 of the deltamethrin experiment discussed in the preceding section, but varying the time of application. This is because these parameter values are based on real data, and under these values, recruitment occurs over a short period of time, the stage structure of the population changes rapidly once the second stage (3rd instar) insects begin to appear, the population eventually attains a homogeneous stage structure with no recruitment, and the differences among the growth stages in pesticide effectiveness are moderate. We will simulate the control and treated populations, with the treatments applied for each of the simulations at one of the 5-day increments between and including 35 and 70 days after the start of emergence.
Fig 5. Computed values of the modified Abbott's formula ($P$) versus time after pesticide application (DAY) based on the numbers of insects in growth stages 1 (solid line), 2 (dashes) and 3 (dotted line) of hypothetical control and treated populations (see text). For reference, the ACPEC values (see text) have been plotted for each of the growth stages using growth stage numbers as plotting symbols. Figs. 5a, b, c, d, e, f, g and h give the results for a pesticide which is applied on days 35, 40, 45, 50, 55, 60, 65 and 70, respectively, of the time scale for phenological development of the insect population.
We calculate and plot the stage-specific $p_r$ values versus time (since pesticide application) (Fig. 5a–5h) as well as the pooled $p_r$ values versus time (Fig. 6). For comparison, we also plot the ACPEC values for each growth stage (Fig. 5a–5h).

The results of the simulations, noting in particular the differences among the simulations corresponding to the various times of application of the pesticide, confirm that estimands of the stage-specific $p_r$ values are very dependent on relative rates of recruitment into the growth stages, and the estimand of the pooled $p_r$ is dependent on the stage structure of the population. A shift of just 5 days in the time of application, especially in the early days of the population, has dramatic effects on the estimand of $p_r$. Note the large differences between the $p_r$ curves for applications dates 35 and 40 (Fig. 5a vs. 5b), or for dates 40 and 45 (Fig. 5b vs. 5c). When the pesticide is applied on day 35, none of the stage-specific $p_r$ curves are monotonic (Fig. 5a), but for stages 1 and 3 they become monotonic for later application dates. The $p_r$ curves for growth stage 2 never become monotonic but rather take on an oscillating behavior as the time of application increases.

As the rate of recruitment decreases to zero, the $p_r$ curve for growth stage 1 converges to the ACPEC for growth stage 1 (Fig. 5). Also, as the rate of maturation of individuals into growth stage 3 declines, the $p_r$ curve for growth stage 3 converges to the ACPEC for growth stage 3. Surprisingly, however, the $p_r$ curve for growth stage 2 never seems to converge to the ACPEC for growth stage 2 even though the rate of maturation of insects...
from stage 1 eventually becomes very small. This is probably because when
the absolute rate of recruitment gets very small, the number of individuals in
growth stage 2 also gets very small (Fig. 2a). Hence the relative rate of
recruitment may not get very small. This situation may be magnified by the
fact that the effect of the pesticide on insects of stage 2 is greater than that
on insects of stage 1 (Fig. 4a).

The idea that the estimands of the stage-specific $p_r$ values might be
similar to the ACPECs for times very close to the time of application is
verified by the simulation results. However, this fact may not be of much use
because at best, the $p_r$ curve for growth stage 2 was close to the ACPEC for
only 2 days (Fig. 5h).

The pooled $p_r$ estimands always appear to be monotonic (Fig. 6). There
are very drastic changes in the $p_r$ curves as the time of application varies
from day 35 to day 50, but eventually the curve converges to the cumulative
pesticide effectiveness curve for growth stage 3, which is not surprising since
the population eventually is dominated by insects of growth stage 3.

Both the analytical and simulation results underscore the dependence of
$p_r$ on the rates of recruitment into the population and rates of maturation
from one growth stage to the next. Because these rates change over time as
the population matures, $p_r$ is dependent on the time of application of the
pesticide. All of this can have important implications. The finding by
Johnson et al. (1986) that deltamethrin had only a 65% rate of control for
grasshoppers after 4 days, based on their estimate of the pooled $p_r$, led to a
trial of a different formulation ("flowable") of deltamethrin (Johnson et al.,
1985) which turned out to have virtually 100% control. Subsequent research
has confirmed the superiority of the flowable formulation (e.g., Johnson and
Hill, 1986). Our findings on the dependence of $p_r$ on the age structure of the
population, however, suggest that if the original trial of deltamethrin had
been carried out at a later date, $p_r$ might have had a much higher value (Fig.
6) and a search for better formulations might not have been undertaken.
Based on $p_r$ alone, several trials at different phenological stages need to be
carried out to get a complete understanding of the effectiveness of a
pesticide.

Another implication of this research is that in pesticide efficacy trials
arranged in randomized blocks, slight differences in phenological status
between the blocks (as was observed in the deltamethrin trial) will result in
variability between the blocks in the comparisons of control plots to treated
plots even if there are no sampling errors nor interblock differences in
population sizes or dynamics, and even if the stage-specific pesticide effects
are identical in both blocks. This non-random variability will inflate the
error term in the analysis of variance and render the analysis inefficient. An
efficient analysis would somehow adjust mathematically for these non-ran-
dom differences.
DISCUSSION

In this paper we have investigated issues relating to the construction of a model for an insect population affected by a pesticide. We have looked at the assumptions one needs to make in order to apply a pesticide effectiveness model (Schaalje, 1988) to a sojourn time model to construct the pesticide-population model, have investigated numerical issues relating to computer implementation of such a model, have fitted such a model to data from a pesticide efficacy trial, and have used the model to investigate properties of the modified Abbott’s (1925) formula ($\rho_r$) often computed in connection with pesticide efficacy trials.

Some results are of a negative nature. For example, in their most general form, sojourn time models for insect population dynamics are little more than a matter of bookkeeping (Schaalje and van der Vaart, 1989) and could be used in virtually all situations if the relevant time-varying instantaneous rates of mortality and maturation from one growth stage to the next were known. But in spite of this generality, they are of limited use in connection with a model of how the instantaneous rates of mortality are affected by a pesticide application to an individual because of the varying lengths of time spent by individuals in the various growth stages, coupled with the fact that many pesticides have stage-specific effects. One would have to keep track of the lengths of time spent in each stage by each member of the population in order to calculate the correct values of the stage- and sojourn-time-specific mortality rates. But this would be no different from modelling the population by modelling each member of the population separately and then summing over individuals to predict population dynamics. Either way, the computing resources needed to carry out such a modelling effort would be very great and the analytical advantages of working with a simplified sojourn time model would be lost.

On the positive side, however, it makes sense in many situations to assume that individuals become free of pesticide as they pass from one growth stage to another. In this case, a very simple pesticide-population model can be constructed from a sojourn time model and a pesticide effectiveness model. We were able to fit such a model to data from a trial of deltamethrin for control of grasshoppers. Our purpose in fitting the model to data was to see if it could exhibit behavior similar to that of observed data, and it could. But there might also be a greater role for the procedure of fitting a pesticide-population model to pesticide efficacy trial data. When investigating the properties of the modified Abbott’s index for estimating pesticide effects in a population, we found it to be very sensitive to the phenological state of the population and thus it could only be recommended as an indicator of pesticide effectiveness if the experiment were to be
repeated at several different phenological stages. One wonders if the routine fitting of pesticide effectiveness parameters to data by means of the model might be a way of extracting more ‘portable’ information from a single set of such data.

The advantages of such a procedure would be that if the model were correct, fewer trials would be needed to predict the effectiveness of the pesticide under a wide variety of circumstances, and the parameters in the model would all have a specific meaning. Some of these parameters might be intriguing and useful. For example, \( \alpha \) in the pesticide-effectiveness model gives the degradation rate of the pesticide from the point of view of the insect. It would integrate the chemical degradation rate, the dilution of the pesticide due to growth of the plants on which it is deposited, and any behavioral changes that the insects might employ to avoid the pesticide.

The disadvantages of the procedure would stem from the fact that the model is highly parametrized and the parameters can only be estimated by means of iterative nonlinear optimization routines. Some of the parameters may be nonidentifiable or highly correlated and thus prone to unstable estimates. Furthermore, the model may not be correct and thus there needs to be some way of verifying the appropriateness of the model. This might involve laboratory experiments to validate the forms of the pesticide effectiveness and population models. On the other hand, advantages may accrue from the generation of hypotheses to be examined in the laboratory. It may be of use in other applications to know more about such things as stage-specificity of the effects of the pesticide, the manner in which pesticide residues in the insects are affected by stage transitions, details of the pesticide effectiveness model (such as time-lags, clearance rates, etc. – see Schaalje, 1988), and whether or not natural mortality rates and maturation rates are affected by pesticides.

Before recommending this data analysis procedure for routine use in connection with pesticide efficacy trial data, one would like to reach a thorough understanding of the model. Greater numerical efficiency both in running the model and fitting the model to data could possibly be achieved. For example, Kempton (1979) took an approach similar to ours as far as the control data are concerned, but he got around having to run through the whole population model at each stage of the iterative fitting process by assuming forms for the distributions of latent maturation times that allowed an analytical computation of the number of insects in each growth stage only at the observation times. The incorporation of the pesticide effectiveness model into our population model rendered his method inapplicable in our case, but some similar kind of simplification may be possible. Some of the computational methods used by Mode and Pickens (1988) for renewal and semi-Markov processes may also be applicable. In addition, simulations
need to done to investigate statistical properties of the procedure such as robustness of the fitting procedure. It may be worth considering a fitting method developed by Braner and Hairston (1989) which requires the fitting of additional parameters to characterize the error structure. Finally, sensitivity analyses need to be carried out to determine individual and joint effects of the parameters on the model.

All in all, the prospects seem good that the models of insect populations affected by pesticides discussed in this paper will prove useful in many applications, one of which is certainly the analysis of data from pesticide efficacy trials.

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REFERENCES


