Modelling the development of herbicide resistance
Modellierung der Entwicklung von Herbizidresistenz

Janin Rummland1*, Dirk Kerlen2, Henning Nordmeyer3 & Otto Richter1
1Technical University of Braunschweig, Institute of Geoecology, Langer Kamp 19c, 38106 Braunschweig, Germany
2Bayer CropScience Deutschland GmbH, Elisabeth-Selbert-Str. 4a, 40764 Langenfeld, Germany
3Julius Kühn-Institute, Federal Research Centre for Cultivated Plants, Institute for Plant Protection in Field Crops and Grassland, Messeweg 11/12, 38104 Braunschweig, Germany
*Corresponding author, j.rummland@tu-braunschweig.de
DOI: 10.5073/jka.2012.434.017

Summary
A population dynamic and genetic model was used to simulate the genetic evolution in an Apera spica-venti (L.) P. Beauv. population. The basis of the model is a life cycle of an annual weed population which is divided into five development stages. These are the seed bank, the seedlings, young plants, the seed producing plants and the produced seeds. The stages are linked through transition probabilities. The interaction between resistant and susceptible genotypes is mediated by a genetic submodel. The model supports the development of management strategies to prevent, reduce or at least retard the development of resistance. Through an embedment into a cellular automaton the dispersal of genetic information is simulated at field scale. Three simulations with different herbicide treatments are compared regarding the resistance evolution.

Keywords: Apera spica-venti, cellular automaton, evolution, genetic model, population dynamics, resistance management

Zusammenfassung

Stichwörter: Apera spica-venti, Evolution, genetisches Modell, Populationsdynamik, Resistenzmanagement, Zellularer Automat

1. Introduction
Apera spica-venti is a major weed in winter cereals in Germany. The number of resistant biotypes is increasing and it is necessary to analyze the development of resistance to find ways to decelerate, stop, avert or perhaps reverse this development. To test management strategies field trials are not always accomplishable due to a long time span of the resistance evolution and the expensiveness of such trials. Mathematical models can help to give an insight in the evolution of herbicide resistance. Different strategies can be tested and evaluated in a short time. A number of models are published. Some are concerned with the general herbicide resistance evolution (MAXWELL et al., 1990; RICHTER et al., 2002; RENTON et al., 2011) others are specified on specific species (VORPAHL and MÖWS, 2007; SANDT et al., 2008). In this paper, a population dynamic and genetic model was used to simulate the genetic evolution in an Apera spica-venti population. Scenarios were simulated to show the effects of different herbicide treatments on the resistance evolution.
2. Materials and method

2.1 Model framework

The used model is based on already existing model approaches (RICHTER et al., 2002; DUNKER, 2002; VORPAHL and MÖWS, 2007; SANDT et al., 2008). The life cycle of Apera spica-venti was divided into five stages. These are seeds in the seed bank (S), seedlings (SE), young plants (Y), mature plants (M) and new seeds (Snew). The life cycle stages are linked via transition probabilities. Furthermore, the Apera spica-venti population was divided in homozygous resistant plants (AA), heterozygous resistant plants (Aa) and homozygous susceptible plants (aa). These genotypes are linked through the inheritance. The life cycle graph is pictured in Figure 1.

![Life cycle graph](image)

**Fig. 1** Life cycle of Apera spica-venti.

**Abb. 1** Lebenszyklus von Apera spica-venti.

The simulation starts with seeds in the seed bank (S) which are germinating with the probability $p_G$.

$$SE = p_G \cdot S$$

The development of seedlings (SE) to young plants (Y) occurs with the probability $p_Y$ and this step is density dependent. The young plants have a survival probability $p_M$ to develop to mature plants. The herbicide application occurs at this stage, so that the survival is also affected by the efficacy of the herbicide ($p_H$). Hereby $p_H$ is different for the three genotypes.

$$M = Y \cdot p_M \cdot p_H$$

The life cycle steps above occur independently for each genotype. The proportion of the three genotypes in the new generated seeds is derived from the Hardy-Weinberg equation. The seed production (Snew) is influenced by the density of the mature weed plants (M) and the crop density (C). The density-dependent seed production was defined by RICHTER et al. (2002) and DUNKER (2002). To simulate the spatial distribution of the seeds, a cellular automaton was used. The cellular automaton consists of a grid with the cell sizes of 1 x 1 m and a total size of 72 x 32 m. In each of these cells the population dynamic is simulated. The major portion of the new generated seeds will remain in the cell but a small amount of the seeds will be distributed into the neighboring cells. This is
modeled by using the Gaussian distribution. The distribution from one cell into the neighboring cells is shown in Figure 2. A ‘von Neumann neighborhood relation’ with the range of two was used. The new seeds and the old seeds, which have not germinated in autumn, will survive with the probability \( p_w \) and a new cycle begins.

Fig. 2  Cellular automaton and the distribution of seeds in the neighboring cells.

Abb. 2  Zellulärer Automat und die Verbreitung der Samen in die Nachbarzellen.

2.2  Simulated Scenarios

Three scenarios with different efficacies of herbicides against resistant and susceptible plants were simulated. All scenarios began with randomly distributed seeds in the cellular automaton. The major part of the seeds were homozygous susceptible, only a few were heterozygous resistant and none of them were homozygous resistant. The simulation runs over 25 years.

In the first scenario, the same herbicide is used over the whole simulation period with an efficacy of 5 % on the resistant plants and 95 % on the susceptible plants.

In scenario two, an efficacy of 5 % on the resistant plants and 95 % on the susceptible plants was assumed for the first five years and after that an alteration with a herbicide with 98 % efficacy on all three genotypes started.

Scenario three also begins with the herbicide with the efficacy of 5 % on the resistant plants and 95 % on the susceptible plants for the first ten years. Than a herbicide was used with an assumed efficacy of 98 % on all three genotypes for five years and after that the same herbicide from the beginning was used again until the end of the simulation.

3.  Results

3.1  Scenario Simulation

Figures 3, 4 and 5 show the results of the three model runs. Pictured are the plants per m² prior and after the herbicide application. In the first scenario, the plant density is declining in the first five years due to the high efficiency of the herbicide on the susceptible plants, which have a great proportion of the population in the beginning of the simulation. With the years the number of resistant plants is slowly increasing and this is leading to a rising total plant density after five years. Over the years the density of susceptible plants is steadily at a very low level and the number of heterozygous and homozygous plants is increasing.

The second scenario shows a declining plant density in the first five years, but an increasing number of resistant plants. A resistant population is building up. In the sixth year the entire number of plants is minimized. In the seventh year a selection of the resistant plants takes place. The alternation of the herbicides goes on with minimizing the whole population in one year and selecting the resistant plants in the next year. The proportion of the susceptible allele is more and more decreasing over the
years until it is not present in the population anymore. In the end the population only exists of homozygous resistant plants and the plant density is steadily increasing over time.

**Fig. 3** First scenario: use of the same herbicide over 25 years.
**Abb. 3** Erstes Szenario: Behandlung mit dem gleichen Herbizid für 25 Jahre.

**Fig. 4** Second scenario: alternation of two herbicides.
**Abb. 4** Zweites Szenario: Wechsel von zwei Herbiziden.

**Fig. 5** Third scenario: herbicide change after 10 and 15 years.
**Abb. 5** Drittes Szenario: Wechsel des Herbizids nach 10 und 15 Jahren.
In the third scenario the resistant plants are selected by the herbicide in the first ten years causing their numbers to rise. The susceptible plants are controlled by the herbicide and thus the density is declining. In the beginning the population consisting of a greater number of susceptible plants causing the overall density to decrease due to the good control. The density of resistant plants is steadily increasing over the years and causing the overall density to increase from the fifth year on. The simulation of herbicide use with the efficacy of 98 % on all three genotypes causes a steep decrease of the plant density. Switching to the herbicide from the beginning after five years shows no increase in plant density.

Figure 6 shows the spatial distribution of the resistant plants from the calculations of scenario one. The resistant plants are spreading out across the field. After five years some local spots with high densities of resistant plants appear. After a longer simulation time it is seen that this spots are expanding and merging into each other. Simulations with other assumptions of herbicide use would lead to other developments of the spatial distributions of resistant plants.

![Distribution of resistant plants from scenario one for the time steps first, fifth, tenth and fifteenth year.](image)

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**4. Discussion**

The presented model offers the potential to predict the spatial and temporal dynamics of the resistant *Apera spica-venti* distribution regarding different herbicide strategies. The model showed that varying herbicide strategies causing an in- or decrease of resistant plants. Computer models can be a useful tool to estimate the effects of anti-resistant managements. Long-term effects of an annual or perennial herbicide treatment can be evaluated and if necessary countermeasures can be taken. The simulation results can only be seen as the basis for modeling the development of herbicide resistance, the model is only consisting of the life cycle and the genetics with the use of one or two herbicides. Extensions are required to simulate crop rotation or tillage. Furthermore the model is only capable of simulating target-site resistance. The next step should be an extension of the model combining it with a metabolic resistance model to achieve even more realistic resistance evolution.

**Acknowledgements**

The authors wish to thank our partners of BAYER Crop Science for financial support.

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