

Low herbicide rates can lead to faster development of herbicide resistance

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Summary We present an individual-based model that simulates the development of polygenic herbicide resistance in the field and explain how this model was used to investigate the issue of whether lower-than-recommended post-emergent herbicide doses could lead to faster development of herbicide resistance. A variety of different possible genetic bases for resistance were considered. The results clearly show that under most assumptions low herbicide rates do indeed lead to faster development of herbicide resistance.

Keywords Herbicide resistance, weeds, simulation model, individual-based, population dynamics.

INTRODUCTION

Herbicide resistant weeds are a growing problem in Australian cropping systems and across the globe (Heap 2007). Using lower herbicide doses in cropping systems has been suggested as an attractive economic option (Blackshaw *et al.* 2006) but field-grown pot experiments have shown that using lower than recommended rates of diclofop-methyl can result in faster development of resistance to that herbicide in annual ryegrass (*Lolium rigidum* Gaudin) (Neve and Powles 2005). Is it possible that increased rates of development of herbicide resistance could also result from low rates of herbicide application in the field?

Simulation modelling of weed population dynamics has previously been used to predict how different management strategies will influence the rate at which herbicide resistance evolves in the field (e.g. Diggle *et al.* 2003, Neve *et al.* 2003). However, this modelling has focused on monogenic resistance, where a high degree of resistance is conferred by the presence of a single allele of a single gene. The resistance that evolved in the pot studies is almost certainly polygenic resistance, where significant resistance is the result of the additive or multiplicative affect of a number of minor alleles that each individually has a relatively minor effect (Neve and Powles 2005).

In this paper we present a modelling approach for predicting the possibility of weeds developing polygenic resistance. The modelling approach taken is a stochastic individual-based approach that

explicitly takes into account the fact that weed populations consist of individual organisms each of a particular genotype, and that some genotypes may be very rare or absent in a particular population. This model shows that using lower than recommended doses of herbicide could lead to increased rates of development of herbicide resistance in the field.

MODELLING APPROACH

The overall model dynamics are illustrated in Figure 1. A number of weed seeds exist in a dormant weed seedbank at the start of each season. Each seed in the weed seedbank then has an 80% chance of germinating and becoming established, otherwise it remains dormant. Each of these established weed seedlings then has a 20% chance of surviving management practices conducted before or at crop seeding. A post-emergent herbicide is then applied. The chance of a weed seedling surviving this spray depends on the spray rate and on the resistance status of the seedling, which in turn depends on its genotype. Seedlings that survive this post-emergent herbicide are assumed to reach maturity and produce seed. The number of seeds produced depends on the density of the weeds and the crop and the genotype of each of the seeds produced depends on the relative proportions of the genotypes of the weeds setting seed. Produced seed is added to the pool of dormant seeds to create the seedbank for the start of the next season.

Genotype and resistance status are represented individually for each weed seed or plant. There are several options for specifying the relationship between genotype and resistance status. The number of genes involved in resistance can be set to be one (monogenic) or any number greater than one (polygenic). A particular individual can then have zero, one or two 'resistance' alleles present at each gene (locus). The effect at each locus can be set to be dominant (one allele has the same effect as two), recessive (one allele has no effect) or intermediate (one allele has a partial effect). The effect at each locus can then combine either additively (linearly) or multiplicatively (non-linearly), to give a number R that represents the resistance status of that

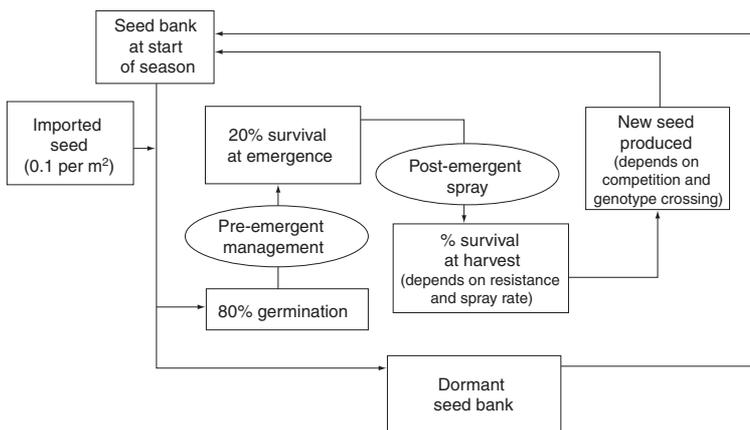


Figure 1. Illustration of the basic model structure.

genotype. The maximum strength of the resistance (the R value for individuals with all possible resistance alleles) must also be specified.

The chance of a weed surviving the post-emergent herbicide application depends on the spray rate and the resistance status R of the individual weed, according to a family of logistic dose response curves, as illustrated in Figure 2. Note that the R value thus corresponds to the ratio between the LD_{50} (dose needed for 50% kill rate) for the individual's genotype and the LD_{50} for the completely susceptible genotype, a commonly used measure of resistance status.

The amount of seed produced is calculated using the hyperbolic competition function commonly used in weed population models (Diggle *et al.* 2003), and depends on the crop and weed densities. This function also gives the crop yield as a percent of the maximum. The genotype of each of these seeds is then chosen by randomly choosing a father and a mother from the weed population, and then randomly choosing an allele from the mother and an allele from the father at each relevant locus.

The area simulated by the model is 10,000 m² and the initial ryegrass seedbank density is assumed to be 100 m⁻², so the initial seedbank population is always one million seeds. The initial resistance allele frequency for each gene (locus) related to resistance must be specified at the beginning of a model run and the initial seedbank is then set up according to these specified initial frequencies.

MODEL RUNS

The model shows that in the absence of herbicide resistance post-emergent kill rates greater than 88% lead to a sustainable system where ryegrass numbers decline each year. This kill rate can be achieved by spray rates as low as 70% of the recommended rate. Even

spray rates as low as 65% control weeds adequately for 20 years. So why would a farm manager spend more money to spray the full recommended rate? Does the possibility of herbicide resistance make a difference?

To consider this question we ran a series of 18 virtual experiments using the model described above. For each experiment we assumed a particular relationship between genotype and resistance status and a particular initial allele frequency. For

each experiment we then considered five spray rates (70, 80, 90, 100, 110 and 120% of the recommended rate) and ran four repetitions at each of these rates. With each of these repetitions the simulation was run over twenty years, and a number of output variables were recorded for each year. These included the weed density at harvest, the crop yield, and the proportion of resistant weeds in the population.

The genetics considered in the 18 experiments included weak and strong dominant monogenic resistance. We also considered weak and strong polygenic (two, three or five genes) resistance, including recessive, co-dominant and dominant effects, and both linear and multiplicative gene interactions. Finally, we considered cases where full resistance could be conferred by either a single major gene or a combination of minor genes. A summary of the 18 experiments is given in Table 1.

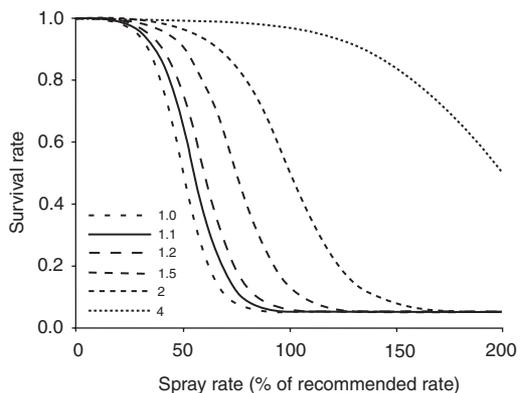


Figure 2. Chance of weeds surviving the post-emergent herbicide application for different spray rates and different resistance statuses ($R = 1, 1.1, 1.2, 1.5, 2, 4$ from the left curve to the right (dotted) curve).

In each experiment we examined whether there was a clear distinction between lower and higher spray rates in terms of the number of years before significant levels of resistance developed. This was defined to be when weed densities at harvest were above 200 m⁻².

RESULTS AND DISCUSSION

Consider Figure 3, which shows results from Experiment 16, where resistance depends on one very rare dominant major gene and four relatively common co-dominant minor genes. It is clear in this example that the lower the spray rate, the faster the weed density at harvest rises above 200 m⁻².

Lower herbicide rates leading to less sustainable systems in terms of herbicide resistance was found clearly in 13 of the 18 experiments. In another two, experiments 7 and 9, there was some complexity or variability, but on average higher rates were still significantly better. In another, experiment 8, results were not clear, but indicated higher rates could be better. The only experiments where weed populations did not reach uneconomic levels faster for lower rates were experiment 1 (strong monogenic resistance) and experiment 5 (two dominant genes combining linearly to give strong resistance), where there was no difference.

Further modelling work needs to be done to clearly identify the range of situations where low rates lead to faster development of herbicide resistance. The model has been written in a modular way to facilitate future planned developments to account for possible fitness penalties, seedbank death, mutation rates, phytotoxicity effects of herbicides on crops, season variability and different emergence times. We also plan to use the model to address polygenic resistance to pre-emergent herbicides and other issues in managing herbicide resistance. However, the work presented in this paper clearly shows that applying lower-than-recommended rates of post-emergent herbicides could lead to faster evolution of herbicide resistance in the field.

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Table 1. Summary of the 18 virtual experiments. From left to right, columns show experiment number, number of genes assumed to be involved in resistance, the maximum level of resistance, dominance (dominant, co-dominant or recessive), whether genes combine linearly or multiplicatively, and the initial allele frequency (IAF).

Exp	Genes	MaxR	Dominance	Combination	IAF
1	1	20	Dominant	—	10 ⁻⁵
2	1	21.5	Dominant	—	10 ⁻⁵
3	2	20	Dominant	Linear	10 ⁻⁵
4	2	20	Dominant	Multiplicative	10 ⁻⁴
5	2	20	Co	Multiplicative	10 ⁻²
6	2	1.5	Co	Linear	10 ⁻²
7	2	20	Recessive	Multiplicative	10 ⁻²
8	3	20	Dominant	Multiplicative	10 ⁻³
9	3	20	Co	Multiplicative	10 ⁻²
10	3	1.5	Co	Linear	0.015
11	3	2	Co	Linear	10 ⁻³
12	3	2	Co	Linear	0.05
13	3	20	Recessive	Linear	0.1
14	5	20	Recessive	Linear	0.2
15	5*	4	Dom/ Co	Linear	10 ⁻⁵ /10 ⁻¹
16	5*	4	Dom/ Co	Linear	10 ⁻⁵ /10 ⁻²
17	5*	4	Dom/ Co	Linear	10 ⁻⁵ /10 ⁻³
18	4*	4	Dom/ Co	Linear	10 ⁻⁵ /10 ⁻¹

*One major dominant gene that gives full resistance and three or four co-dominant genes where alleles can combine additively to give full resistance when all are present. IAF is for major/minor genes.

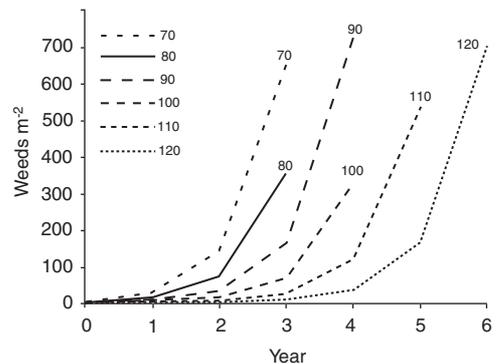


Figure 3. Experiment 16 results: weed densities increasing over time for different spray rates (as percentage of recommended rate: 70, 80, 90, 100, 110, 120% from the left to the right as labelled – repetitions are barely distinguishable).

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