Modelling the effects of ecology, management and genetics on the evolution of herbicide resistance with PERTH

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Summary The way weeds evolve resistance to herbicides depends on a complex interaction between the underlying genetics, the weed management used by the farmer, and the biology of the weed species. PERTH (Polygenic Evolution of Resistance To Herbicides) is an individual-based model created to simulate these complex interactions and predict the rates and patterns of resistance evolution in a wide range of different conditions. PERTH can help evaluate the efficacy of management options such as herbicide rotation, maintaining robust herbicide rates at high efficacy, and using non-chemical methods of control such as strategic tillage, and how the efficacy of these options depends on the genetics underlying the resistance. PERTH can also help predict how biological or ecological factors can affect the evolution of resistance, including factors such as seed bank longevity and dormancy, fecundity, seed dispersal, and rates of self-fertilisation versus out-crossing.

Keywords Herbicide, resistance, weeds, rotation, rate, dose, genetics, genetics, polygenic, monogenic, ecological traits.

INTRODUCTION

Weeds are a major problem in Australian cropping systems, greatly exacerbated by the increasing prevalence of evolved herbicide resistance (Heap 2012). As weeds evolve resistance, previously effective herbicide options lose efficacy and then fail. Growers must then find new weed control options that are more expensive, less convenient or less environmentally benign; adapt their farming system to include options that are less reliant on the herbicides in question; or in some cases adopt new systems entirely to deal with this problem. Rather than wait until resistance has evolved, it is preferable to look for management options that may help avoid or delay the evolution of resistance.

However, predicting the evolution of resistance under different management options is difficult, because the way weeds evolve resistance to herbicides depends on a complex interaction between the underlying genetics, the weed management used by the farmer, and the biology of the weed species. Moreover, it is a long-term problem that results from rare mutations in huge populations, so experimental results based on short-term studies of relatively small populations are always of limited value. Simulation modelling provides a tool for integrating existing knowledge to predict how these complex interactions will occur across realistic spatial scales and time periods, and thus evaluate the likely efficacy of different management options (e.g. Maxwell *et al.* 1990, Diggle *et al.* 2003, Thornby and Walker 2009).

PERTH (Polygenic Evolution of Resistance To Herbicides) is an individual-based model created to simulate the complex interactions between the underlying genetics, the weed management used by the farmer, and the biology of the weed species in order to predict the rates and patterns of resistance evolution in a wide range of different conditions (Renton *et al.* 2011, Renton 2012). In this paper, we describe how PERTH has been used to help evaluate the efficacy of management options such as herbicide rotation, maintaining robust herbicide rates at high efficacy and using non-chemical methods of control such as strategic tillage, and how the efficacy of these options depends on the genetics underlying the resistance. We also show how PERTH has been used to help predict how biological or ecological factors can affect the evolution of resistance, including factors such as seed bank longevity and dormancy, fecundity, seed dispersal, and rates of self-fertilisation versus out-crossing.

THE MODEL

The PERTH model is an individual-based simulation model that tracks the population of weed plants across single growing seasons and the population of weed plants across multiple years (Renton *et al.* 2011 for details). The overall model dynamics are illustrated in Figure 1. At the beginning of a model run an initial population of weed seeds is created using specified initial resistance allele frequencies, while in subsequent years the weed seed population carries over from the previous year. In either case, the model represents a collection of weed seeds existing in a dormant weed seed bank at the start of each season. A number of cohorts are simulated through the year. For each cohort, any seed currently in the seed bank has a certain chance of germinating, emerging and becoming established; otherwise it remains in the seed bank. Any established weed seedlings will face certain weed management, such as a pre-emergent herbicide, a post-emergent in-crop selective herbicide, or soil cultivation, depending on which cohort it is part of. Later cohorts will not be affected by the pre-emergent herbicide for example. Soil cultivation also affects the seed bank, possibly killing seeds, but usually just moving them within the soil. Soil residual herbicides may also be simulated, and these will cause mortality to seeds germinating at relevant times and locations in the soil. Any weed that germinates and survives all weed management that it faces, then sets seed.

Genotype and resistance status are represented individually for each weed seed or plant, and all individuals of the same resistance genotype will have the same resistance status. The total number of genes involved in resistance can be set as one (monogenic) or any number greater than one (polygenic). The maximum strength of resistance (the resistance status value for individuals with all possible resistance alleles) must also be specified. The gene effect at each locus can be set as dominant (one allele has the same effect as two), recessive (one allele has no effect) or intermediate (one allele has a partial effect). The gene effects from each locus can be set to combine additively (with neutral or no epistasis), antagonistically (negative epistasis) or synergistically (positive epistasis).

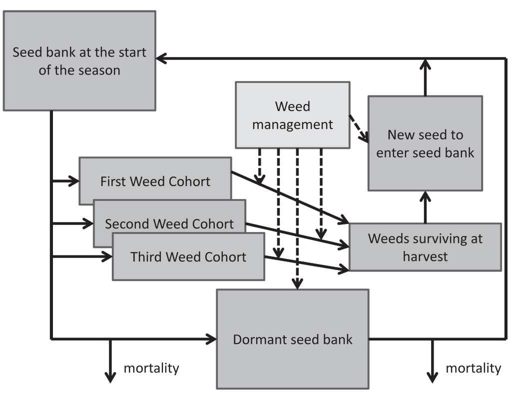

Figure 1. The overall dynamics of the model.

In a simulated population, each particular individual has zero, one or two 'resistance' alleles present at each gene (locus), and *R,* the resistance status of that individual (and its genotype), depends on which alleles are present, according to the specified maximum resistance, epistasis and dominance. More complicated relationships between genotype and resistance can be specified if required, to represent multiple resistance statuses regarding multiple herbicides with partial cross-resistance for example (as described elow). Each individual has a certain chance of surviving a management event. For non-chemical management, this probability will usually be set to be the same for all individuals (independent of an individual's genotype and resistance status), and this may also be used for some herbicides, if we are not interested in evolution of resistance to that particular herbicide. However, for the herbicide(s) of interest, the chance of mortality can be set to be dependent on the individual's resistance status, so that more resistant weeds are more likely to survive the herbicide application. In this case, the effective herbicide dose received by an individual weed is assumed to vary around the target application dose, and is then simply divided by its resistance status (*R*). This procedure results in a family of logistic dose response curves for different genotypes, 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; this ratio is a commonly used empirical measure of resistance status.

The amount of weed seed produced is calculated using the hyperbolic competition function (Firbank and Watkinson 1985) commonly used in weed population models (e.g. Diggle *et al.* 2003). This depends on

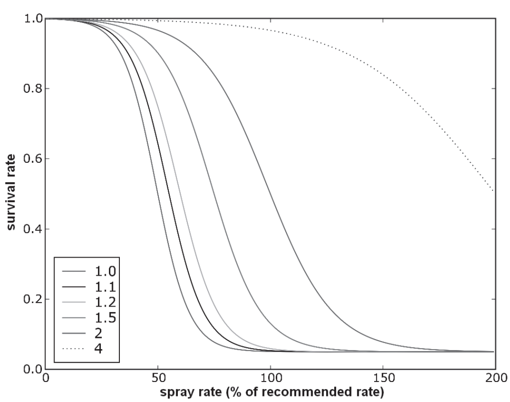

Figure 2. Chance of weeds of different resistance statuses (R = 1, 1.1, 1.2, 1.5, 2, 4) surviving different herbicide spray rates.

the relative competitiveness of the weed and crop, the crop density and the density of the surviving weeds. This function also gives the crop yield as a percent of the maximum possible yield. The genotype of each of the new seeds is chosen by randomly selecting a father and a mother from the weed population, and randomly choosing one allele from the mother and one allele from the father at each relevant locus.

The area to be simulated and the initial seed bank density are set at the beginning of a model run, and size of the initial seed bank population is then the product of these two numbers. The initial resistance allele frequency for each gene (locus) related to resistance is also specified at the start of each run and the initial seed bank is then set up according to these initial frequencies. As the simulation continues, the frequencies of allele and genotypes and the total weed population fluctuates according to the management applied and the biological processes simulated. Simulation is generally continued for a specified numbers of years, or until weed populations or crop yield penalties reach a specified critical threshold that makes continued cropping not feasible.

APPLICATIONS

The PERTH model has now been applied to investigate a number of herbicide resistance issues.

Herbicide rate The original version of the PERTH model was designed to address the question of whether using herbicides at rates that were lower or higher than a standard registered rate could hasten or delay the evolution of resistance to the herbicide. This study found that the evolution of resistance to an in-crop selective herbicide could be significantly hastened by reducing herbicide rates, but only for certain cases where the underlying resistance genetics was effectively polygenic (Figure 3a; Renton *et al.* 2011).

Weed biology The PERTH model was adapted to investigate how the biological or ecological characteristics of a weed species affected the chance of it evolving resistance to a herbicide. The main change to the model was allowing for different mating systems i.e. the degree of outcrossing versus self-fertilisation. Other changes included adding a greater number of plant cohorts within a year, to allow different patterns of dormancy and germination across and between years. In addition we investigated the effect of mortality rates, fecundity, competitiveness and phenotypic variability. We also considered interactions with the usage pattern of the herbicide (pre-sowing versus in-crop) and different underlying genetics. Results show that ecological traits have a strong influence on the evolution of resistance, and this influence depends significantly on genetics and herbicide usage pattern (Figure 3b).

Tillage effects The PERTH model was adapted to investigate whether no-tillage systems were likely to lead to faster evolution of resistance to soil-residual herbicides applied with sowing, compared to more traditional systems based on full-cut or mouldboard tillage. We also investigated whether strategic use of soil inversion with a mouldboard plough could delay resistance by burying resistant seed and thus 'resetting the resistance clock'. The main change to the model was dividing the seed bank into a number of soil layers to represent surface, shallowly-buried and deeply-buried seeds. Other changes included incorporating soil-residual herbicides that only affect germinating seeds into the model. We also considered interactions with different underlying genetics, and biological characteristics such as seed dormancy and mortality rates. The model predicts that no-tillage systems do not increase the rate at which resistance evolves, and that strategic use of soil inversion could delay resistance by burying resistant seed and also by bringing susceptible seed back to the surface (Figure 3c). Efficacy of the strategy is affected by underlying genetics, dormancy and mortality rates to some degree.

Optimal herbicide rotation The PERTH model was adapted to investigate optimal herbicide rotation strategies for soil-residual herbicides. This was motivated by the current availability of new herbicide options, and the question of whether these should be immediately included in weed management strategies as an alternative to the currently used soil-residual herbicide to reduce selection pressure, or instead reserved for when resistance to the currently used soil-residual herbicide has emerged. The model had already been adapted to represent soil-residual herbicides, as mentioned above, so the main change to the model was extending the link between genotype and resistance status to allow the model to simulate the evolution of resistance to a number of herbicides simultaneously, while accounting for different fitness penalties and different levels of possible cross-resistance to these herbicides. The study predicts that rotating all available herbicides can extend weed management sustainability compared to using herbicides one by one until they are no longer effective, but the difference is very small, and the particular rotation strategy used has very little overall effect (Figure 3d).

CONCLUSION

PERTH is an individual-based model that simulates the complex interactions between the genetics underlying

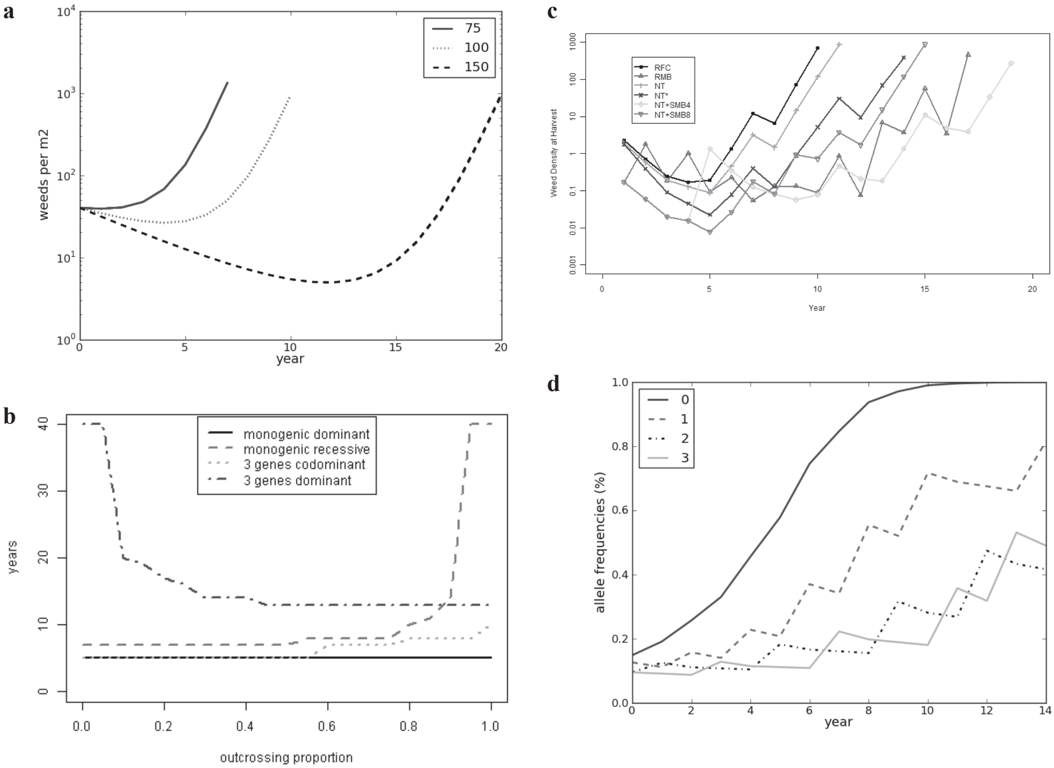

Figure 3. Examples of PERTH output illustrating the different applications discussed in this paper, showing a) weed populations changing over time under different herbicide rates, b) number of years to evolve resistance to an in-crop selective herbicide for four genetic scenarios across different levels of outcrossing, c) weed populations changing over time under different tillage strategies, d) resistance allele frequencies changing over time under different herbicide rotation strategies.

herbicide resistance, the weed management used by the farmer, and the biology of the weed species in order to predict the rates and patterns of resistance evolution. PERTH can help evaluate the efficacy of management options and predict how genetic, biological and ecological factors can affect the evolution of resistance.

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REFERENCES

Diggle, A., Neve, P. and Smith, F. (2003). Herbicides used in combination can reduce the probability of herbicide resistance in finite weed populations. *Weed Research* 43, 371-382.

Firbank, L.G. and Watkinson, A.R. (1985). On the analysis of competition within two-species mixtures of plants. *The Journal of Applied Ecology* 22, 503-517.

Heap, I. (2012). The International Survey of Herbicide Resistant Weeds. www.weedscience.com Accessed 15/5/12.

Maxwell, B., Roush, M. and Radosevich, S. (1990). Predicting the evolution and dynamics of herbicide resistance in weed populations. *Weed Technology* 4(1), 2-13.

Renton, M., Diggle, A., Manalil, S. and Powles, S. (2011). Does cutting herbicide rates threaten the sustainability of weed management in cropping systems? *Journal of Theoretical Biology* 64, 360-365.

Renton, M. (2012). Shifting focus from the population to the individual as a way forward in understanding, predicting and managing the complexities of evolution of resistance to pesticides, *Pest Management Science*. doi: 10.1002/ps.3341

Thornby, D. and Walker, S. (2009). Simulating the evolution of glyphosate resistance in grains farming in northern Australia. *Annals of Botany* 104, 747-756.