Does cutting herbicide rates threaten the sustainability of weed management in cropping systems?

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Abstract

Evolution of herbicide resistance in weeds is a growing problem across the world, and it has been suggested that low herbicide rates may be contributing to this problem. An individual-based simulation model that represents weed population dynamics and the evolution of polygenic herbicide resistance was constructed and used to investigate whether using lower herbicide rates or standard rates at reduced efficacy could reduce the sustainability of cropping systems by causing faster increases in weed population density as herbicide resistance develops. A number of different possible genetic bases for resistance were considered, including monogenic resistance and polygenic resistance conferred by several genes. The results show that cutting herbicide rates does not affect the rate at which weed densities reach critical levels when resistance is conferred exclusively by a single dominant gene. In some polygenic situations, cutting herbicide rates substantially reduces sustainability, due to a combination of faster increase in resistance gene frequency and reduced kill rates in all genotypes, while in other polygenic situations the effect is small. Differences in sustainability depend on combined strength of the resistance genes, variability in phenotypic susceptibility and rate delivered, level of control due to alternative measures, and degree of genetic dominance and epistasis. In the situation where resistance can be conferred by both a single dominant major gene or a number of co-dominant minor genes in combination, the difference made by low rates depends on the relative initial frequency of the major and minor genes. These results show that careful consideration of herbicide rate and understanding the genetic basis of resistance are important aspects of weed management.

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1. Introduction

“Do you think that using low rates of herbicides will reduce the sustainability of weed management by causing faster evolution of herbicide resistance?” Recently this question was asked to an audience of eminent international weed scientists at a conference. About half the audience raised their hands to answer “yes”, while the other half shook their heads to indicate “no”. It seems that there is still no clear answer to this perennial question, probably due to the long time scales involved, the impracticality of very large-scale experimentation, spatial and temporal variability, a lack of understanding of underlying population genetics and the inherent complexity of the problem.

There is no doubt that the evolution of herbicide resistant weeds is a growing problem across the globe (see www.weedscience.com, Heap, 2011), and it seems possible that this problem is being exacerbated by the practise of reducing herbicide rates. It has been suggested that the evolution of polygenic resistance is likely to be faster at lower rates of herbicide or other pesticide, due to reduced kill rates of intermediate resistant individuals and subsequent accumulation of resistance alleles producing highly resistant individuals, particularly in cross-pollinated species (e.g. Christoffers, 1999), and modelling work using quantitative genetics approaches has supported this hypothesis (Gardner et al., 1998; Gressel, 2002; Groeters and Tabashnik, 2000; Shaw, 1989). Glasshouse experiments have shown that using lower-than-recommended rates of the selective herbicide diclofop-methyl can result in rapid evolution of resistance to that herbicide in annual ryegrass (Lolium rigidum Gaudin) (Neve and Powles, 2005a,b), as well as cross-resistance to other herbicides (Manalil et al., 2011). High rates typically select for major gene inherited dominant target site or reduced translocation resistance whereas low rates tend to select for metabolic resistance, often
resulting in much broader resistance to herbicides with different modes of action (Neve and Powles, 2005a,b; Busi and Powles, 2009; Powles and Yu, 2010). Similar results have been shown for glyphosate (Busi and Powles, 2009) and in the field for diclofop-methyl (Manalil et al., 2011), although none of these experiments have directly compared the rate of resistance evolution under two or more different herbicide rates. In addition to concerns that low rates of herbicide resistance will hasten the evolution of herbicide resistance, low rates also increase the risk of ineffective or variable control (Doyle and Stypa, 2004).

Conversely, there may be advantages to be gained from reducing herbicide rates. Using lower herbicide rates can be an attractive economic option because of reduced input costs, at least in the short term (Blackshaw et al., 2006). Applying herbicides at significantly reduced rates can result in kill rates that are only slightly lower than the kill rate achieved at the full regulation rate, and can thus maintain good ongoing weed control (Friesen et al., 2000). Under an economics analysis, even if low rates reduce longer-term sustainability, any longer term losses from more rapid evolution of herbicide resistance would have to exceed the short term saving in dollar value in order to offset them, while taking into account economic discounting. It has also been argued that selection pressure for monogenic herbicide resistance may be decreased by using lower rates (e.g. Friesen et al., 2000). There are also many concerns regarding the environmental effects of high herbicide rates (Blackshaw et al., 2006; Kudsk and Streibig, 2003; O'Donovan et al., 2007).

There is often substantial difference in the rate of a particular herbicide that is regulated or recommended by different countries. For example, the regulated rate (label rate) for diclofop-methyl control of the same weed species in various countries ranges between 375 g ha\(^{-1}\) in Australia, 640 g ha\(^{-1}\) in the United States, 500 g ha\(^{-1}\) in France, 560 g ha\(^{-1}\) in Argentina, 850 g ha\(^{-1}\) in India and 925 g ha\(^{-1}\) in China (Bayer, 2010). In some countries with low regulated rates, it may be common to use rates that are even lower than regulated rates for the above-mentioned economic reasons, and factors such as mistiming herbicide application, poor application technique, or environmental stress on plants can result in effective rates being still lower than the nominal reduced rate applied. For example, in Australia, which has some of the lowest regulated rates in the world, rates are often cut still lower in practise and drought stress can reduce the effective rate still further, which has been suggested as one reason among many why the incidence of herbicide resistance is higher in Australia than other countries (Gardner et al., 1998; Neve and Powles, 2005a,b). However, herbicide efficacy varies between environments due to factors such as crop density and weed characteristics, so it is unclear whether and to what extent these differences in regulated rates translate into real differences in effective application rate at the level of individual weeds and thus into real differences in levels of control.

There is a long history of using simulation modelling of genetics and population dynamics to predict how different management strategies will influence the rate at which resistance to biocides evolves for various organisms, including weeds, insects, fungal pathogens and bacteria (Cavan et al., 2000; Comins, 1977, 1979, 1986; Crowder et al., 2006; Diggle et al., 2003; Gardner et al., 1998; Gressel and Segel, 1990; Groeters and Tabashnik, 2000; Jaffe et al., 1997; Jasieniuk and Maxwell, 1994; Josepovits and Dobrovolsky, 1985; Levy et al., 1983; Maxwell and Mortimer, 1994; Neve, 2008; Neve et al., 2003b; Peck, 2001, Richter et al., 2002; Roush and Daly, 1990; Roux et al., 2008; Shaw, 1989, 1993, 2000; Thornby and Walker, 2009; van den Bosch and Gilligan, 2008; Vendete and Chini, 1999). Much of 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. However, for cross-pollinated weed species, reducing herbicide rates may be more likely to decrease the sustainability of weed control systems in the polygenic case, where significant resistance (beyond the ability of typical rates to give satisfactory control) is the result of the additive or multiplicative effect of a number of minor alleles that each individually has a relatively minor effect. While it is commonly assumed that polygenic resistance is likely to be rare in the field (Roush and McKenzie, 1987) there is now evidence that non-target-site resistance based on mechanisms such as increased metabolism or reduced translocation, which are likely to be polygenic, may be much more common in the field than previously thought (Delye et al., 2007, Dinelli et al., 2008). Polygenic resistance can also be more problematic than monogenic resistance because organisms with quantitative resistance often have cross-resistance to totally unrelated chemicals (Gardner et al., 1998).

Some previous modelling has focussed on the relationship between pesticide rate and build-up of resistance, and has thus considered polygenic resistance (Birch and Shaw, 1997; Gardner et al., 1998; Groeters and Tabashnik, 2000; Shaw, 1989, 2000). However, these studies have usually followed other previous modelling work in using a deterministic population density approach based on quantitative genetics assumptions, which implicitly assumes an infinite population (Holst et al., 2007), and thus neglects the possibility of rare genotypes or alleles being eliminated from the population; this may be of particular importance with polygenic resistance where the number of possible genotypes is relatively large and thus individual genotype frequencies are likely to be relatively small. The reality of the modelled herbicide-weed systems may also violate some of the assumptions of these approaches, such as the absence of linkage disequilibrium (Diggle et al., 2003). More recently developed stochastic individual-based approaches that represent populations as finite (Diggle et al., 2003; Neve et al., 2003a), have been used to give new insight into questions regarding the probability and rate of developing herbicide resistance, but these approaches have not yet been used to address polygenic resistance. Groeters and Tabashnik (2000) used an individual-based approach with a finite population to represent insects, with a population that was assumed to remain constant over generations and complex agent-based multi-focus models have been used to study the emergence of resistance to biocides in theoretical systems (e.g. Jaffe et al., 1997). However, individual-based population dynamics approaches have not been applied to represent the interaction between changes in population density and the evolution of polygenic resistance to herbicides in weeds. There is therefore a clear need to return to the question of low herbicide rates and to use individual-based modelling with a focus on polygenic resistance and population dynamics to examine this issue in more detail and in a new light.

This paper addresses this need by presenting an individual-based model constructed to predict the possibility of cross-pollinated weeds developing both monogenic and polygenic resistance, and describes how this model was used to address the question of low rates. The approach taken is stochastic and explicitly takes into account the fact that weed populations consist of a finite number of individual organisms each of a particular genotype, and that some genotypes may thus be very rare or completely absent in a particular population. It also explicitly represents the fact that each individual receives a particular rate of herbicide, but that this rate will vary between individuals. In this way it captures more of the real complexity of the interactions between herbicide rates, genetics, kill rates, density-based competition, population dynamics and variation between individuals than a more traditional deterministic approach based on the frequency of resistance alleles within a population or previous individual-based approaches that represented only monogenic resistance. We then use the new model to investigate whether
and in what situations low rates can cause faster or more frequent evolution of resistance, to what extent this affects the sustainability of cropping systems and how other factors, such as the number of genes involved in herbicide resistance and the strength of the resistance, affect these issues.

2. Methods

2.1. Model dynamics overview

The overall dynamics of the Polygenic Evolution of Resistance To Herbicides (PERTH) model are illustrated in Fig. 1. The model represents the life-cycle of a cross-pollinated annual weed in an annual seasonal cropping system, such as the winter-cropping summer-fallow system common in Australia. A number of weed seeds exist in a weed seedbank at the start of each season. A small number of weed seeds may then be added to the seedbank, to represent seed being introduced for example by wind. Each seed in the weed seedbank then has a chance of germinating and becoming established before the crop is sown. Each of these established weed seedlings then has a chance of surviving pre-emergent management (management that occurs before and during the sowing of the crop). Seeds that have not germinated by this point may remain dormant through the rest of the season or may die during the season. A post-emergent herbicide is then applied, and each weed has a chance of surviving this spray. Weeds that survive the post-emergent herbicide application are assumed to reach maturity and produce seed. Thus the model includes two weed cohorts that experience different management strategies; pre-emergent management affects the first cohort, while post-emergent management (selective herbicide) affects the second cohort and the survivors from the first cohort. The number of weed seeds produced depends on the density of both 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 producing seed. Produced seed is added to the pool of ungerminated weed seeds remaining from the start of the season and all seeds in this pool then have a chance of dying before the break of the following season. Surviving seed in this pool then makes up the weed seedbank at the start of the next cycle.

The genotype of every seed and weed in every pool in the PERTH model is represented separately. The parameters and variables used in the model are summarised in Tables 1 and 2.

Fig. 1. Graphical representation of the dynamics of the model.

2.2. Modelled system

We chose model parameter values to represent annual ryegrass (Lolium rigidum), an important weed in global cropping systems that has evolved resistance to many herbicides (Burnet et al., 1994; Heap, 2011). For simplicity, and in order to focus on the reduced rates issue that is the focus of this paper, we chose to model a continuous cropping system with a single crop each year. Every year, the same pre-emergent management is applied, wheat is the crop, and the same selective herbicide is applied in-crop. Only evolution of resistance to the selective herbicide is modelled, efficacy of pre-emergent management is assumed to remain constant through a simulation. For the purpose of this paper, it does not matter what the actual selective herbicide is, or what the pre-emergent management is, as long as it is different from the selective herbicide. The model assumes a constant average season, so parameters such as seed death, pre-emergent kill rate, germination rates and competition parameters maintain a constant value from year to year.

2.3. Initial seedbank

Model initialisation requires defining the area simulated, area, and the initial weed seedbank density, den. The number of seeds in the initial seedbank is then simply area \times den. The initial resistance allele frequency, ia, for each gene (locus) related to resistance must also be specified at the beginning of a model run and the genotype of each seed in the initial seedbank is then generated according to these specified initial frequencies. For this study, the area simulated by the model was set to be 1,000,000 m² (10 ha) and the initial ryegrass seedbank density was assumed to be 100 m⁻², so the initial seedbank population was always 100,000,000 seeds, which was big enough to make the effects of stochastic variability relatively small. The initial resistance allele frequency ia was set to be 0.01 for all four genes in the default four gene example. This value was chosen because it results in resistance becoming obvious in approximately the same amount of time (approximately six years) as previous monogenic models of evolution of resistance to in-crop selective herbicides (Diggle et al., 2003).

2.4. Weed dormancy characteristics

The parameter values used to represent the dormancy and germination characteristics of the modelled ryegrass are based on
2.5. Death, survival, genetics and resistance

For the purpose of this paper, we focussed on resistance to post-emergent (selective) herbicides. Therefore, the chance of a seed dying during the season or during the break-of-season and the chance of a weed surviving pre-emergent management were all set to a constant value that was independent of the seed or weed genotype (Table 1). The genotype of a weed determines its level of resistance to the post-emergent herbicide, and the resistance level of a weed determines its chance of surviving the post-emergent spray, as we now explain. Note that although the model is explained in terms of weeds surviving or dying, a proportion of weeds dying has a similar effect to weeds surviving but with a penalised seed production. For example, 80% of weeds dying has a similar effect to all weeds surviving but producing 20% less seed (there will be less production. For example, 80% of weeds dying has a similar effect to weeds surviving but with a penalised seed production).

The number of genes or loci involved in resistance (\( ng \)) is a model parameter that can vary from one for monogenic resistance to any positive integer greater than one for polygenic resistance.

We assume that the weed is a cross-pollinated diploid, and that each allele is either a susceptibility (\( S \)) allele or a resistance allele (\( R \)), so that at each locus the possibilities are that there are either zero, one, or two \( R \) alleles. The genotype \( gt \) of a given individual can thus be represented as a list of length \( ng \)

\[
gt = [x_1, x_2, \ldots, x_{ng}] \tag{1}
\]

where each element of the list \( x_i \) is either 0, 1 or 2. For example, if three loci are involved in resistance, then \( gt = [1,0,2] \) represents a genotype with one \( R \) allele at the first locus, no \( R \) alleles at the second locus and two \( R \) alleles at the third locus. The number of possible genotypes is thus \( 3^{ng} \). Every individual in the model at a given time in the simulation has a genotype that is represented in this way, and whenever a seed germinates the resulting weed plant is of the same genotype as that specific seed.

In this model, the resistance level \( r \) of a weed is defined by stating that if a weed has a resistance level \( r \), then the rate of herbicide required to kill this weed is exactly \( r \) times the rate of herbicide required to kill a weed that is completely susceptible (one that has no \( R \) alleles at all). This resistance level \( r \) of a weed is then assumed to be a function of its genotype. The model was constructed to allow any relationship between genotype and resistance level, but in this paper we assume that the relationship between genotype and resistance depends on four model parameters: the threshold herbicide level to kill fully susceptible weeds \( \text{thresh}_R \), the threshold herbicide level to kill fully resistant weeds \( \text{thresh}_R \), dominance \( \text{dom} \) and epistasis \( \text{epis} \). The derived value \( Rmx \), which is the ratio of \( \text{thresh}_R \) to \( \text{thresh}_R \), represents the resistance level of a genotype that has all possible \( R \) alleles (is homozygous resistant) at all possible loci. \( Rmx \) can take any value greater than or equal to one. The parameter \( \text{dom} \) represents whether the genes involved are dominant (\( \text{dom} = 1 \)), recessive (\( \text{dom} = 0 \)) or partially dominant (\( 0 < \text{dom} < 1 \)). The parameter \( \text{epis} \) represents the way that genes at different loci combine, and can take any value. Negative values of \( \text{epis} \) represent negative or antagonistic epistasis, where the effect of two genes is less than twice the effect of one gene; positive values represent positive or synergistic epistasis, where the effect of two genes is more than twice the effect of one gene; and \( \text{epis} = 0 \) represents neutral or additive epistasis, where the effect of two genes is exactly the same as twice the effect of one gene. To be precise, if a genotype is written in the form of a list, as described above (Eq. (1)) then the

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Explanation</th>
<th>Value(s) used</th>
</tr>
</thead>
<tbody>
<tr>
<td>area</td>
<td>Simulated area</td>
<td>1,000,000 m² (10 ha)</td>
</tr>
<tr>
<td>den₀</td>
<td>Initial weed seedbank density</td>
<td>100 m⁻²</td>
</tr>
<tr>
<td>( P_{\text{post}} )</td>
<td>Probability of in-season seed death</td>
<td>0.1</td>
</tr>
<tr>
<td>( P_{\text{pre}} )</td>
<td>Probability of between-season seed death</td>
<td>0.2</td>
</tr>
<tr>
<td>chsprem</td>
<td>Probability of surviving pre-emergent management</td>
<td>0.1</td>
</tr>
<tr>
<td>germ₁</td>
<td>Probability of germinating before sowing</td>
<td>0.4</td>
</tr>
<tr>
<td>germ₂</td>
<td>Probability of germinating after sowing</td>
<td>0.4</td>
</tr>
<tr>
<td>ng</td>
<td>Number of genes involved in resistance</td>
<td>4</td>
</tr>
<tr>
<td>( iaf )</td>
<td>Initial resistance allele frequency</td>
<td>0.01</td>
</tr>
<tr>
<td>dom</td>
<td>Dominance</td>
<td>0.5</td>
</tr>
<tr>
<td>epis</td>
<td>Epistasis</td>
<td>0</td>
</tr>
<tr>
<td>( p_{\text{esc}} )</td>
<td>Probability of weed completely escaping herbicide</td>
<td>0.05</td>
</tr>
<tr>
<td>( sd_{\text{rate}} )</td>
<td>Rate variability parameter</td>
<td>0.4</td>
</tr>
<tr>
<td>( \text{thresh} )</td>
<td>Threshold herbicide level to kill fully susceptible weeds</td>
<td>20</td>
</tr>
<tr>
<td>( \text{thresh}_R )</td>
<td>Threshold herbicide level to kill fully resistant weeds</td>
<td>200</td>
</tr>
<tr>
<td>( Rmx )</td>
<td>Resistance level when all ( R ) alleles present</td>
<td>10</td>
</tr>
<tr>
<td>( d_c )</td>
<td>Crop sowing density</td>
<td>150 m⁻²</td>
</tr>
<tr>
<td>( k_c )</td>
<td>Crop size/competitiveness parameter</td>
<td>1/11</td>
</tr>
<tr>
<td>( k_s )</td>
<td>Weed size/competitiveness parameter</td>
<td>1/33</td>
</tr>
<tr>
<td>( SS_{\text{max}} )</td>
<td>Maximum weed seed production</td>
<td>30,000 m⁻²</td>
</tr>
<tr>
<td>( \text{pen}_{\text{in-crop}} )</td>
<td>Competitiveness penalty for weeds emerging in-crop</td>
<td>0.5</td>
</tr>
<tr>
<td>rate²</td>
<td>Selective herbicide rate applied</td>
<td>Various</td>
</tr>
</tbody>
</table>

a All rates written as percentage of recommended (label) rate.
b Note \( Rmx \) is a derived value rather than a parameter and is equal to \( \text{thresh}_R/\text{thresh}_R \).
c As the focus parameter of the paper, rate is only varied when all other parameter values have been fixed.

Table 2: Model variables.

---

2.5. Death, survival, genetics and resistance

For the purpose of this paper, we focussed on resistance to post-emergent (selective) herbicides. Therefore, the chance of a seed dying during the season or during the break-of-season and the chance of a weed surviving pre-emergent management were all set to a constant value that was independent of the seed or weed genotype (Table 1). The genotype of a weed determines its level of resistance to the post-emergent herbicide, and the resistance level of a weed determines its chance of surviving the post-emergent spray, as we now explain. Note that although the model is explained in terms of weeds surviving or dying, a proportion of weeds dying has a similar effect to weeds surviving but with a penalised seed production. For example, 80% of weeds dying has a similar effect to all weeds surviving but producing 20% less seed (there will be less production. For example, 80% of weeds dying has a similar effect to weeds surviving but with a penalised seed production).

The number of genes or loci involved in resistance (\( ng \)) is a model parameter that can vary from one for monogenic resistance to any positive integer greater than one for polygenic resistance. We assume that the weed is a cross-pollinated diploid, and that each allele is either a susceptibility (\( S \)) allele or a resistance allele (\( R \)), so that at each locus the possibilities are that there are either zero, one, or two \( R \) alleles. The genotype \( gt \) of a given individual can thus be represented as a list of length \( ng \)

\[
gt = [x_1, x_2, \ldots, x_{ng}] \tag{1}
\]

where each element of the list \( x_i \) is either 0, 1 or 2. For example, if three loci are involved in resistance, then \( gt = [1,0,2] \) represents a genotype with one \( R \) allele at the first locus, no \( R \) alleles at the second locus and two \( R \) alleles at the third locus. The number of possible genotypes is thus \( 3^{ng} \). Every individual in the model at a given time in the simulation has a genotype that is represented in this way, and whenever a seed germinates the resulting weed plant is of the same genotype as that specific seed.

In this model, the resistance level \( r \) of a weed is defined by stating that if a weed has a resistance level \( r \), then the rate of herbicide required to kill this weed is exactly \( r \) times the rate of herbicide required to kill a weed that is completely susceptible (one that has no \( R \) alleles at all). This resistance level \( r \) of a weed is then assumed to be a function of its genotype. The model was constructed to allow any relationship between genotype and resistance level, but in this paper we assume that the relationship between genotype and resistance depends on four model parameters: the threshold herbicide level to kill fully susceptible weeds \( \text{thresh}_R \), the threshold herbicide level to kill fully resistant weeds \( \text{thresh}_R \), dominance \( \text{dom} \) and epistasis \( \text{epis} \). The derived value \( Rmx \), which is the ratio of \( \text{thresh}_R \) to \( \text{thresh}_R \), represents the resistance level of a genotype that has all possible \( R \) alleles (is homozygous resistant) at all possible loci. \( Rmx \) can take any value greater than or equal to one. The parameter \( \text{dom} \) represents whether the genes involved are dominant (\( \text{dom} = 1 \)), recessive (\( \text{dom} = 0 \)) or partially dominant (\( 0 < \text{dom} < 1 \)). The parameter \( \text{epis} \) represents the way that genes at different loci combine, and can take any value. Negative values of \( \text{epis} \) represent negative or antagonistic epistasis, where the effect of two genes is less than twice the effect of one gene; positive values represent positive or synergistic epistasis, where the effect of two genes is more than twice the effect of one gene; and \( \text{epis} = 0 \) represents neutral or additive epistasis, where the effect of two genes is exactly the same as twice the effect of one gene. To be precise, if a genotype is written in the form of a list, as described above (Eq. (1)) then the
function $f$ for resistance used in this model is

$$r = f(g(t)) = f(x_1, x_2, \ldots, x_{ng}) = R_{max} \left( \frac{\sum_{k=1}^{ng} g(x_k)}{ng} \right)^{2^{sdrate}}, \quad (2)$$

where

$$g(x_k) = \begin{cases} 0 & \text{if } x_k = 0 \\ \text{dom} & \text{if } x_k = 1 \\ 1 & \text{if } x_k = 2 \end{cases} \quad (3)$$

An illustrative example of the way that the epistasis parameter $epis$ interacts with the number of $R$ alleles to affect resistance level $r$ is shown in Fig. 2.

The chance of a weed surviving post-emergent herbicide treatment depends on the level of resistance $r$ of the weed, the rate of herbicide application rate and a number of other model parameters, including the $thresh$, $sd_{rate}$, and $put$. In this paper, for the sake of generality, herbicide rate is usually presented as a percentage of some standard rate, which could represent the standard field application rate (recommended or label rate) for a given country. For example, if the field rate for an herbicide is actually 375 g ha$^{-1}$ then rate=80% means that the herbicide is applied at 300 g ha$^{-1}$. Similarly, an actual application rate of 520 g ha$^{-1}$ would be presented as 140%. The parameter $thresh$ represents the threshold herbicide rate that is just enough to kill a fully susceptible weed (no $R$ alleles) of an ‘average’ phenotype in an ‘average’ environment. We assume that even though herbicide is applied at a nominal rate rate, the actual effective rate received by individual plants will vary due to environmental and phenotypic variability, and variability in plant size and maturity, and the parameter $sd_{rate}$ represents this variability. To be more precise, the effective rate received by an individual plant will be a normally distributed random variable with mean rate and standard deviation $sd_{rate} \times rate$. The parameter $put$ represents the proportion of plants that completely escape (are completely untouched by) the herbicide, due to being covered or sheltered by other plants for example.

In summary, whether a given plant does or does not survive a selective herbicide application is thus decided in the model as follows. First, the plant has a $put$ chance of avoiding the herbicide application altogether. Otherwise, the genotype of the plant is translated to a resistance level $r$, according to Eqs. (1)–(3). An effective rate is stochastically generated for the plant, using the parameters rate and $sd_{rate}$. If this rate is greater than or equal to the threshold herbicide rate $thresh \times r$ then the plant dies, otherwise it survives. This process is carried out individually for every weed each time the selective herbicide is applied. The result of this process is that 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, as illustrated in Fig. 3.

Note that $r$ is a variable that describes the resistance level of an individual weed or a genotype, rather than a population. On the other hand, the standard meaning of $LD_{50}$ is the herbicide rate needed to kill 50% of a given weed population, which would normally consist of a mix of genotypes, and the standard meaning of the resistance level of a given population is the ratio between the experimentally determined $LD_{50}$ of that population and the experimentally determined $LD_{50}$ of some reference population. However, the parallel between $r$ as we have defined it and the standard concept of the resistance level of a population can be demonstrated as follows. If we

1. set $put$ equal to zero, as it would be in a controlled experimental situation,
2. generate a ‘resistant’ population consisting entirely of individual weeds of a genotype with resistance level $r=r_0$ where $r_0 > 1$,
3. generate a reference susceptible population of weeds consisting entirely of individuals without $R$ alleles, and thus of resistance level $r=1$,
4. simulate herbicide application as described in the previous paragraph for a range of herbicide rates for both these populations, and
5. use the results of the simulated herbicide application to calculate the $LD_{50}$ for both populations,

then the $LD_{50}$ for the susceptible population will be approximately equal to $thresh$, and the $LD_{50}$ for the resistant population will be approximately equal to $thresh \times r_0$. The ratio between these two $LD_{50}$ values, which corresponds to the standard concept of resistance level, will thus be approximately equal to $r_0$. Note that the simulated population resistance will only be approximately equal to $r_0$ because of the stochasticity and variability incorporated into the model. Moreover, simulated populations, like real populations, will usually consist of a mix of genotypes, each with a different resistance level $r$, and so the relationship between $r$ and population resistance level is complex.
2.6. Competition, seed production and seed genotype

The amount of weed seed produced is calculated using the hyperbolic competition function commonly used in weed population models (Diggle et al., 2003; Firbank, 1985; Firbank and Watkinson, 1985; Pannell et al., 2004), and depends on the crop and weed densities. The equation can be written as

\[
\text{seedset} = \frac{SS_{\text{max}} k_w a_w d_w}{1 + k_w a_w + k_c d_c},
\]

where seedset is the actual weed seed produced (m\(^{-2}\)), \(SS_{\text{max}}\) is a model parameter representing the theoretical maximum possible weed seed production (seeds m\(^{-2}\)), \(k_p\) is a model parameter representing the competitiveness of the weed, \(k_c\) is a model parameter representing the size or competitiveness of the crop species, \(d_w\) is the weed density (plants m\(^{-2}\)) and \(d_c\) is the crop density (plants m\(^{-2}\)). This function is also modified to give the crop yield,

\[
\text{cropyield} = \frac{\text{Yield}_{\text{max}} k_d d_c}{1 + k_w a_w + k_c d_c},
\]

and thus calculate \(pyield\), which is the crop yield as a percent of the potential weed-free crop yield, using the equation

\[
\text{yield} = \frac{1 + k_w a_w}{1 + k_w a_w + k_c d_c}. \tag{6}
\]

For this paper, we assume that the crop species is always wheat. The parameter values used to represent the competition of the modelled ryegrass and wheat are based on the published RIM model of ryegrass seedbank dynamics and are shown in Table 1 (Pannell et al., 2004).

Once the number of seeds produced has been determined, the genotype of each of these seeds is chosen by randomly choosing two parents from the weed population, and then randomly choosing an allele from one chosen parent and an allele from the other chosen parent at each relevant locus. Thus the number of \(R\) alleles at each locus in the new seed depends on the number of \(R\) alleles at the same locus in the parent plants (Table 3). It is assumed that there is no linkage between genes.

2.7. Model implementation

The model was implemented using the Python programming language (Python, 2009).

2.8. Stochasticity

At various points in the simulated life cycle, plants may either survive or die with a certain probability, effective herbicide rates are drawn from a random distribution, and parents and alleles are chosen at random from a number of possibilities. All of these processes occur stochastically and individually, which means that different results will occur each time the model is run. However, the area simulated was chosen to be big enough to ensure stochastic variability was relatively small, which was checked by carrying out three repetitions for every model run.

2.9. Model output examples

The model outputs various results, including yearly weed densities before pre-emergent management, before selective herbicide application, and at harvest; seedbank density at the start of the season; crop yield as a percent of the weed-free crop yield (\(pyield\)), which shows the yield penalty due to weeds; the percentage of all alleles in the weed population or seedbank that are \(R\) alleles; ‘proportion of resisters’—the percentage of weeds at harvest that have a greater than 25% chance of surviving the selective herbicide; the kill rate actually achieved with a given selective herbicide application; and the year of collapse of the system, which is defined to be the year in which \(pyield\) falls below 75%. This collapse is of the specific system we are analysing, continuous wheat cropping system based on the particular selective herbicide. For our purpose, simulations are halted when the system collapses, but in reality there would be a switch to a different herbicide or system. The resistance and collapse thresholds of 25% and 75% were chosen to represent levels that would be noticeable and of practical significance in the field.

In order to see the effects of changing herbicide rates, the model can be run for a number of different herbicide rates while holding all other parameters constant, and the output for the different rates compared. We compared three herbicide rates; a reduced rate (75% of label rate), a standard rate (100%) and an increased rate (150%). The reduced rate could represent a deliberately chosen lower application rate, or a standard rate at reduced efficacy due to ineffective application, mistiming or plant stress, while the increased rate could potentially represent a higher rate introduced by changed legislation, or simply improved application technique. First we ran the simulation, generated output and compared rates for the default parameter values (Table 1) describing weak four-gene polygenic resistance (\(threshR = 200\), \(Rmx = 2\), or a 2-fold resistance). We then chose some similar co-dominant polygenic examples with contrasting results to illustrate the range of possible outcomes. These included default parameter values but with a higher final level of resistance (\(threshR = 500\), 5-fold resistance); default parameter values but with a higher final level of resistance (\(threshR = 500\)) and a lower initial resistance allele frequency (\(iaf = 5 \times 10^{-5}\)); and default parameter values but with a higher final level of resistance (\(threshR = 500\)) and a greater number of genes (\(ng = 8\)). Finally we ran the simulation and generated output assuming strong rare dominant monogenic resistance (\(ng = 1, d = 1, threshR = 2000\) or 20-fold resistance, \(iaf = 10^{-6}\)).

2.10. Sensitivity analysis

As we were particularly interested in the way the strength of resistance, the number of genes and herbicide rate interact to affect the sustainability of the system, we conducted sensitivity
analyses by varying the $threshR$ parameter for cases where the number of genes conferring resistance was assumed to be one, two, three, four and five. For this analysis, all other model parameters were fixed to their standard values (Table 1). We then conducted further analyses on the model to investigate whether and to what extent the value of other model parameters would affect the relationship between herbicide application rate and the sustainability of the system. The model parameters that were varied for this analysis were $chsprem$, $dom$, $epis$, $iaf$, $thresh$ and $put$. Each of these parameters was considered separately, in six separate analyses. For simplicity, the genetic parameters $dom$, $epis$ and $iaf$ were each kept equal across all genes for this analysis. The particular parameter being considered in a given analysis was varied through a range of values, while all other parameters were held constant at default values. For each value of the parameter being considered, simulations were carried out assuming selective herbicide application rate was 75%, 100% and 150% of standard or regulated rate. The average year of collapse for each of the three rates was recorded. By plotting how the average year of collapse for the three rates changed with the changing parameter value we could then identify parameter combinations where low rates reduced sustainability, increased sustainability or made no real difference. Note that this 'sensitivity analysis' was conducted across a range of values as it was aimed at giving insight into how various factors interact with herbicide rate to affect sustainability in the modelled system, rather than the more restricted and common aim of sensitivity analysis of simply identifying which parameters have the greatest effect on model outcomes.

We also tested the sensitivity of results to other changes that we suspected would have little effect on the results. These included simulating the use of an alternative selective herbicide every second or third year, using a different crop with different herbicides every second or third year, assuming a fitness penalty for possession of resistance genes (simulated as a reduction in seed and pollen production) and varying the size of the initial weed seedbank.

2.11. Mixed resistance

We also considered the situation where there are two possible resistance mechanisms for conferring resistance to the one herbicide present in the same population: one conferred by a relatively rare dominant gene where the presence of a single allele confers effective resistance and one by several other relatively common co-dominant genes where each allele confers a relatively weak resistance. More precisely, we considered a situation based on the default parameters (four relatively common co-dominant genes conferring relatively weak resistance $iaf=0.01$, $threshR=200$), but then assumed that there was also a single rare dominant gene conferring strong resistance. This strong gene was based on the monogenic example described above ($ng=1$, $dom=1$, $threshR=2000$, $iaf=10^{-6}$). We then conducted a sensitivity analysis by examining the effect of varying $iaf$ for the major and minor genes on the sustainability of the system at various herbicide rates.

Fig. 4. Detailed results from simulating with all parameter values taking their standard values (Table 1), except that initial resistance allele frequencies have been set to 0.005, 0.01, 0.015, and 0.02, for the four genes (labelled 0,1,2, and 3, respectively). All graphs show model output values changing over time. Individual plots show (a) the percentage of weeds resistant (> 25% chance of surviving herbicide), the percentage of all alleles in the population that are $R$ alleles, and the yield as a percentage of weed-free yield; (b) the $R$ allele frequency for the four genes separately; (c) the seedbank density, separating genotypes that have only $S$ alleles, genotypes that have only $R$ alleles and genotypes that have a mix of $R$ and $S$ alleles; and (d) the weed densities before knockdown herbicide application, before selective herbicide application and at harvest.
3. Results

3.1. Detailed example model outputs

Fig. 4 illustrates detailed model output for a single herbicide rate (100% of standard rate). This example assumes the default parameter values given in Table 1, except that for illustration purposes the initial resistance allele frequencies have been set to different values (0.005, 0.01, 0.015 and 0.02), so that they can be distinguished in the figure. Note that in this and subsequent figures the results of three different runs are included, although in many cases the results for repetitions are very similar and thus cannot be distinguished visually in figures due to the relatively large population simulated.

3.2. Contrasting examples of effects of herbicide rate

Changing herbicide rate had a wide range of effects on the sustainability of the cropping system, and even changes to a single model parameter could significantly alter this effect (Figs. 5 and 6).

For the first example with polygenic co-dominant resistance (default parameter values, Fig. 5), the different herbicide rates make a clear difference to the rates at which weed numbers increase and a smaller difference to the rate at which the proportion of 'resistants' in the population increases. For the lowest rate, weed density is approximately constant at the beginning, and then begins to increase; for the intermediate rate, weed density decreases at first, and then begins to increase, while for the higher rates weed densities initially decrease substantially and then eventually begin to increase again.

A range of possible outcomes can be obtained by changing just one or two parameters (Fig. 6). Changing just one parameter (\(\text{threshR} = 500\), Fig. 6a) greatly reduces the difference made by the different herbicide rates on the rates at which weed numbers increase and the proportion of 'resistants' in the population increases. Making the same change in combination with another either results in similar patterns to the original values (\(\text{threshR} = 500, \text{iaf} = 5 \times 10^{-6}\), Fig. 6b) or in different rates making a clear difference to the rates at which weed numbers increase, but a less clear difference to the rate at which the proportion of 'resistants' increases (\(\text{threshR} = 500, \text{ng} = 8\), Fig. 6c). With strong monogenic dominant resistance (Fig. 6d), the different herbicide rates make no discernible difference to the rates at which weed numbers increase. Proportion of resistants increase slightly more quickly at higher herbicide rates, but the lower rate of kill at the lower herbicide rates cancels out this effect in terms of total weed population. Also note how the initial trend in weed density (increasing, decreasing or constant at time=0) varies with the different rates in different ways in the five examples.

3.3. Sensitivity analysis

Varying values of \(\text{threshR}\) (and thus \(\text{Rmx}\)) makes a large difference to how much of a difference in sustainability results from increasing or decreasing herbicide rates and herbicide rate
makes more difference when the number of genes is greater (Fig. 7). Varying other model parameters also has a large effect on the degree of difference between herbicide rates (Fig. 8), with the parameters controlling genetics and resistance (dom, epis, iaf, thresh) having more of an effect overall than other parameters (chsprem, put).
Tests of the sensitivity of results to other changes, including simulating the use of an alternative selective herbicide every second or third year; using a different crop with different herbicides every second or third year; assuming a fitness penalty for possession of resistance genes; and varying the size of the initial weed seedbank, showed that these factors did not interact with the effects of herbicide rate, and thus did not affect the answer to the question this research is focussing on (results not shown).

3.4. Mixed resistance

Fig. 9 shows the results of the sensitivity analysis on the ‘mixed resistance’ situation where there is a ‘strong’ gene and four ‘weak’ genes. Note how the difference between rates decreases as the initial frequency of the strong allele increases, and how the difference between rates also varies with the weak allele’s initial frequency. At the default initial allele frequencies, the major gene puts an upper bound on the year of collapse, which means changing rates makes little difference, but if the major alleles are rarer, or the minor alleles are more common, then differences in sustainability due to differences in rates are evident again.

4. Discussion

Our results indicate that reducing herbicide rates can potentially affect the sustainability of herbicide-based weed management in cropping systems to a significant degree. For example, for the modelled polygenic resistance situation with our assumed default parameter values (Fig. 5), cutting herbicide rate from 100% to 75% of standard label rate decreased the longevity of the cropping system from 10 to 7 years, while increasing the herbicide rate from 100% to 150% increased the longevity of the weed management system from 10 to 19 years. The rate at which resistant weeds appeared in the population was also higher at lower herbicide rates. Similar results can be observed for the examples illustrated in Fig. 6b and c. Conversely, the results indicate that changing herbicide rate does not always affect the sustainability of cropping systems to a significant degree. For example, for the modelled monogenic resistance situation (Fig. 6d), cutting or increasing herbicide rate made no difference to the sustainability of the system and cutting herbicide rates slightly decreased the rate at which resistant weeds appeared in the population. Note that these results indicate that when population dynamics and kill rates are taken into account, higher herbicide rates are not predicted to reduce the sustainability of the system in the face of monogenic resistance due to increased selection pressure, despite previous concerns (Christoffers, 1999; Friesen et al., 2000).

We argue that these results can be summarised by saying that lower rates or standard rates at reduced efficacy will have a significantly detrimental effect on the sustainability of cropping
systems when resistance is 'effectively poly-allelic'. By 'effectively poly-allelic' we mean that a significant increase in the ability of a weed to resist the effect of a herbicide is conferred only by the presence of a number of alleles at a single locus or across a number of loci, and not by any single allele. This contrasts with 'effectively mono-allelic' resistance where significant resistance is conferred by a single allele, even if more alleles can confer still higher resistance. This argument is supported by considering Fig. 8, as follows. When dominance is greater, the number of alleles required to give the same boost to resistance is lower, and it can be seen in Fig. 8 that this corresponds to a reduced effect of difference in herbicide rate. More negative epistasis also reduces the number of alleles required to give the same boost to resistance, because it means that the first allele has a relatively strong effect and alleles at other genes a relatively weak effect; it can be seen in Fig. 8 that this also corresponds to a reduced effect of difference in herbicide rate. Even more clearly in Fig. 7, when the number of genes involved in resistance is higher, then the effect of difference in herbicide rate is greater. And when resistance is stronger (higher threshR), and thus any one allele

Fig. 8. Results of analysis on the effect of varying six model parameters on sustainability of the system at three different herbicide rates (75%, 100% and 150%), while holding other parameters at default values (Table 1). From the top, left to right, the parameters varied are the chance of surviving the pre-emergent treatment (chsprem), dominance (dom), epistasis (epis), initial allele frequency (iaf), the selective herbicide threshold required to kill fully susceptible weeds (thresh), and the probability of an individual weed being completely untouched by a selective herbicide application (put). The vertical difference between the lines represents the difference in years of sustainable herbicide use due to difference in rates.
makes a greater contribution to resistance, then the effect of
difference in herbicide rate is lower. Thus, even for relatively
strong resistance, herbicide rate can have a significant effect on
the sustainability of the system if the initial allele frequency is
low enough (Fig. 6b) or if the number of genes involved in
confering that resistance is high enough (Fig. 6d). If the number
of genes involved in conferring resistance is relatively low,
herbicide rate will only have a significant effect on the sustain-
ability of the system at relatively weak resistance levels (Fig. 7).
Note that even with a single gene there are resistance levels
where herbicide rate can have an effect. However at those very
low resistance levels, weed control could probably be maintained
even after evolution of resistance by raising the herbicide rate or
by increasing the other control levels in the system.

In the modelled mixed resistance case, resistance can be con-
ferred by either a single strong dominant ‘major’ gene or the
additive effect of a number of relatively common and relatively
weak ‘minor’ alleles across several loci. Our analysis shows that in
this case the effect of difference in herbicide rate on the sustain-
ability of the system depends on the relative initial frequency of
the major and minor alleles (Fig. 9). In effect, it is simply a race between
the two types of resistance. If the frequency of the major gene allele
is high enough, compared to the frequency of the minor gene allele,
then ‘major gene’ mono-allelic resistance occurs first, and there is no
real effect due to difference in herbicide rate. On the other hand, if
the frequency of the minor gene allele is high enough, compared to
the frequency of the major gene allele, then ‘effectively poly-allelic’
resistance occurs first, and there is an effect due to difference in
herbicide rate. These results contrast with previous results from
modelling evolution of resistance in beetles that showed that major
genes always dominate (Groeters and Tabashnik, 2000) and support
recent evidence that evolution of polygenic resistance in the field
is not as rare as previously thought (Delye et al., 2007; Dinelli et al.,
2008).

The main default example and the subsequent analyses illus-
trate that there are at least four interrelated but distinct causes
for the observed cases where lower rates reduced sustainability.
The first and perhaps the most obvious cause is that lower
herbicide rates mean lower kill rates, which in turn means weed
populations are not driven down as quickly, even in the absence of
differences in resistance allele frequency or in the absence of
any resistance alleles at all. This is illustrated clearly in Figs. 5 and 6.
If effective resistance should appear in the population, the weed
numbers will then be building from a higher baseline and thus reach
critical thresholds more quickly. This mechanism is not related to
herbicide resistance; instead it is the accumulated effect of lower kill
from using lower rates. The decrease in sustainability as the
proportion of escapes increases above 0.1 in Fig. 8f is likely similarly
related to a simple reduction in kill rates.

A second closely related cause is that the presence of some
resistance alleles in the population, even at the assumed low
initial frequencies, combined with the assumed variability in
effective application rate between individual weeds, reduces the
selective herbicide kill rate even without any evolution occurring.
At low rates, this may be enough to reduce the kill rate below
the threshold needed to drive down the weed population, even
without evolution and even though the population would be
easily driven down at this same low rate in the absence of these
resistance alleles. This is illustrated in Fig. 6, where for some
examples weed density is increasing from the start for the cut 75%
rate, where in others this same rate is enough to drive down the
weed density at the beginning.

A third cause is real differences in rates of resistance evolution
through changes in allele frequencies at the population level and
a related but distinct fourth cause is evolution through the
emergence of novel genotypes as a result of recombination. These
are evident in the examples in Figs. 5 and 6 as differences in the
rate of increase in the percentage of resists. At low rates the
small difference in kill rate between genotypes with one or two
resistance alleles compared to genotypes with no resistance
alleles allows these resistance alleles to remain in the population
at increased frequencies over successive generations (the third
cause). Even though the total numbers of resistance alleles in the
population may be dropping, as their frequency increases they
will eventually combine to produce genotypes with three or four
alleles, and then five or six, and eventually enough alleles to
effectively confer resistance at the applied rate (the fourth cause).
At this point their numbers will begin to increase again. The
difference between rates is that at low rates the difference in kill
rate between genotypes with one or two resistance alleles
compared to genotypes with no resistance alleles will be larger,
so frequencies of resistance alleles in the population will increase
faster, increasing the chance of breeding genotypes with multiple
resistance genes. Also kill rates for all genotypes are lower, so
overall resistance allele numbers are driven down slower, which
means that there is less chance of the alleles being eliminated
completely from a local population. Of course for the strong
dominant monogenic example the converse is true; low rates
decrease the rate of selection for resistance because fewer
susceptible individuals are killed, resulting in lower selection
pressure. In Fig. 8f, the increase in sustainability as the proportion of escapes initially increases from zero is likely due to a refuge effect, with susceptible escapes diluting the resistance alleles in the next generation and slowing their accumulation.

In most of the examples shown in this paper, the four causes all contributed to the observed differences. This is clearly illustrated in Fig. 5, where weed densities are driven down at different rates in the period before significant resistance appears, but there are also different amounts of time before significant resistance appears and weed populations start increasing.

In this paper, we have considered a particular ecosystem, a cropping system where each year weeds compete with the same crop and the same selective herbicide is applied, but we argue that the results would apply to any system where herbicides are used regularly to control weeds. There is no doubt that adding diversity to the system, such as using alternative herbicides with different modes of action, rotating different crops that allow different herbicide options, or employing non-herbicide weed control measures, would increase the longevity of the system. However, our results indicate that the pattern of lower rates leading to decreased sustainability will still be found for many polygenic cases if alternative controls were included in the system, since in the absence of very large fitness penalties, cross-resistance, or local elimination of resistance genes, the effect of using alternative weed controls is simply to delay the accumulation of resistance genes. Alternative weed controls are an essential part of delaying resistance, but do not affect the question of whether lower rates or reduced effectiveness of normal rates will lead to faster resistance evolution.

We also considered a particular weed: annual ryegrass. However, we argue that the pattern of lower rates leading to decreased sustainability may be found for many cross-pollinated weed species, since the effect of differences in weed biology characteristics such as competitiveness, maximum seed production and longevity is probably just to delay or hasten the increase of resistance genes. In fact, the results are also likely to extend to other sexually reproducing organisms. On the other hand, the effect of different dormancy rates is not so simple; the dormant seedbank provides a refuge for non-resistant weeds, which could delay evolution of resistance to varying extents depending on the dominance and epistasis of the genes involved. Certainly, in cases where the breeding system of the weed is different (self-fertilising rather than out-crossing for example), ploidy levels are higher (hexaploid for example) or the biology affects factors such as the variability of effective herbicide rate among individuals (more variable growth rates for example) then the results found in this study may not hold, and further investigation is required.

Although lower rates commonly lead to decreased sustainability in the set of conditions considered in this paper, it is important to note that farmers should not rely only on high rates to keep herbicide resistance under control. Ensuring that the correct herbicide rate is applied at full efficacy, with optimal coverage in suitable environmental conditions on unstressed weed plants at the optimal growth stage, will ensure that the weeds do not receive an effectively reduced rate through reduced efficacy. If major herbicide resistance genes are present in the population at high enough levels relative to minor resistance genes then the effect of different rates will be minimal or non-existent. If farmers think that they can control resistance with high rates without using alternative methods to control resistance, then they will hasten the evolution of major gene resistance and may miss important opportunities for local elimination of minor and major resistance genes. Using robust herbicide rates should be considered as just one tool for delaying resistance and further research is clearly needed to determine in exactly which situations in the field low rates of herbicide application will make an important difference.

In general, our results agree with previous modelling studies (e.g. Birch and Shaw, 1997; Gardner et al., 1998; Groeters and Tabashnik, 2000; Shaw, 1989, 2000) in showing that lower doses can hasten polygenic resistance but delay monogenic resistance, but that the effect of dose on the rate of evolution of resistance is dependent on a variety of factors, particularly the underlying genetics of resistance, as well as the strength of the resistance and the level of refuge (proportion of population that escapes treatment). However, by incorporating more realistic population dynamics into our model, our results take into account and can be framed in terms of field weed density, which is of primary importance to farmers, instead of the more theoretical resistance allele frequency. This is important, because the negative effects of low rates are clearer when considering weed density than resistance frequency, and the positive effects of low rates on slowing rates of resistance evolution through reduced selection pressure are no longer evident when considering weed densities.

There are also many other questions and issues related to the evolution of herbicide resistance that could be addressed with an individual-based stochastic model that accounts for polygenic herbicide resistance, such as PERTH. These include:

- simulating polygenic resistance to pre-emergent herbicides;
- finding optimal strategies and farming systems for avoiding, minimising, or delaying the evolution of polygenic herbicide resistance;
- investigating what effect strategies for minimising the evolution of polygenic herbicide resistance are also likely to have on the evolution of monogenic herbicide resistance and determining what sorts of strategies would be effective for both major and minor gene resistance;
- determining whether weed biology factors such as breeding system (cross-pollinated or self-pollinated), dormancy, competitiveness, longevity, fecundity or dispersal mechanisms will make some species develop resistance faster or more often than other species, and determine if this depends on whether the resistance is monogenic or polygenic;
- simulating controlled experiments that have selected for herbicide resistance in weeds at relatively low rates over several generations, with the aim of determining what resistance genetics could possibly have led to the observed results; and
- evaluating the tradeoffs involved in reducing herbicide rates between possible beneficial economic and environmental effects and possible negative effects of reduced overall system sustainability.

In conclusion, since the results show that it is possible in different conditions for lower herbicide rates to either decrease the sustainability of cropping systems or alternatively to have no significant effect, perhaps we can argue that all of the weed scientists, whether they put up their hand or not, were at least partially correct. The results certainly show that it is possible that lower rates or reduced efficacy of standard rates could have an important detrimental effect on the sustainability of cropping systems when resistance is effectively polygenic, that they will have little or no effect if single major resistance genes are present at significant frequencies, and that further research is needed to determine how often circumstances in which lower rates will promote resistance actually occur in the field.

References


