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Simulation modelling identifies polygenic basis of herbicide resistance in a weed population and predicts rapid evolution of herbicide resistance at low herbicide rates

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ABSTRACT

The potential for low rates of diclofop-methyl to result in rapid evolution of herbicide resistance in a herbicide-susceptible *Lolium rigidum* (annual ryegrass) population was demonstrated in a recent crop-field study. In this present study, the data from the crop-field study was used together with simulation modelling to identify possible genetics of the herbicide resistance that was selected for. This analysis clearly indicated that the herbicide resistance was polygenic. Subsequently, the estimated genetic possibilities were used to parameterise a model of herbicide resistance evolution in a simulated crop-field situation, and the potential of different rates of diclofop-methyl (ACCase herbicide) to cause herbicide-resistance evolution in *L. rigidum* was explored and compared using the calibrated model. The calibrated model outputs indicated that the evolution of diclofop-methyl resistance would generally be faster at low herbicide rates than at higher rates due to the rapid selection of minor gene herbicide resistance traits at low rates and their subsequent recombination by cross-pollination. The results of the study therefore indicate potential risks in herbicide rate cutting and highlight the need for careful scientific evaluation of any herbicide use rate for its potential to select for minor gene herbicide resistance from a weed population.

1. Introduction

In global agriculture, crop-infesting weed species are a ubiquitous and important challenge and constraint to crop yield and quality. The potential crop yield losses due to weeds are higher than for any other pest species (Oerke, 2006). In most parts of the world, crop-infesting weeds are controlled by herbicides. In response to widespread and persistent herbicide usage, evolved herbicide resistance in crop weed species is occurring worldwide (Heap, 2011; Powles and Yu, 2010). There are many biological, genetic, herbicide and operational factors driving the dynamics of herbicide resistance evolution in weed species (Jasieniuk et al., 1996). Here we are concerned with the effect of herbicide use rates (selection intensity) on the dynamics of resistance evolution. It is self-evident that herbicide use rate will affect the percentage mortality of a targeted plant population (Bravin et al., 2001; Hidayat and Preston, 2001; Powles et al., 1998). At high herbicide use rate there will be very high weed mortality (95–100%) and thus the legal, registered rate of a herbicide, when correctly applied at the right weed growth stage, causes very high weed mortality. Conversely, if applied at a lower rate, there will be more weed survivors. The legal registered herbicide rate can vary from country to country and there are examples in global agriculture where herbicides are used at lower rates (Zhang et al., 2000). At lower rates, while the majority of a target weed population is killed, there is a percentage of the population that survives and goes on to produce viable seed. It is widely believed that use of low herbicide rates can contribute to herbicide resistance evolution in weed plants, although there has been limited evidence of this (Neve, 2007).

Some weed species have biological and other characteristics that confer many evolutionary advantages. *Lolium rigidum* (Gaud.)

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is an example of such a major herbicide resistant weed (Burnet et al., 1994; De Prado et al., 1997; Gill, 1995; Heap and Knight, 1986; Powles and Howat, 1990; Powles and Matthews, 1992; Preston et al., 2009). Herbicide resistance in *L. rigidum* is widespread in several countries including Australia, Chile, France, Iran, Israel, Italy, Saudi Arabia and the USA (Heap, 2011) and results from both target site and non target site resistance mechanisms (De Prado et al., 2005; Powles and Yu, 2010). Recent work with this highly genetically variable cross-pollinated species provides evidence that selection with low rates of herbicides can result in rapid herbicide resistance evolution (Busi and Powles, 2009; Manalil et al., 2011; Neve and Powles, 2005a,b). This may be because low rates of herbicides select for any minor herbicide resistance mechanisms, which are then combined by cross-pollination. However, the genetic basis underlying the resistance observed in these studies has not yet been fully determined. Moreover, it is yet to be proven experimentally that lower herbicide rates cause faster evolution, and while previous modelling work has indicated the possibility of lower herbicide rates causing faster evolution for certain genetic scenarios (Renton et al., 2011), this is yet to be shown for any genetic scenario based on experimental data.

In this study, we seek to address these two issues by using a computer model, known as the PERTH (Polygenic Evolution of Resistance To Herbicides) model (Renton et al., 2011). To simulate the evolutionary dynamics of herbicide resistance in *L. rigidum*, PERTH was first modified to simulate a crop-field study of resistance evolution under low herbicide rates (Manalil et al., 2011). A series of runs of this modified version was used to simulate the crop-field study under a wide range of hypothesised genetic scenarios in order to identify possible genetic scenarios that resulted in model predictions that matched the observed patterns of resistance evolution. Subsequently, the PERTH model was parameterised with a number of these identified possible genetic scenarios and then used to predict the evolution of herbicide resistance evolution within a range of different herbicide rates, in order to address the question of whether lower herbicide rates cause faster evolution of herbicide resistance.

2. Methods

2.1. Experimental data for model calibration

A crop-field experiment demonstrated the evolution of herbicide resistance when a susceptible population of *L. rigidum* WALR1, which had not previously been subjected to herbicides, was subjected to two cycles of selection by low rates of diclofop-methyl (a common grass herbicide used to control this weed) in a wheat field (Manalil et al., 2011). This field experiment was conducted at the field station of the University of Western Australia. This site is at least 100 km away from cropping regions and was chosen to avoid any herbicide resistant gene flow from commercial growing regions. The first cycle of field selection was at 281 g diclofop-methyl ha^{-1} (75% of the Australian registered rate) in 2006. There was high mortality after herbicide spray; however 5% of these plants survived although they showed symptoms of herbicide spray. Periodic inspections were conducted to ensure that there was not a flush of unsprayed *L. rigidum*. The *L. rigidum* survivors grew to maturity to produce seed progeny 1F. The next generation was selected again in the field at the Australian registered rate of diclofop-methyl (375 g diclofop-methyl ha^{-1}) and that resulted in the 2F population. In May 2008, progeny 1F and 2F were compared with the original unselected herbicide susceptible *L. rigidum* in a diclofop-methyl dose-response study. The LD_{50} values obtained for the 1F and 2F progeny were used in the model calibration in this study. For full details on this experiment see Manalil et al. (2011).

2.2. PERTH model

For the simulation study, the above mentioned field study (hereinafter referred to as 'the selection experiment') was simulated using the PERTH herbicide resistance model (Renton, 2009; Renton et al., 2011). The general PERTH model structure (please see Fig. 1 for model dynamics) simulates a winter cropping farming system, as practiced in much of Australia. The model uses a stochastic individual-based approach, where each weed seed and plant is represented explicitly. The model has been parameterised to represent *L. rigidum* grown in a wheat crop, based on Diggle et al., 2003 and Pannell et al., 2004 (Table 1). PERTH was used to first simulate the selection experiment to explore the possible genetics of herbicide resistance that evolved in that study, and second to use the estimated genetic parameters from the selection experiment to simulate broad-scale field conditions to explore the effects of a range of diclofop-methyl rates on herbicide resistance evolution. The second component uses the model structure and dynamics described in Renton et al. (2011), with parameters values as shown in Table 1, to represent broad-scale crop-field conditions, and the reader is referred to that paper for full details of the model assumptions and dynamics in this case. The simulation of the smaller-scale selection experiment in the first component was done by modifying key parameters and a few aspects of the dynamics of the PERTH model as described below (see Section 2.3).

For the purposes of this paper, the important model parameters are the ones that define the genetics basis of resistance. It is assumed that there are a certain number (ng) of potential unlinked resistance genes in a *L. rigidum* population, with three genetic possibilities at each locus (homozygous susceptible, heterozygous, or homozygous resistant) and thus 3^{ng} possible genotypes ranging from fully susceptible (homozygous susceptible at every locus) to fully resistant (homozygous resistant at every locus). The herbicide resistance level of each *L. rigidum* plant depends on its individual genotype, and therefore the overall herbicide resistance level of

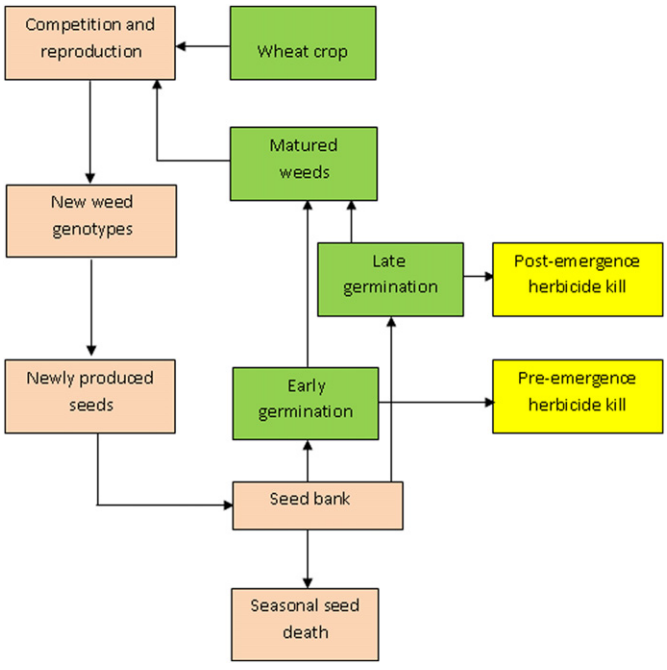

Fig. 1. Flow chart showing dynamics of the model.

Table 1
Descriptions, values and units of model parameters.

Explanation	Parameter	Values
Initial seed bank	*isb*	100 m^{-2}
Area of the field	*area*	100,000 m^2
Summer death of seeds	*sd*	10%
Winter death of seeds	*wd*	20%
Germination before sowing	*germ1*	40%
Germination after sowing	*germ2*	40%
Survival after pre-emergent herbicide and sowing	*pre-surv*	10%
Probability of weed completely escaping herbicide	P_{ut}	5%
Variability parameter	*var*	20%
LD_{50} of susceptible *L. rigidum*	LD_{50}	69 g diclofop-methyl ha^{-1}
Number of genes	*ng*	Variable
Maximum resistance	*Rmx*	12–22
Crop sowing density	*Pm′*	150 m^{-2}
Inter species antagonism factor	*a*	1.3
The crop plant size coefficient	*kp′*	1/11
The weed plant size coefficient	*Kp*	1/33
Maximum weed seed production per unit area	*Psp* max	30,000/m^{-2}

a *L. rigidum* population will be a function of genotypic frequency. Genotypic frequency is initialised at the beginning of the simulation based on an assumed initial resistance allele frequency. After this initialisation, genotypic frequency will change with each generation as the simulation runs, depending on the surviving genotypes (after herbicide selection) and cross-pollination.

The chance of survival of a *L. rigidum* plant treated with diclofop-methyl is mainly a function of three factors: the presence or absence of herbicide resistance genes in an individual, the herbicide application rate and the LD_{50} of a fully susceptible *L. rigidum* population. The parameter *thresh* is defined to be the threshold effective diclofop-methyl rate that is just sufficient to kill a completely homozygous susceptible *L. rigidum* plant and the parameter *Rmx* (maximum resistance level) is defined so that $Rmx \times thresh$ is the theoretical threshold effective rate that would be just sufficient to kill a completely homozygous resistant *L. rigidum* plant. This means that *thresh* is the LD_{50} of a fully susceptible *L. rigidum* population and *Rmx* is the ratio between the resistance level of a completely homozygous susceptible plant and the resistance level of a completely homozygous resistant plant (Renton et al., 2011).

2.3. Model adaptation for simulating the selection experiment

The PERTH model was originally designed to simulate selection for herbicide resistance in a normal farming scenario, so some changes to the model dynamics and the parameters presented in Table 1 were required to simulate the selection experiment. For example, in the experiment, *L. rigidum* seeding was carried out along with the wheat crop, and all *L. rigidum* seed produced was hand harvested, while in the base PERTH model weed seed germinates from an existing seedbank and new weed seed produced is returned to the seedbank. In order to simulate the selection experiment, 100,000 seedlings of parent herbicide susceptible population (representing WALR1) and then 5000 1F seedlings were treated with 281 and 375 g diclofop-methyl ha^{-1} respectively to simulate the two cycles of herbicide selection as in the field experiment. The plants that survived diclofop-methyl treatment were assumed to flower evenly, randomly cross-pollinate, and produce seeds that were 'collected', thus providing two simulated *L. rigidum* populations matching the two actual populations resulting from the experiment. To simulate the actual dose response evaluation of these two populations, three replicates of 50 seeds were randomly chosen from the simulated seed pools of each of the two herbicide selected populations and also the original unselected *L. rigidum* population. These were then assumed to germinate, application of 375 g diclofop-methyl ha^{-1} was simulated, and the number of survivors recorded. Thus we obtained the predicted percentage survival for the three populations (one unselected, two selected) based on the assumed genetics.

2.4. Factorial analysis to determine the possible genetics of resistance

We then conducted a factorial analysis to identify possible genetics that could explain the patterns of resistance evolution observed in the selection experiment. A large number of potential hypothetical combinations of genetic parameter values (as described below) were evaluated in order to ascertain which of these combinations of parameter values could have resulted in the observed data. The fit between the model and the selection experiment data was evaluated by comparing the mean survival percentage observed in the field experiment with the simulated mean survival percentage (at 375 g diclofop-methyl ha^{-1}). In all cases, we assumed that resistance was semi-dominant and additive across genes such that each resistance allele present contributes equally to the total resistance level.

The parameters that were varied and tested were the number of genes involved in resistance (*ng*), the initial allele frequency (*iaf*), and the maximum resistance (*Rmx*). We simulated a range of *Rmx* values from 12 to 22 (11 values). As mentioned above, the diclofop-methyl rate that results in mortality of a completely homozygous resistant *L. rigidum* plant is $thresh \times Rmx$ (Renton et al., 2011). The LD_{50} of the reference susceptible population WALR1 is 69 g diclofop-methyl ha^{-1}, and we can assume that this value is a good approximation of *thresh*. Therefore the *Rmx* values 12 and 22 roughly correspond to threshold mortality doses of 800 and 1500 g diclofop-methyl ha^{-1} for completely homozygous resistant plants. For each of these eleven *Rmx* resistance values we then tried a range of *ng* values: one, two, three, four or five, and for each combination of *Rmx* value and *ng* value, a range of possible values for initial allele frequency (*iaf*) was then tested through simulation. Through this factorial analysis, we identified possible combinations of number of resistance genes, initial gene frequency, and maximum resistance that resulted in simulated survival percentages matching the actual observed survival percentages at 375 g diclofop-methyl ha^{-1} in all three populations (the once- and twice-selected and unselected *L. rigidum* populations).

Table 2
Eleven possible genetics identified by increasing the parameter value for maximum resistance (12–22).

Scenarios	Maximum resistance (*Rmx*)	Number of genes (*ng*)	Gene frequency (*iaf*)
1	12	2	0.02
2	13	2	0.02
3	14	3	0.02
4	15	3	0.02
5	16	3	0.01
6	17	4	0.02
7	18	4	0.02
8	19	4	0.01
9	20	4	0.01
10	21	5	0.02
11	22	5	0.02

2.5. Running the calibrated model to simulate field conditions

Eleven possible genetic scenarios were identified through the factorial analysis (Table 2), each one corresponding to a different *Rmx* value. These eleven possible genetic scenarios were then used to simulate the impact of different herbicide rates on the evolution of diclofop-methyl resistance in a more realistic crop-field situation (area = 10 ha, non-genetic parameters set to their values in the original model as in Table 1). For this analysis, the model was run for up to 40 years with a series of assumed doses of diclofop-methyl, starting from 70 percent of the Australian recommended rate up to 200 percent. The herbicide rates used were 263, 300, 338, 375, 563, 656 and 750 g diclofop-methyl ha^{-1}. For each of these assumed herbicide rates, and for each possible genetic scenario from Table 2, several model outputs were recorded.

2.6. Model outputs recorded

The recorded model outputs included the weed density at harvest corresponding to the *L. rigidum* survivors after the diclofop-methyl treatment (weeds per m^2). Another output recorded was the percentage of 'resistants', that is, the percentage of weed plants at harvest that would have a greater than 25% chance of surviving the selective herbicide if it was applied at its standard rate; the threshold of 25% represents a weed survival rate at which a herbicide is no longer providing effective control. The model output 'resistance allele frequency' is the frequency of resistance alleles present, expressed as a percentage of the total possible alleles in the *L. rigidum* population at harvest.

3. Results and discussion

3.1. Possible genetics of herbicide resistance

Fig. 2(a–d) shows the comparison between the simulated versus actual survival of susceptible and field selected *L. rigidum* populations, when tested at the Australian registered rate of diclofop-methyl (375 g diclofop-methyl ha^{-1}), based on an assumed maximum resistance (*Rmx*) value of 12. The four sub-Figures (Fig. 2a–d) illustrate simulated survival results for each possible gene number (1–4), respectively, for a range of initial resistance gene frequencies. These Figures show that the simulated survival for the susceptible and selected populations matched best with the actual survival when the gene number was two at an initial allele frequency (*iaf*) of 0.02 (for both the genes) (Fig. 2b). For one, three and four genes a good match could not be obtained over the range of initial gene frequencies examined. Initial gene frequencies above 0.1 and below 0.01 were also examined for gene numbers (*ng*) (1–4) to rule out any further matching genetic possibilities at this *Rmx* level of 12. Similar Figures were generated to identify possible combinations of initial allele frequency and

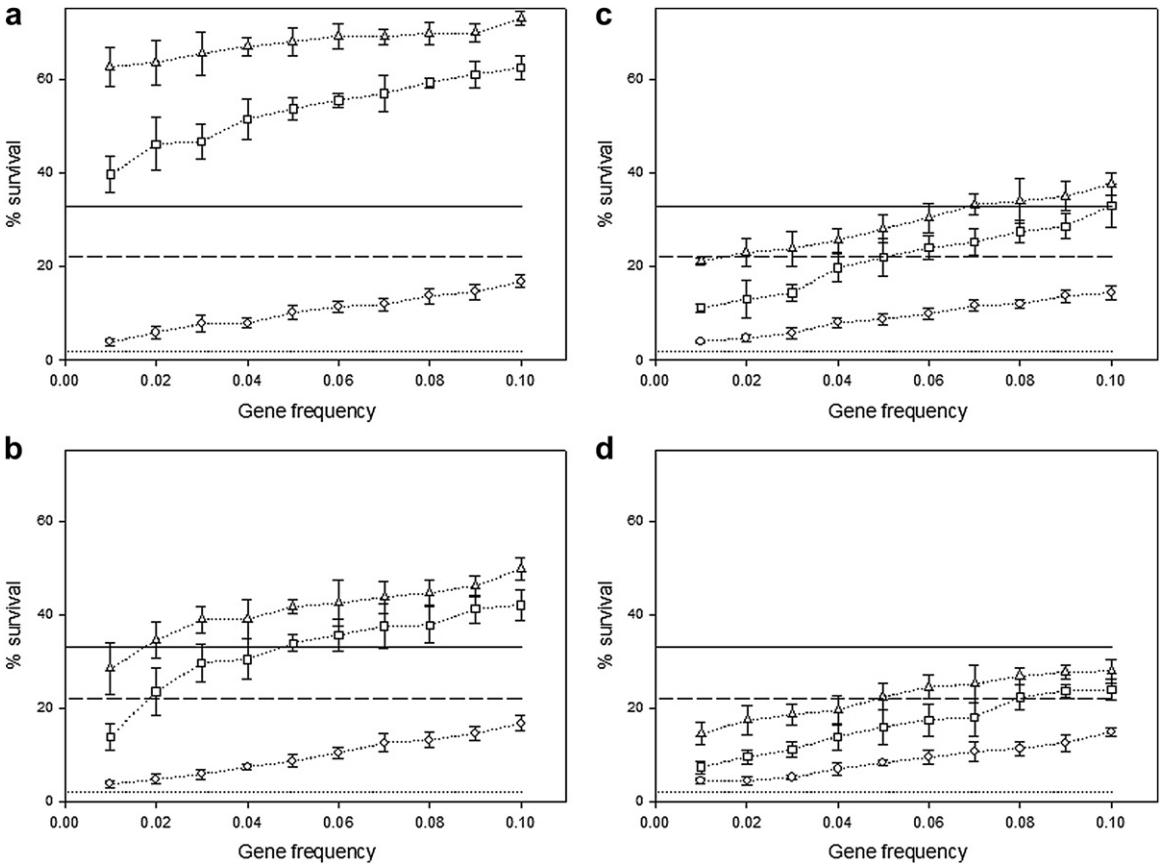

Fig. 2. Sensitivity analysis on initial resistance gene frequency at a maximum resistance value of 12. Graphs a, b, c and d represent model runs corresponding to one gene, two genes, three genes or four genes respectively. The straight lines parallel to the *x*-axis indicates the actual mean survival ($n = 3$) of the unselected WALR1 (dotted line), 1F (broken line) and 2F (solid line) at 375 g diclofop-methyl ha^{-1}. WALR1 (broken line, open circle), 1F line (broken line, open square) and 2F (broken line, open triangle) represent the simulated survival. The symbols represent the mean value of 10 model runs ($n = 10$) of the simulated survival for each gene frequency. Error bars are ±one standard deviation from the mean. Graph b represents a possible matching scenario (simulated and actual survival) gene frequency (0.02) for two genes.

gene number/s for the rest of the *Rmx* values (13–22) (not shown). For none of the eleven scenarios was it possible to find a scenario where one gene was able to result in a match with the observed data (Table 2), indicating the probable involvement of at least two genes in herbicide resistance evolution in this situation. We initially suspected that stacking of two alleles at a single locus might provide an explanation of the data, but we failed to find a scenario where a semi-dominant monogenic basis provided a reasonable explanation. We did consider a very wide range of possibilities representative of the range of levels of resistance observed in naturally evolved resistance populations, and we can be confident that within this range all possible cases have been identified. Interestingly, for this particular experiment and population, the data could only be explained by higher *Rmx* as the number of genes increased. Table 3 shows how the simulated resistance gene frequencies and the frequencies of different genotypes change over the three generations when genetic scenario 1 is assumed. The results indicate an increase in the proportion of the resistant genotypes (%) in the selected populations with a concomitant increase in the resistance gene frequency. There was no completely homozygous resistant plant present in the original population, but recurrent selection resulted in an increase in the proportion (%) of the completely homozygous resistant genotype from its initial value of zero to 0.01 and 0.35 in the first and second *L. rigidum* populations respectively (Table 3).

3.2. Likely role of polygenes in herbicide resistance evolution at low rates

This study demonstrates how simulation modelling can be used to identify possible genetic scenarios underlying the evolution of herbicide resistance observed in selection experiments or trials. An important conclusion of this analysis is the probable involvement of polygenes in the evolution of herbicide resistance in *L. rigidum* at low rates of diclofop-methyl selection. The polygenes at their initial frequency (Table 3) imparted low levels of herbicide resistance in the parent *L. rigidum* population due to a high proportion of the fully susceptible genotype, low levels of intermediate genotypes and the complete absence of the most highly resistant genotypes (Table 3). However, selection at low rates of diclofop-methyl resulted in a rapid increase in the proportion of resistant genotypes. This included the low-level-resistant genotypes that were present in low frequencies in the initial population, but also included more highly resistant genotypes, such as the completely homozygous resistant genotype, that were not present in the original *L. rigidum* population (Table 3). Importantly, this indicates that selection at low rates of diclofop-methyl allowed genotypes with low and intermediate levels of resistance to survive, with subsequent recombination of resistance genes by cross-pollination thus resulting in novel genotypes with higher levels of resistance. The observed increase in herbicide resistance level of *L. rigidum* plants in the field would thus appear to have been a result of both a) differential rates of survival between fully susceptible genotypes and genotypes with low and intermediate levels of resistance, and b) sexual recombination resulting in the appearance of novel genotypes with higher levels of resistance than any genotypes found in the initial population.

3.3. Effect of diclofop-methyl rates on model outputs

Simulated weed densities of *L. rigidum* (at harvest) following selection at different rates of diclofop-methyl, assuming genetic scenario 1 (Table 2), are presented in Fig. 3. Similarly to the actual crop-field experiment, there were high *L. rigidum* densities at low rates, and understandably, density decreased with an increase in herbicide use rates. *L. rigidum* density increased faster for low diclofop-methyl rates of 263, 300 and 338 g ha^{-1} compared to the Australian registered rate (375 g ha^{-1}). Increasing the herbicide rate further continued to reduce the rate of increase of weed density in the simulated crop-field environments. A very similar trend in the way that weed density changed over time with varying levels of diclofop-methyl was observed for all the other 11 genetic scenarios tried (data not shown).

The model output 'percentage of resistants' followed trends similar to weed density (Fig. 4). The proportion of resistant weeds increased faster at lower rates. This was true when rates were lower than the registered Australian rate, although, the difference was greater as herbicide rates increased above the registered Australian rate. The same trend in 'resistants' increasing slower with higher levels of diclofop-methyl was observed for all the other 11 genetic scenarios tried (data not shown).

Simulated resistance allele frequencies of *L. rigidum* (at harvest) are shown in Fig. 5. The rate of increase in resistance allele

Table 3
The simulated genotypic frequency of the susceptible line WALR1 and selected populations 1F and 2F indicating a progressive increase in resistant genotypes in the low rate diclofop-methyl selected *L. rigidum* populations for scenario 1 (two resistance genes with semi-dominance).

Genotypes[a]	Genes[b]		Frequency (%)		
	R1	R2	WALR1	1F	2F
1	0	0	92.24	56.06	18.53
2	1	0	3.8	18.69	22.10
3	2	0	0.04	1.55	6.70
4	0	1	3.72	17.91	20.86
5	1	1	0.17	4.08	19.31
6	2	1	0.00	0.17	3.05
7	0	2	0.03	1.36	6.21
8	1	2	0.00	0.18	2.89
9	2	2	0.00	0.01	0.35
			Freq R1 [c] = 0.02	Freq R1 [c] = 0.13	Freq R1 [c] = 0.32
			Freq R2 [d] = 0.02	Freq R2 [d] = 0.12	Freq R2 [d] = 0.31

Freq R1 [c] & Freq R2 [d] indicate the resistance gene frequency for the three *L. rigidum* populations.

[a] Nine genotypes corresponds to a two gene scenario (with an equal resistance allele frequency of 0.02).

[b] R1 and R2 corresponds to the two resistance genes, the values 0, 1 and 2 indicate zero, one and two (homozygous) resistance alleles in the genotype.

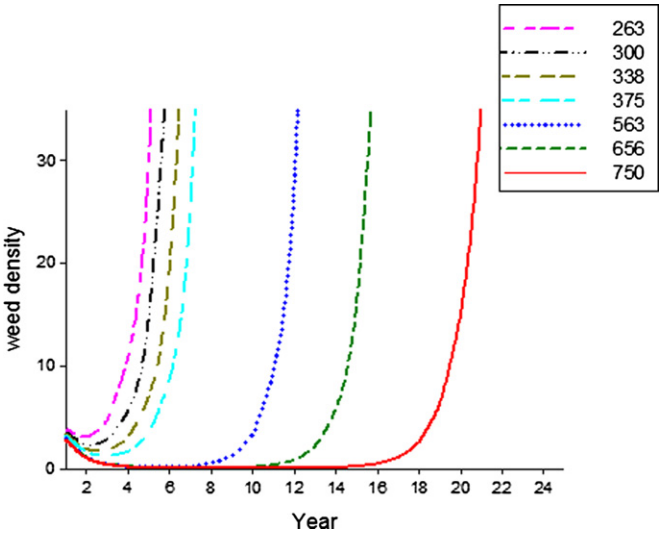

Fig. 3. The simulated weed density of *L. rigidum* at harvest corresponding to scenario 1 (two resistance genes with semi-dominance) following continuous application of low to high rates of diclofop-methyl in a wheat crop.

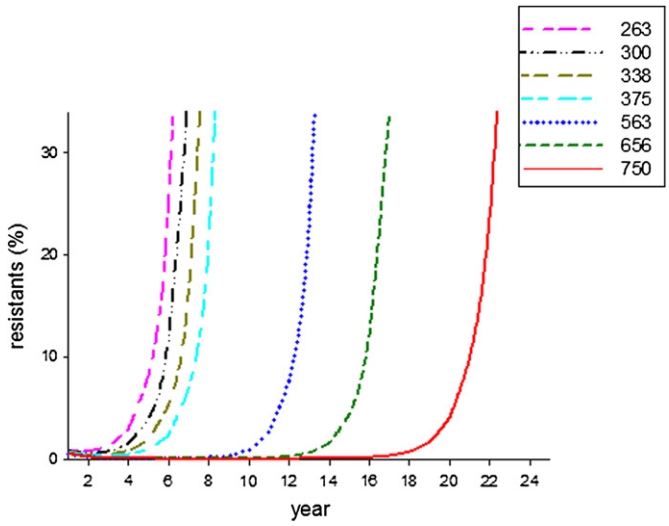

Fig. 4. The simulated 'resistants' (resistant weeds with >25% chance of surviving selective herbicide) corresponding to scenario 1 (two resistance genes with semi-dominance) following continuous application of low to high rates of diclofop-methyl in a wheat crop.

frequency was generally slower with increasing herbicide rates. Unlike the model outputs for weed density and resistant weeds (resistants), differences in resistance allele frequency between the registered Australian diclofop-methyl rate and lower rates were not clearly observed. However, a clear reduction in resistance allele frequency was observed for rates higher than the registered Australian diclofop-methyl rate.

3.4. High herbicide rates slow resistance evolution mediated by polygenic resistance traits

Importantly, when calibrated to the possible genetics matching the real trial data, the simulations of long-term broad-scale crop-field situations showed faster increase in weed population densities at lower rates of diclofop-methyl. The different model outputs indicated that this was partly due to increase of the proportion of resistant weeds (resistants) (Fig. 4) as diclofop-methyl rate increased from low to high. There was also reduction in the rate of

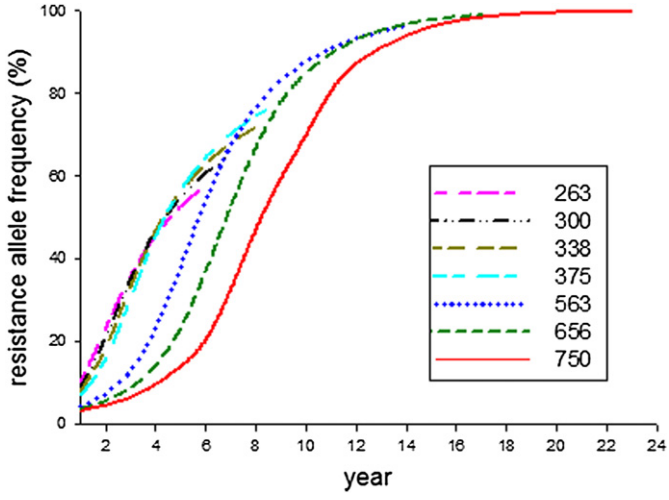

Fig. 5. The simulated resistance allele frequency of *L. rigidum* at harvest corresponds to scenario 1 (two resistance genes with semi-dominance) following continuous application of low to high rates of diclofop-methyl in a wheat crop.

increase of the proportion of resistance alleles (Fig. 5) as diclofop-methyl rate increased from lower rates to higher rates.

Crop models are used in agriculture to make better weed management decisions (Benjamin et al., 2010; Colbach and Philippe, 1998; Dauer et al., 2007; Diggle et al., 2003; Doole and Pannell, 2008; Gardner et al., 1998; Jasieniuk and Maxwell, 1994; Madsen and Streibig, 2000). Modelling studies exclusively addressing strong monogenic herbicide resistance have suggested that lower herbicide rates result in weaker selection and thus slower increase in frequency of resistance genes (reviewed by Jasieniuk et al., 1996, Diggle and Neve, 2001). The modelling work presented by Renton et al. (2011) confirmed that higher herbicide rates can increase the speed of development of monogenic resistance, but also showed that this effect is probably offset by higher kill rates, and thus that herbicide rate is likely to have little effect on weed density and cropping system sustainability in the case of monogenic resistance. Nonetheless, theoretically there could be an ideal herbicide rate that is not low enough to select for accumulated low-level polygenic resistance traits but not high enough to result in more rapid herbicide resistance evolution due to strong monogenic herbicide resistance traits. Accurately identifying such herbicide rates through modelling would require knowledge of the genetic basis of the relevant herbicide resistance mechanisms, but when such knowledge is available or when reasonable assumptions can be made, this kind of modelling could provide insight into the precise herbicide application rate that should be followed in a herbicide management system to delay resistance as long as possible, or avoid it completely. This kind of analysis would also need to take into account the fact that low rates are likely to select for polygenic traits that are of particular concern because they are more likely to confer cross-resistance to other herbicides than monogenic target-site resistance (Neve and Powles, 2005b).

4. Conclusion

Eleven possible genetic scenarios were identified that could result in simulated evolution of herbicide resistance that matched the herbicide-resistance evolution observed in *L. rigidum* in a field trial under selection at low rates of diclofop-methyl. All these identified genetic scenarios were polygenic. Simulations of crop-field conditions indicated more rapid evolution of herbicide resistance at lower rates of diclofop-methyl for all the possible identified genetic scenarios, because low herbicide rates selected and accumulated (by cross-pollination) the low-level herbicide resistance traits present in the original population. Overall, this modelling study warns against herbicide rate-cutting and indicates the need for careful consideration of herbicide rates.

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