Feature

Predicting the Evolution and Dynamics of Herbicide Resistance in Weed Populations[1]

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Abstract. Herbicide resistance jeopardizes the usefulness of valuable chemical tools and, therefore, weed management in many crop systems. Models must be developed to evaluate management tactics that prevent, delay, or reduce resistance. The complexity of biological processes involved in herbicide resistance also requires models to focus research and to integrate experiments. A population model was developed that improves upon previous attempts to predict herbicide resistance dynamics. The model incorporates plant population demographics with the Hardy-Weinberg concept for gene segregation. The model simulates the evolution, spread, and subsequent dynamics of resistance in the presence and absence of a herbicide. Analysis of model simulations identified two sets of biological processes as key factors in the evolution and dynamics of herbicide-resistant weed populations. These are processes that influence ecological fitness and gene flow. Several options are suggested as examples for the management of resistant weed populations.
Additional index words: Population model, resistance management, seed immigration, pollen immigration, population genetics.

INTRODUCTION

Herbicides are used extensively in agriculture because they are cost-effective tools to reduce weed abundance and to improve crop yields. Recent trends in herbicide development have produced extremely specific and selective chemicals that are used intensively and routinely in cropping systems. The intensive and widespread use of such herbicides has precipitated an alarming increase in the evolution of resistance (9, 15, 28), which jeopardizes product usefulness, availability, and longevity (15). Since the first reported cases of herbicide resistance (26, 27, 30), over 50 plant species resistant to triazine herbicides have been reported (25). In addition, numerous weed species have developed resistance to chemical classes of herbicides other than triazines (13, 22, 33, 35).

The complexity of biological processes that influence herbicide resistance dictates a research approach that focuses on the interaction between life history processes and population genetics. Models can serve such a function and can provide a tool for evaluating management tactics. Review papers on the population biology of pesticide resistance have indicated similar approaches for studying and managing resistance (7, 19, 29, 34, 36, 37). Gressel and Segel (10, 11) developed a model that suggested important factors that influence occurrence and evolution of herbicide resistance. However, their model did not include the gene flow processes of immigration and important factors influencing fitness that may improve prediction of local evolution, spread, and subsequent dynamics of the R[3] trait in a S[3] population of weeds.

Population processes that determine the relative fitness of phenotypes are survivorship (demography of seeds, seedlings, and mature plants), fecundity (pollen and seed production), and plant competition. When a herbicide is used, its selection pressure (reduced survivorship of susceptible individuals) overwhelmingly increases the relative fitness of the resistant genotype (10, 11). However, when herbicide selection pressure is removed, population dynamics are determined by differences in all processes that contribute to the fitness of each biotype.

We have developed a population model that simulates the evolution, spread, and dynamics of R and S weed biotypes (Figure 1 and Table 1). This model improves upon the biological interpretations suggested by Gressel and Segel (10, 11) and provides a refined approach for evaluating the importance of specific biological processes involved in the dynamics of herbicide resistance. The model combines plant population demographics (18, 21) and the Hardy-Weinberg equation (2, 3) to determine proportions of R and S genotypes in successive generations.

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[3]Abbreviations: R, herbicide resistant; S, herbicide susceptible.

Many herbicides inhibit a specific enzyme that can be coded for by a single gene; thus, use of the Hardy-Weinberg inheritance model is a reasonable approach. The model incorporates differential fitness (i.e., survivorship, competitive ability, and fecundity) of R and S genotypes in the presence and absence of a herbicide (selection pressure). Genotype proportions also are modified by gene flow (i.e., immigration, seed bank dynamics, inbreeding, and random genetic drift) and by mutation.

Simulations using this model predict rapid early evolution of resistance from repeated herbicide applications in the absence of an adjacent source population of the S phenotype (Figure 2). After herbicide use is suspended, the model forecasts a decline in resistance with return to populations dominated by the S type. The rate of decline in resistance depends on life-history processes, immigration processes, mechanisms of inheritance, reproductive mechanisms, and the relative fitness of the R and S phenotypes.

MODEL DEVELOPMENT

The model, like earlier herbicide resistance models (8, 10, 11), is theoretical. It was developed to generate hypotheses on the influence of demographic and inheritance processes on resistance evolution and management. The simulation model was constructed by linking eight submodels representing life-history stages (seed bank, seedling, mature plants, pollen producers, seed yield), immigration, and inheritance (Figure 1). This structure allows alternative submodels to be inserted and tested as new information on particular processes becomes available.

The current model assumes for mathematical simplicity that the weed is a single-cohort (one germination time) annual, that all parameters are held constant over time unless indicated otherwise (Table 1), and that herbicide resistance is associated with a single gene locus. Thus, it is assumed that herbicide resistance is determined by a single pair of alleles, where **a** denotes the recessive allele and **A** the dominant allele. The computer model allows the user to select (Figure 3) if the genotype **aa**, the homozygous recessive, results in the R phenotype and **AA** and **As** represent S phenotype plants or if **aa** results in the S phenotype and **AA** and **Aa** are R. Throughout this paper the **aa** genotype was assumed to confer resistance. The model will allow for different inheritance patterns by inserting a different inheritance submodel. A separate description of each submodel follows.

Seed bank submodel. The number of R and S weed seed in the seedbank was assumed to be a function of seed mortality and germination rates for each phenotype. This amount is supplemented by the number of seed entering the seedbank from the treated population and seed immigration.

The number of R and S seed in the seedbank before germination (RSB and SSB, respectively) is expressed as

$$RSB = RSB_{t-1} + Ryld_{t-1} + RISD_{t;\ -\ Rm}(RSB_{t-1}) \quad [2.1]$$

$$SSB = SSB_{t-1} + Syld_{t-1} + SISD_t - S_m(SSB_{t-1}) \quad [2.2]$$

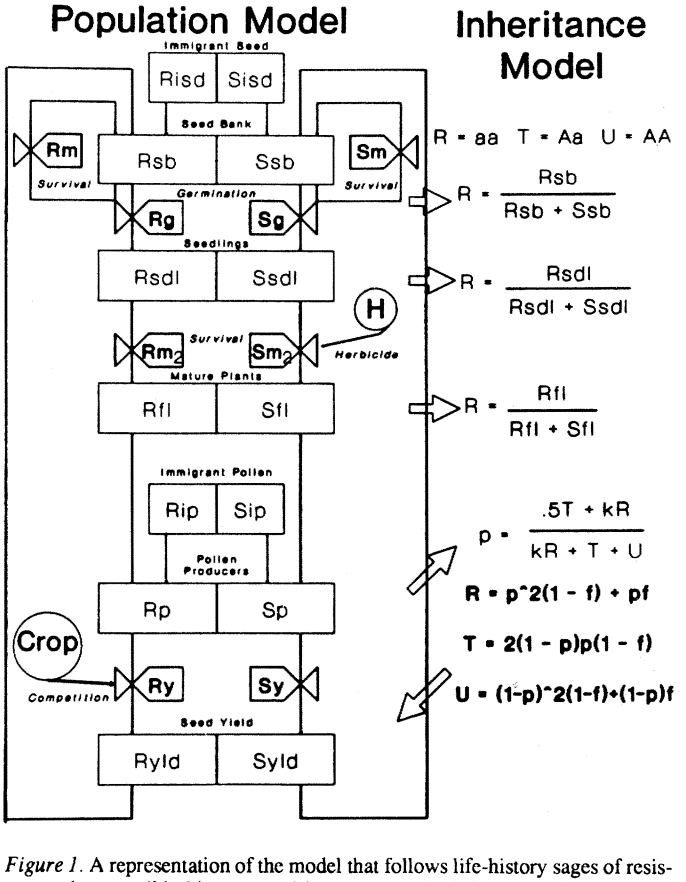


Figure 1. A representation of the model that follows life-history sages of resistant and susceptible biotypes and incorporates the influences of fitness processes and gene flow. Open arrows indicate the flow of information between the population model and the inheritance model. State variables are named above each box and processes are indicated in italics. Abbreviations are explained in the text and Table 1.

Table 1. Definitions used in the development of the herbicide resistance model (see Figure 1).

Parameter	Definition and default values	Equation number
t	Current generation	
Immigration parameters		
a_{sd}	The number of pollen-produced per unit area by the source population. a_{sd} = TOT = model input value (Figure 5).	1.1
a_p	The number of pollen-producing plants per unit area representing the total amount of pollen produced by the source population. $a_p = a_{sd}/10$. 5.1	
x′	The distance from the center of the source population to the center of the treated population.	1.1 & 5.1
c_s	The radius of the total source area (Figure 4).	1.1 & 5.1
c_o	The radius of the interior source population that is equal to the treated population radius (0.5) (Figure 4).	1.1 & 5.1
c	The square root of the scaling factor [$c = (c_s^2/c_o^2)^{\frac{1}{2}}$] (Figure 4).	
bp	The steepness of the diffusion gradient for pollen (1.7).	5.1
bsd	The steepness of the diffusion gradient for seed (7.4).	1.1
Demographic parameters		
RSB	The number of R seed in the seed bank before germination.	2.1
SSb	The number of S seed in the seed bank before germination.	2.2
R_m	The proportion of R seed that die over 1 generation in the seed bank (0.7).	2.1
S_m	The proportion of S seed that die over 1 generation in the seed bank (0.7).	2.2
ISD	The total number of seed added to the seed bank through immigration over 1 generation.	1.1
RISD	The number of immigrant seed that is R-phenotype.	2.1
SISD	The number of immigrant seed that is S-phenotype.	2.2
R_g	The proportion of R seed that germinate over 1 generation (0.3).	2.3
S_g	The proportion of S seed that germinate over 1 generation (0.3).	2.4
RSDL	The number of R seedlings produced from seed germinating in the seed bank.	3.1
SSDL	The number of S seedlings produced from seed germinating in the seed bank.	3.2
R_{m2}	The proportion of R seedlings that die over 1 generation (0.75).	3.3
S_{m2}	The proportion of S seedlings that die over 1 generation (0.75).	3.4
RFL	The number of R mature (flowering) plants.	4.1
SFL	The number of S mature (flowering) plants.	4.2
h	Herbicide efficacy on seedlings (95%).	4.2
IP	The total number of pollen-producing plants represented by immigrant pollen.	5.1
RP	The number of R pollen-producing plants represented by immigrant pollen.,	6.1
SP	The number of S pollen-producing plants represented by immigrant pollen.	6.2
TM	The total number of mating plants.	7.7
TMr	The number of mating plants that will produce R seed.	7.8
TMs	The number of mating plants that will produce S seed.	7.9
RY	The number of R seed produced per R plant.	8.1
SY	The number of S seed produced per S plant.	8.2
Ryld	The R seed yield per unit area.	8.3
Syld	The S seed yield per unit area.	8.4
Tyld	The seed yield per unit area with genotype Aa.	2.7
Inheritance parameters		
Rsb	The proportion of the seed bank that is R-phenotype.	2.5
Tsb	The proportion of the seed bank that is Aa genotype.	2.7
Usb	The proportion of the seed bank that is Aa genotype.	2.8
Ssb	The proportion of the seed bank that is S-phenotype.	2.6
Rsdl	The proportion of seedlings that are R-phenotype.	3.5
Tsdl	The proportion of seedlings that are Aa genotype.	3.7
Usdl	The proportion of seedlings that are AA genotype.	3.8
Ssdl	The proportion of seedlings that are S-phenotype.	3.6

(continued)

Table 1. (*continued*) Definitions used in the development of the herbicide resistance model (see Figure 1).

Parameter	Definition and default values	Equation number
Inheritance parameters		
Rfl	The proportion of mature (flowering) plants that are R-phenotype.	4.3
Tfl	The proportion of mature (flowering) plants that are Aa genotype.	4.5
Ufl	The proportion of mature (flowering) plants that are AA genotype.	4.6
Sfl	The proportion of mature (flowering) plants that are S-phenotype.	4.4
Rof	The proportion of R pollen-producing plants in the outside (source for immigration) population.	1.2
Sof	The proportion of S pollen-producing plants in the outside (source for immigration) population.	1.3
Tof	The proportion of pollen-producing plants with Aa genotype in the outside (source for immigration) population.	2.7
Rp	The proportion of R pollen-producing plants in the treated population.	6.3
Sp	The proportion of S pollen-producing plants in the treated population.	6.4
Tp	The proportion of pollen-producing plants with Aa genotype in the treated population.	6.5
Up	The proportion of pollen-producing plants with AA genotype in the treated population.	6.6
p	The probability of an individual's donating the a allele in a mating.	7.1
kr	The fitness of the R pollen relative to the S pollen (0.9).	7.1
f	The inbreeding coefficient.	7.5
m	The forward mutation rate to resistance (10^{-6}).	7.5
Ne	The effective size of the population (related to the number of reproducing adults).	7.6
R	The proportion of the total matings that will produce R seed.	7.2
T	The proportion of the total matings that will produce seed with Aa genotype.	7.3
U	The proportion of the total matings that will produce seed with AA genotype.	7.4
S	The proportion of the total matings that will produce S seed.	7.4
RY_{max}	The maximum yield per R plant (900 seeds/plant).	8.1
SY_{max}	The maximum yield per S plant (1000 seeds/plant).	8.2
a_r	The area required to produce RY_{maxi} (1).	8.1
a_s	The area required to produce SY_{maxi} (1).	8.2
z_{sr}	The influence of S plant density on the seed yield of R plants (1).	8.1
z_{rs}	The influence of R plant density on the seed yield of S plants (1).	8.2
z_{ir}	The influence of another weed or crop plant (i) density on the seed yield of R plants (1.2).	8.1
z_{is}	The influence of another weed or crop plant density on the seed yield of S plants (1.1).	8.2
N_i	The density of the other weed or crop (200 plants/m^2).	8.1 & 8.2
br	The coefficient which determines the form of the relationship between RY_t and the total density (0.8).	8.1
bs	The coefficient which determines the form of the relationship between SY_t and the total density (0.8).	8.2

where $Ryld_{t-1}$ is the number of R seed produced per unit area by the previous generation and $Syld_{t-1}$ is the number of S seed produced per unit area by the previous generation in the treated population. The mortality rates for R and S seed are R_m and S_m, respectively.

Seed immigration submodel. Immigration of genes has two points of origin: seed and pollen from outside the treated population. The immigration of seed from an outside population is treated as an influx into the seedbank. Pollen immigration enters the model in the pollen producer submodel. The proportions of genotypes immigrating from a source are assumed to be the same as the proportions in the source (outside field) population. Emigration was not included in the model.

Seed immigration is a function of dispersal which involves several variables: a) heights and distance of the seed source, b) concentration at the seed source, c) dispersibility of the seed (e.g., weight, possession of wings, plumes, etc.) and d) activity of distributing agents (e.g., wind direction and velocity) (12). These variables are generally species specific and require specific models. In the current model, seed immigration is based on a diffusion gradient model used to predict a plant disease gradient (24) and pollen dispersal (6).

$$ISD_t = (a_{sd} (x' + c_o)^{-bsd}) (c_s^2 / c_o^2) \quad [1.1]$$

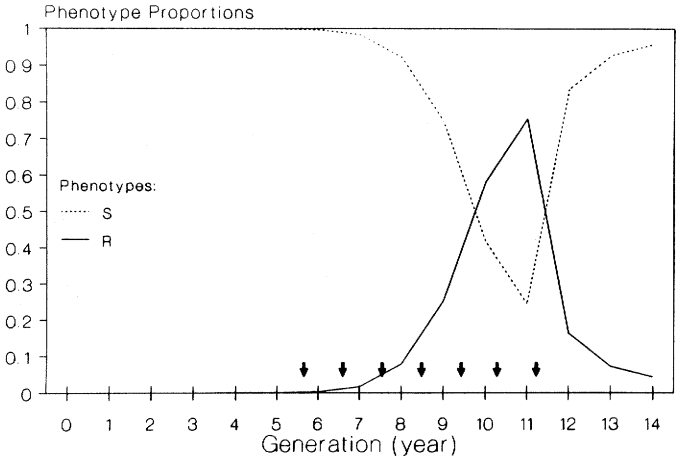


Figure 2. Model simulations describing the evolution of resistance when the herbicide is used in the system and the subsequent dynamics of resistance in the weed population after the herbicide has been removed. The solid arrows indicate the years of continuous herbicide use.

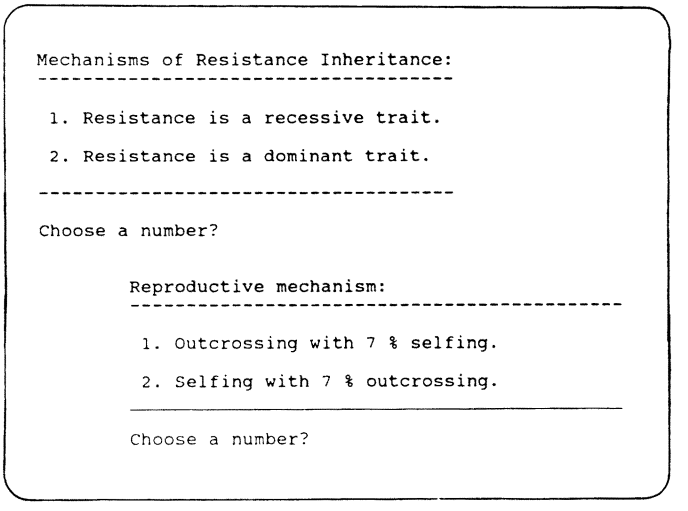


Figure 3. The first input screen for RSIM (the interactive computer program version of the model) used for selecting the mechanism of resistance inheritance and the reproductive mechanism for a weed species of interest.

$$RISD_t = ISD_t(Rof) \quad [1.2]$$

$$SISD_t = ISD_t(Sof) \quad [1.3]$$

ISD is the total number of immigrant seed per unit area entering the treated population seedbank from outside populations (Figure 4). The parameter a_{sd} is the number of seed produced per unit area at $1–c_o$ units of distance from the center of the source, which is equivalent to the total number of seed produced by the source when it is equal in size to the treated population. Since the source was assumed to be equal in size to the treated population, it was also assumed that the source would produce the same amount of seed as the treated population.

The x′ parameter is the distance (in treated population diameters) from the center of the source population to the center of the treated (receptor) population, and c_o is a truncation factor which approximates the radius of a source population (23) in the center of the total source that is equal in size to the treated population (Figure 4). Setting all measures relative to the size of the treated population allows for assessment of immigration at all scales. The parameter bsd is the slope of a linear regression of log(Isd) on $\log(x' + c_o)$. To accommodate immigrant sources larger or smaller than the treated population, a scaling factor (c^2/ c_o^2) was added to the immigration equation, where c_s is the total source (a set of source populations) area radius in units of treated population diameters.

The number of R and S seed reaching the treated population as a result of immigration are RISD and SISD, respectively. The proportions of each genotype in the outside (source) population are Rof (genotype **aa**), Tof (genotype **Aa**), and Uof (genotype **AA**). The proportion of the susceptible phenotype in the source population is Sof = Tof + Uof.

After germination the numbers of R and S seeds that remain in the seedbank until the next generation are

$$RSB_t = RSB - R_g(RSB) \quad [2.3]$$

$$SSB_t = SSB - S_g(SSB) \quad [2.4]$$

where R_g and S_g are germination rates for R and S seed.

The proportion of each genotype (Rsb = **aa**, Tsb = **Aa**, Usb = **AA**) in the seed bank at time t is calculated by

$$Rsb_t = \frac{RSB}{RSB + SSB} \quad [2.5]$$

$$Ssb_t = \frac{SSB}{RSB + SSB} \quad [2.6]$$

$$Tsb_t = \frac{Tsb_{t-1}\,(RSB_{t-1} + SSB_{t-1}) + Tyld_{t-1} + ISD_t\,(Tof)}{RSB + SSB} \quad [2.7]$$

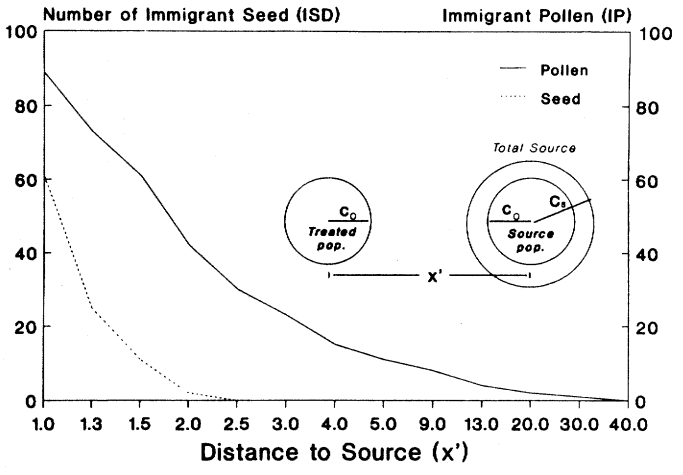


Figure 4. Pollen and seed dispersal as a function of distance (in units of treated population diameter) to a source population.

$$Usb_t = Ssb_t - Tsb_t \quad (2.8)$$

Rsb_t, Ssb_t, and Usb_t are the proportions of R phenotype (**aa** genotype), S phenotype, **Aa** genotype, and **AA** genotype, respectively, in the seedbank of the current generation. Tsb_{t-1} is the proportion of the heterozygous (**Aa**) genotype in the seedbank during the previous generation; $Tyld_{t-1}$ is the number of seed produced per unit area by the treated population in the previous generation that were the heterozygous (**Aa**) genotype.

Seedling submodel. The number of R and S seedlings (RSDL and SSDL, respectively) in the treated population at time t is calculated as follows:

$$RSDL = R_g(RSB) \quad [3.1]$$

$$SSDL = S_g(SSB) \quad [3.2]$$

where R_g and S_g are germination rates for R and S seed, respectively.

The number of seedlings of R and S phenotype that survive to become flowering plants is regulated by seedling mortality rates for the R (R_{m2}) and S (S_{m2}) phenotypes:

$$RSDL_t = RSDL(R_{m2}) \quad [3.3]$$

$$SSDL_t = SSDL(S_{m2}) \quad [3.4]$$

The proportion of seedlings of each genotype at time t are then calculated by

$$Rsdl_t = \frac{RSDL_t}{RSDL_t + SSDL_t} \quad [3.5]$$

$$Ssdl_t = \frac{SSDL_t}{RSDL_t + SSDL_t} \quad [3.6]$$

$$Tsdl_t = \frac{Tsb_t\ (Ssdl_t)}{Ssb_t} \quad [3.7]$$

$$Usdl_t = Ssdl_t - Tsdl_t \quad [3.8]$$

The parameters are defined in Table 1.

The current model includes the influence of the herbicide at the transition from the seedling to mature plant life-history stage. The effect of the herbicide on the weed population is based on its efficacy in S populations. The herbicide efficacy (h) is equivalent to the percent control relative to an untreated control plot of a S population.

Mature plant submodel. The number of R and S mature individuals in the population (RFL and SFL, respectively) at time t is equal to the number of seedlings in the population after accounting for the effect of the herbicide and R and S seedling mortality.

$$RFL_t = RSDL_t \quad [4.1]$$

$$SFL_t = SSDL_t - h(SSDL_t) \quad [4.2]$$

The proportions of each genotype at the mature stage are calculated as follows:

$$Rfl_t = \frac{RFL_t}{RFL_t + SFL_t} \quad [4.3]$$

$$Sfl_t = \frac{SFL_t}{RFL_t + SFL_t} \quad [4.4]$$

$$Tfl_t = \frac{Tsdl_t\ (Sfl_t)}{Ssdl_t} \quad [4.5]$$

$$Ufl_t = Sfl_t - Tfl_t \quad [4.6]$$

The parameters are defined in Table 1.

Pollen immigration submodel. The same equation that is used for seed immigration (Equation 1.1) is used for predicting pollen immigration but with different coefficients (a_p and bp). The immigration submodel has been adapted to predict the total number of pollen-producing plants (IP) represented by pollen reaching the treated population (Figure 4). It, therefore, is assumed that all the plants in the source population produce the same amount of pollen and that the proportions of immigrating R and S pollen are the same as the phenotype proportions of individual plants in the outside source population.

$$IP_t = (a_p\ (x' + c_o\ )^{-bp}\ )\ (c^2\ /\ c_o^2\ ) \qquad [5.1]$$

The a_p parameter is the number of pollen-producing plants per unit area at $1–c_o$ units of distance from the source population. This value is equivalent to the total number of pollen producers in the source population which is assumed to be the number of seed produced in the source population divided by a constant. The number of seed produced in the source is assumed to be equal to that produced in the treated population of the same size. The bp parameter is the slope of a linear regression of log(Ip) on $\log(x' + c_o)$. This parameter controls the steepness of the diffusion gradient which is a function of the pollen grain mass and shape as well as air flow properties. The other parameters are described in the seed immigration submodel (Equation 1.1) and in Table 1.

Pollen producer submodel. The total number of pollen producers for the treated population that are R (RP) and S (SP) at time t are calculated as

$$RP_t = Rfl_t(RFL_t + SFL_t - IP_t) + Rof(IP_t) \qquad [6.1]$$

$$SP_t = Sfl_t(RFL_t + SFL_t - IP_t) + Sof(IP_t) \qquad [6.2]$$

The proportions of pollen producers represented by each genotype are

$$Rp_t = \frac{RP_t}{RP_t + SP_t} \qquad [6.3]$$

$$Sp_t = \frac{SP_t}{RP_t + SP_t} \qquad [6.4]$$

$$Tp_t = \frac{Tfl_t\ (RP_t + SP_t\ ) - IP_t\ ) + Tof(Ip_t\ )}{RP_t + SP_t} \qquad [6.5]$$

$$Up_t = SP_t - Tp_t \qquad [6.6]$$

The parameters are defined in Table 1.

Inheritance submodel. The probability (p) of an individual's donating the **a** allele in a mating is based on the proportions of each genotype at the pollen production stage. The fitness of the R pollen relative to the S pollen (k_r) is included in the equation.

$$p = \frac{0.5\ Tp_t\ + k_r\ R_{pt}}{k_r\ R_{pt} + T_{pt} + U_{pt}} \qquad [7.1]$$

The basic Hardy-Weinberg model is based on the assumption that populations are infinitely large and mating is random (panmictic). In populations of defined size or breeding behavior that are not random, there is a potential for inbreeding. The Hardy-Weinberg equation may be modified by inclusion of an inbreeding coefficient (32, 38). The following equations result:

$$R_{t+1} = p^2(1–f) + p(f) \qquad [7.2]$$

$$T_{t+1} = 2(1–p)(p)(1–f) \qquad [7.3]$$

$$U_{t+1} = (1–p)^2(1–f) + (1–p)(f) \qquad [7.4]$$

$$S_{t+1} = T_{t+1} + U_{t+1}$$

$$f = \frac{1–2\ m}{4\ N_e\ m–2m+1} \qquad [7.5]$$

In this equation m is the mutation rate which is fixed in the current model at 10^{-6}, which is in the range suggested by Georghiou and Taylor (7). N_e is the effective size of the population which is related to the number of reproducing adults and is calculated as follows when there are differences in the number of male (N_{em}) and female (N_{ef}) adults:

$$N_e = \frac{1}{0.25(1/\ N_{ef} + 1/N_{em}\ )} \qquad [7.6]$$

It is assumed that the male part of a plant population (pollen grains) far exceeds the number of female ovules. Therefore, N_{em} approaches 0 and N_{ef} is approximated by the number of seed produced per unit area by the treated population. The unit area thus defines the finite population size.

The proportions of each genotype proceeding into the next generation have been determined above. To calculate the number of seed representing each genotype, the number of total plants involved in mating first must be calculated; then the number of those matings that will represent production of each phenotype (R and S) is determined.

$$TM_t = RFL_t + SFL_t \quad [7.7]$$

$$TMr_t = R_{t+1}(TM_t) \quad [7.8]$$

$$TMs_t = S_{t+1}(TM_t) \quad [7.9]$$

TM_t is the total number of mating plants in the current generation. TMr_t and TMs_t are the number of mating plants that will produce seed with the R and S phenotypes, respectively.

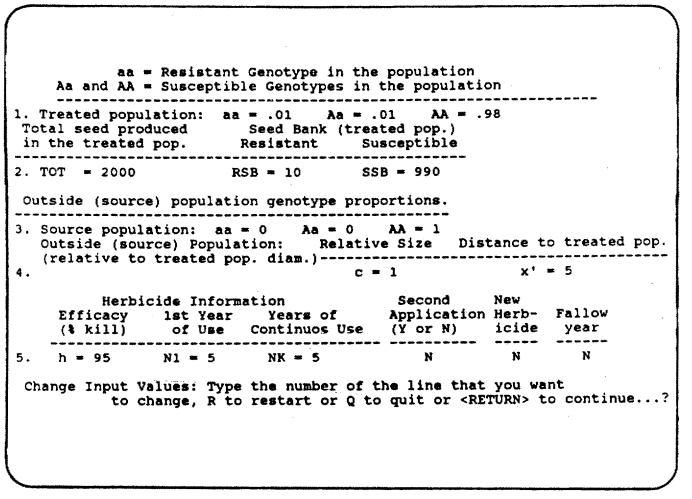
Seed yield submodel. A competition model proposed by Firbank and Watkinson (5) was adapted to predict the seed yield for individual R and S plants. Separate equations for the R and S phenotypes account for differential competitive abilities. The influence of a crop and other weed species can be included in the model in addition to intra- and inter-phenotype competition.

$$RY_t = RY_{max}[1 + a_r(RFL_t + z_{sr}SFL_t + z_{ir}N_i)]^{-br} \quad [8.1]$$

$$SY_t = SY_{max}[1 + a_s(SFL_t + z_{rs}RFL_t + z_{is}N_i)]^{-bs} \quad [8.2]$$

RY_{max} and SY_{max} are the maximum yield per plant that can be attained by the R and S phenotypes, respectively. The areas required to attain R_{max} and SY_{max} are a_r and a_s, respectively. The inter-phenotype competition coefficient which expresses the influence of the S phenotype density on the R phenotype is z_{sr}. The inter-phenotype competition coefficient that expresses the influence of the R phenotype density on the S phenotype is z_{rs}. The inter-specific competition coefficients that express the influence of the crop or other dominant weed density on the R phenotype and the S phenotype are z_{ir} and z_{is}, respectively. N_i is the density of the crop or other dominant species in the system, and br and bs are the coefficients which determine the forms of the relationship between RY_t and SY_t and the total density, respectively.

The seed yields per unit area for the R (Ryld) and S (Syld) phenotypes are calculated as follows:



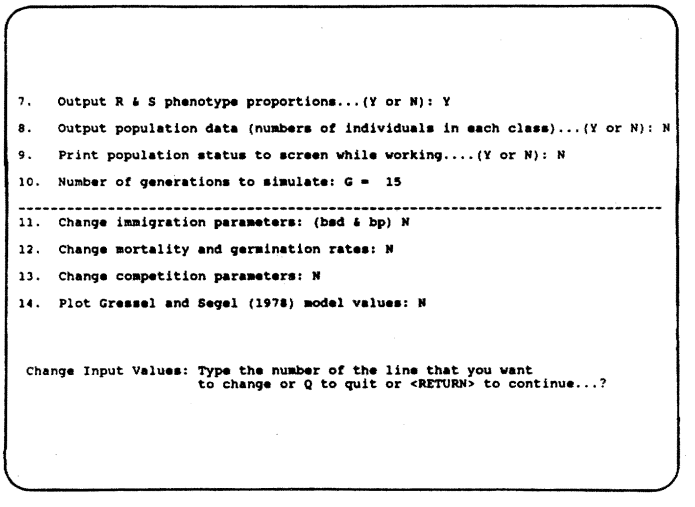


Figure 5. The second and third input screens for RSIM (the computer program version of the resistance simulation model) used for entering the initial conditions for starting a simulation and changing certain model parameter values.

$$Ryld_t = RY_t(TM_t) \quad [8.3]$$

$$Syld_t = SY_t(TM_t) \quad [8.4]$$

Ryld and Syld become the inputs to the seedbank in the next generation (t+1).

Simulations are conducted by inputting a set of initial conditions (Figure 5) and calculating in sequence equations 1.1 through 8.4. The computer program version of the resistance simulation model (RSIM) pro-

duces two types of output: a) Proportions of the R and S phenotype once per generation at the flowering stage before reproduction and b) numbers of individuals at each life-history stage for each generation.

MODEL BEHAVIOR ANALYSIS

Sensitivity and elasticity analysis (18, 20) on the complete simulation model identified two sets of life-history processes that are important for understanding and managing the dynamics of herbicide resistance: a) processes that influence fitness of the R phenotype relative to the S phenotype and crop species and b) processes that contribute to gene flow in space and time.

Fitness. Fitness describes the evolutionary advantage of a phenotype, which is based on its survival and reproductive success (2, 31). Relative fitness of R and S phenotypes has important consequences for the management of resistance. Reduced fitness in the R type (1, 14) infers that R plants will be replaced by S individuals over time after herbicide use is abandoned. Alternatively, if the fitness of the R type is not less than the S type (35), resistance should decline slowly, if at all. Fitness also will be influenced by the presence of other species. (i.e., crop or other weeds) in the system, especially if they are strong competitors. These premises have not been examined experimentally, although the alternatives lead to very different tactics for managing resistance (9).

Gene flow. Gene flow describes the processes that influence the maintenance of a particular genotype in a population. Gene flow processes directly alter the frequencies of R and S alleles in plant populations (16). Immigration of pollen and seed introduce genes into a population, while inbreeding and genetic drift result from limited gene flow. Seed dormancy conserves genes within a plant population. Seed bank dynamics include these gene flow processes, as well as seed survivorship (a fitness process).

Pollen and seed from outside populations are involved in two important management scenarios: a) the spread of resistance over the landscape and b) the use of S-genotype sources (e.g., fence rows, untreated rows, fields, addition of seed) to prevent or slow the evolution of resistance. Attempts to manage herbicide resistance are dominated by tactics to use other herbicides to remove R plants from populations that have developed resistance. Our model simulations suggest that manipulation of S-type gene flow is an alternative for resistance management. Such tactics could be more cost effective than control measures that only reduce R-type plants in already resistant fields.

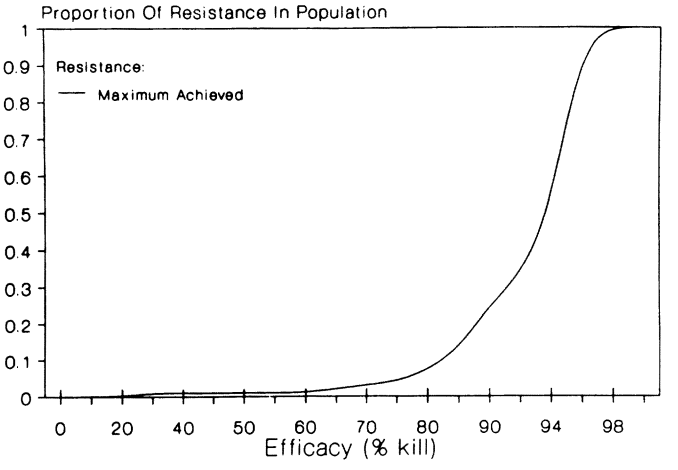


Figure 6. The influence of herbicide efficacy on the maximum level of resistance achieved in a weed population that had 5 continuous years of herbicide applications.

MANAGEMENT SCENARIOS

Simulations. The model was used to assess the influence of gene flow and fitness processes on the evolution of resistance in a weed population. Each of these assessments has management implications. In each analysis, all the parameters except for one were held constant. All the simulations were initiated with selected proportions of each genotype in the treated weed population and an adjacent (source for immigration) population of the same species. Herbicide application begins at Year 5 and is continued through Year 9 in the simulations. All references to the herbicide in the first three scenarios assume that there was a single herbicide in the system and that resistance to that herbicide is a single-gene, homozygous-recessive trait.

The relative fitness of the R and S phenotypes were arbitrarily set equal except in their relative competitive abilities with the crop (R = 1 = S = 1 and R = 0.7 that of the crop, S = 0.8 that of the crop) and their abilities to pollinate and fertilize (R = 0.9 that of S). The crop density was arbitrarily fixed at 200 plants m^{-2}. The following discussion illustrates four scenarios where model simulations were used to explore gene flow and fitness processes and their management implications.

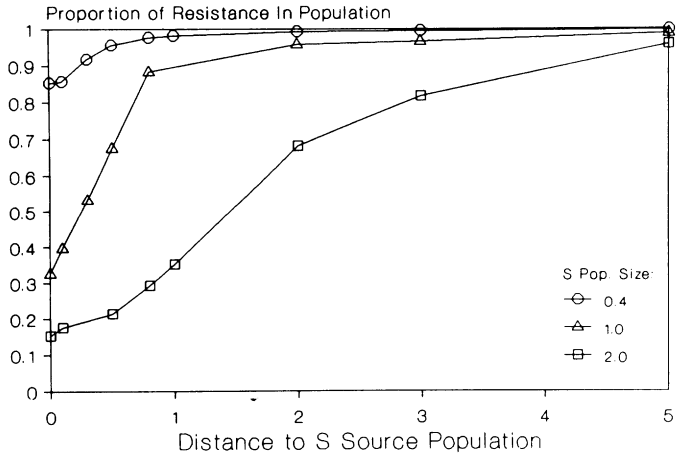


Figure 7. The influence of the size (relative to the treated population) and distance (in units of treated population diameter) to (edge of source to edge of treated) a 100% susceptible source population on the level of resistance achieved after 5 continuous years of herbicide applications.

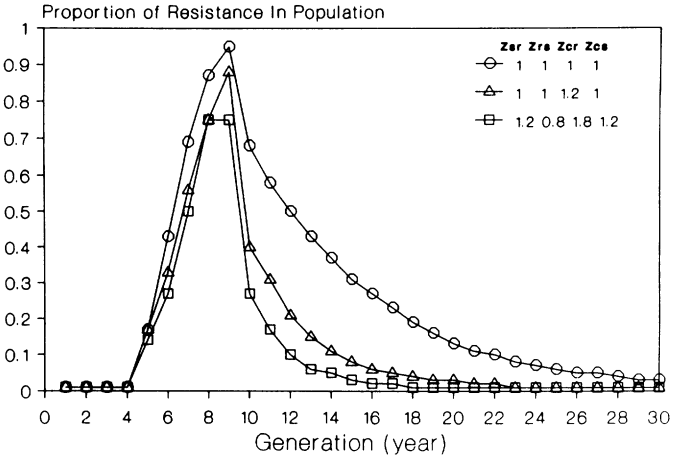


Figure 8. The influence of relative competitive abilities of resistant and susceptible phenotypes in the presence of the crop on evolution of and recovery from resistance (Equations 8.1 and 8.2).

Scenario 1. The influence of herbicide efficacy (h in Equation 4.2) on the evolution of resistance was explored (Figure 6). The simulations were initiated with no resistance in the treated populations, but resistance was introduced in each generation (year) by immigrant pollen from an adjacent (source) population. The model is designed to remove (kill) a proportion of the S individuals equivalent to the efficacy. The maximum level of resistance achieved in the treated population increased sharply when efficacy was about 80%.

The response to efficacy suggested from the simulations has important management implications. Reducing efficacy by intentionally leaving skips in the herbicide application would provide for enough healthy S individuals in the population to reduce the levels of resistance through fitness and gene flow processes. Susceptible individuals also may escape treatment naturally by following a different phenology. The potential for establishing an efficacy threshold to maintain a low proportion of resistance in a weed population is apparent. Competition and economic thresholds have been identified in many weed/crop systems (4) which indicate that high efficacy is often associated with "cosmetic" weed control rather than direct economic gain. Therefore, reducing efficacy to discourage the evolution of resistance may not reduce crop yields.

Scenario 2. This scenario examines the potential for immigrating S pollen and seed to decrease the role of evolution of resistance (Figure 7). The initial conditions for the simulations assumed 1% resistance already in the weed population followed by an increase to 90% resistance at the end of 5 continuous years of herbicide use. The influence of the size of a S source population (relative to the size of the treated population) and distance (in units of treated population diameter) to an outside S source of the weed was assessed for its ability to influence R levels in the treated population.

The simulations indicated that source areas equal to or larger than the treated population can decrease the maximum level of resistance (Figure 7). The management tactic implied by these simulations is to leave untreated adjacent rows or to maintain S populations of the weed dispersed through the treated population within a distance of one treated-population diameter.

Scenario 3. This scenario addresses the influence of competition on the evolution of and recovery from resistance (Figure 8). Competitive abilities of the R and S phenotypes relative to each other and the crop (Equations 8.1 and 8.2) were varied systematically in a set of simulations. Relative competitive ability had little influence on the rate of resistance evolution. The levels of resistance in the population over the first 3 yr of herbicide use did not differ in the simulations. However, the maximum level of resistance after 5 continuous years of herbicide use was highest (95%) when the R and S phenotypes and the crop were assumed to have equal competitive abilities. The maximum level of resistance was reduced to 85% when the R phenotype was assumed to be less competitive than both the crop and the S phenotype of the weed.

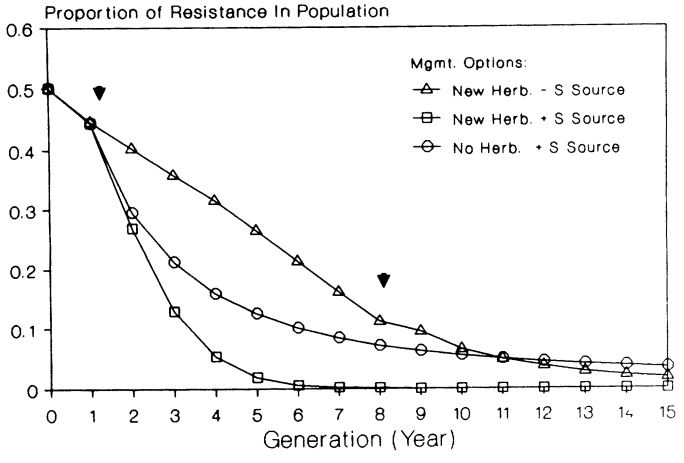


Figure 9. The use of a new herbicide (one that has 85% efficacy on both R and S individuals) and an S source population for managing recovery from herbicide resistance in a weed population. Arrows indicate the beginning and ending of continuous new herbicide applications.

The model simulations indicate that the most significant influence of relative competitive ability on resistance dynamics occurs in the recovery period following suspension of herbicide use (Figure 8). Three years after stopping herbicide use, 30% resistance remained in the population where competitive abilities were equal for the R and S phenotypes and the crop. The proportion of R plants was lower in the recovery period when the relative competitive ability of the R phenotype was decreased relative to the crop and the S phenotype.

The management tactic implied from manipulation of relative competitive abilities is to use or rotate to a crop with a greater competitive ability than the R phenotype. Manipulating competitive pressure by increasing the crop density is an equivalent management option. Changing crop densities had little influence on early evolution and maximum levels of resistance; however, critical crop densities were identified which maximized the rate of recovery to a susceptible weed population following the suspension of herbicide use.

Scenario 4. The model was used to assess management options for recovery after resistance is recognized in a weed population. The simulations were started with 50% resistance in the population. A new herbicide with 85% efficacy on R and S phenotypes was introduced at Year 1 and continued for 8 yr, then all herbicide application was suspended (Figure 9). The simulations indicated that the presence of an adjacent (source) population of S individuals decreased the time for recovery from resistance. Using a new herbicide without a source of immigrant S pollen or seed was not as effective at decreasing the level of R population as was an adjacent population without the use of a new herbicide.

The management implications suggested by these simulations further supports the potential of managing resistance by creating a source of the S phenotype to augment the effect of a new herbicide which will control both R and S weeds.

SUMMARY

The biological complexity and management implications of herbicide resistance can be explored with accurate simulation models that include pertinent biological processes. Gene flow and fitness were identified as important processes influencing resistance dynamics. These processes deserve further experiments to determine their potential for manipulation and management of resistance.

The potential management of resistance suggested by this model represents some alternative strategies and tactics with respect to other attempts to study herbicide resistance. These tactics include methods to decrease the R phenotype and to manipulate the S phenotype of the weed population. Each approach is reasonable, although greatest success should result from multiple integrated tactics for manipulating both R- and S-type weeds in a population.

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