

Does weed control by precision spray technology favour the emergence of resistance?

Begünstigt die teilflächenspezifische Unkrautbekämpfung die Entwicklung von Resistenzen?

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Abstract

Weed control by precision farming is recommended both by economic and ecological reasons. It is still unclear whether precision weed control favours the emergence of herbicide resistant biotypes. To investigate this, the cellular automaton model of Sandt et al. (2008) for the simulation of precision weed control was extended to resistant biotypes and their genetic interactions. The model is capable of simulating the emergence of resistant biotypes in dependence of weed control thresholds, application rates and initial distribution of biotypes. Examples are shown for the case of polygenic inheritance of resistance involving three loci and thus 27 biotypes. Preliminary simulation results hint that precision farming can delay the emergence of resistance at high weed control thresholds.

Keywords: Metabolic herbicide resistance, polygenic inheritance, population dynamics, population genetics, precision agriculture, weed control

Zusammenfassung

Teilflächenspezifische Unkrautbekämpfung ist eine Methode für eine umweltschonende Landwirtschaft. Es ist jedoch unklar, wie diese Methode die Entwicklung von Herbizidresistenzen beeinflusst. Zur Untersuchung dieser Fragestellung wurde das von SANDT et al. (2008) für die Simulation der teilflächenspezifischen Unkrautbekämpfung entwickelte Modell durch die Hinzunahme resistenter Biotypen und ihrer genetischen Interaktion erweitert. Das Modell ermöglicht die Simulation der Entwicklung resistenter Biotypen in Abhängigkeit von der Schadschwelle, Aufwandmengen, Fruchtfolgen und der Anfangsverteilung resistenter Biotypen in der Population. Es werden Beispiele gezeigt für eine polygene Vererbung von metabolischer Resistenz unter Beteiligung von 3 Loci und damit 27 Biotypen. Erste Simulationsergebnisse deuten darauf hin, dass teilflächenspezifische Unkrautbekämpfung die Entwicklung resistenter Biotypen verzögern kann.

Stichwörter: Metabolische Herbizidresistenz, polygene Vererbung, Populationsdynamik, Populationsgenetik, teilflächenspezifische Unkrautbekämpfung

Introduction

In the last decade herbicide resistance has become a major issue for many weeds (BECKIE, 2006; POWLES et al., 2010). In recent years the adoption of precision farming has been promoted as a means to reduce herbicide input. Currently, commercial equipment to perform the identification and spraying of weeds in real time appears on the market, e.g. WEEDit and WeedSeeker. Whereas the short term economic benefit of site-specific herbicide management seems to be obvious especially at high weed infestations, the influence of precision farming on the long term development of herbicide resistance is not quite clear. However, in recent years it has been shown that low dose application rate favour the emergence of polygenic resistance. There are quite a few examples such as the evolution of polygenic herbicide resistance in *Lolium rigidum* by low-dose herbicide selection (BUSI et al., 2013; YU et al., 2013). So the question arises whether precision farming might have the same effect on resistance development as low dose application. To this end a model was developed comprising population genetics and dynamics and dispersal in the frame of a cellular automaton.

Materials and Methods

The model

General structure

A cellular automaton model is set up (cf. Fig. 1). At each grid cell the model comprises a time discrete population dynamic model of the life cycle of an annual plant with the stages seed (S), seedling (K), young plant (J) and adult plant (R). Biotypes of different resistance factors are linked by a genetic submodel. The model allows for both target site and metabolic resistance. Polygenic inheritance is described by a new approach based on tensor products of heredity matrices (RICHTER et al., 2016).

Polygenic inheritance

Polygenic inheritance is described by a new approach based on tensor products of heredity matrices (LANGEMANN et al., 2012). Biotypes with indices i , are ordered lexicographically with respect to the alleles X_i and x_i occurring in the gene. Biotype densities c_i are combined in a vector $c=(c_1, \dots, c_n)^t$. Rather simple combinatorial investigations in the case of a single gene with $n=3$ biotypes show that the amount of off-springs is proportional to a quadratic form. Since the amount of off-springs is related to the rate of change of biotype i , we find c_i' proportional to $c^t W_i c$ with a matrix W_i , which is called heredity matrix and elementary heredity matrix in the particular case of one gene locus. These elementary heredity transmission matrices for the single genes X_i, X_j can be fixed as $W_i = V_i$ with

$$V_1 = \begin{pmatrix} 1 & 1/2 & 0 \\ 1/2 & 1/4 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (1)$$

$$V_2 = \begin{pmatrix} 0 & 1/2 & 1 \\ 1/2 & 1/2 & 1/2 \\ 1 & 1/2 & 0 \end{pmatrix} \quad (2)$$

$$V_3 = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 1/4 & 1/2 \\ 0 & 1/2 & 1 \end{pmatrix} \quad (3)$$

and they can be written as the tensor products

$$V_1 = v_x \otimes v_x^t \quad (4)$$

$$V_2 = v_x \otimes v_x^t + v_x \otimes v_x^t \quad (5)$$

$$V_3 = v_x \otimes v_x^t \quad (6)$$

with

$$v_x = (1 \ 1/2 \ 0)^t$$

$$v_x = (0 \ 1/2 \ 1)^t$$

In the multi loci case transmission matrices W_i are obtained from the Kronecker product of the single transmission matrices

$$W_i = V_{i1} \otimes V_{i2} \otimes \dots \otimes V_{im} \quad (7)$$

with the number m of gene loci and with biotypes ordered again lexicographically with respect to multiple gene-loci. The reason for the tensor product lies in the fact that inheritance follows Mendel's law separately at every gene locus. The fraction of biotype " i " in the population after random mating is termed normed heredity function and is derived via the hereditary matrices as

$$g_i(c) = \frac{c^t W_i c}{c_{sum}^2} \quad (8)$$

Equation (8) gives the rates of off-springs of each biotype and thus, it is the basic for dynamic population genetic models.

Resistance factors

This general approach is taken from the paper of RENTON et al. (2011). A genotype with n_g loci is presented by the vector

$$G_i = [x_1, x_2, x_3, \dots, x_{n_g}] \quad (9)$$

where $x_i \in [0,1,2]$. For $x_i = 0$ there is no resistant gene at locus i , for $x_i = 1$ there is one resistant gene at locus i and for $x_i = 2$ there are two resistant genes at locus i . Weights are allocated to the x_i in the following way

$$w(x_i) = \begin{cases} 0 & \text{if } x_i = 0 \\ dom & \text{if } x_i = 1 \\ 1 & \text{if } x_i = 2 \end{cases} \quad (10)$$

where $dom \in [0,1]$. If the resistant gene is dominant, dom takes the value of one, if it is recessive it takes the value of 0. Intermediate values are also possible. We define the resistance factor r via the ED_{50} value (cf. Eq. 15), i.e. $ED_{50}(\text{resistant}) = r ED_{50}(\text{sensitive})$. It is related to the genotype via

$$r(x_1, x_2, x_3, \dots, x_{n_g}) = 1 + (R_{max} - 1) \left(\frac{\sum_{i=1}^{n_g} w(x_i)}{n_g} \right)^{2^{epis}} \quad (11)$$

The parameter $epis$ is called the epistasis factor and is a measure for the synergy between different genes. This is a sensitive parameter, which determines the pattern of resistance in the biotypes as was shown in the paper of RENTON et al. (2011). Dose response curves are described by the log-logistic model

$$Su_i(d) = \frac{1}{1 + \exp[b(\log(d) - \log(e_i))]} \quad (12)$$

The parameter d denotes the dose, b determines the steepness of the curve and e_i is the ED_{50} value of biotype i , i.e. $Su(e_i)=0.5$.

Population dynamic and genetic model

The model is derived from the life cycle graph of an annual plant (cf. Fig. 10) with the stages seed (S), seedlings (K), young plants (K) and adult plants (R). The transitions between the stages are mediated by the probabilities of emergence, p_A , survival of seedlings, p_K , survival of young plants, p_J , survival of seeds in soil during summer, p_S and during winter, p_W . The seed bank is restocked by the number of seeds (A) produced per adult plant. For each biotype a model is set up differing with respect to the survival probabilities under herbicide treatment. The biotypes are linked by a genetic submodel. This conceptual model is transferred to the following time discrete equations. Note that the model comprises 2 density dependent processes: seedling survival (eq. 14) and seed production (eq. 16). This is important to ensure that the population is bounded. At the beginning of a cycle K seedlings germinate from the seed bank (eq. 12). The index i denotes the biotype

$$K_i = p_A S_i \quad (13)$$

The survival of the seedlings is density dependent. The number of seedlings which can develop to juvenile plants is limited by D_{max} .

$$J_i = \frac{D_{max} K_i}{K_i + L} \quad (14)$$

The development to mature plants depends on the survival probability $Su_i(d)$ under herbicide dose d , which is given by a dose response function with biotype specific parameters.

$$R_i = J_i Su_i(d) \quad (15)$$

Seed production (A) is determined by the density of adult plants R and by competition by the crop (F).

$$A_i = \frac{A_{max_i} R_{total}}{(1 + \varepsilon R + \varphi F)^\lambda} g_i(R) \quad (16)$$

where the portion of seeds of biotype, $g_i(R)$, is given by equation (8). Finally, at the beginning of the next cycle the seedbank (S) is updated taking into account the dormant seed of the previous period, the surviving seed produced by mature plants in the previous period and the gain or loss by immigration (I) from and migration (E) to adjacent plots. The indices t, i, s, w denote time, biotype, summer and winter respectively.

$$S_{(t+1)_i} = (S_{ti}(1 - p_A) p_s + A_{it}) p_w + I_i - E_i \quad i = 1, \dots, m \quad (17)$$

Cellular automaton model

The local population dynamic model is embedded into a cellular automaton approach. The area under study has the size of 180x360 m and is partitioned into rectangular grid cells of size 1x1 m. For the simulation of precision spraying the field is portioned into subareas of size 25x36 m. According to the weed density in a subarea, spraying is turned on or off. On each cell, the population dynamic processes take place. Between cells, an exchange of seeds is mediated by a bivariate Gaussian distribution. The range of seed exchange is determined by this distribution and

in addition by the Moore radius. Layers of soil properties such as the pH-value or sand content influence basic parameters of the population dynamic model. Details are given in the paper of SANDT et al. (2008).

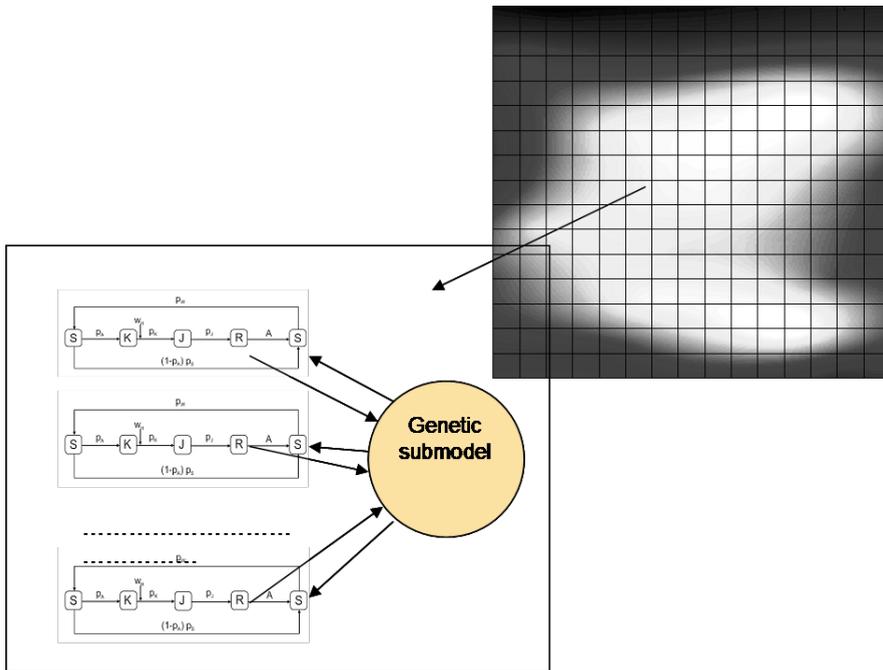


Fig. 1 Basic model structure: a population dynamic- and genetic model is embedded into a cellular automaton. The gray scale symbolizes soil properties such as pH value or sand content.

Abb. 1 Modellstruktur: ein populationsdynamisches -und genetisches Modell ist in einen zellulären Automaten eingebettet. Die graue Skala symbolisiert Bodeneigenschaften wie pH Werte oder Sandgehalte.

Model input

The dynamic behaviour of the model is determined by plant specific parameters such as D_{max} , A_{max} and resistance factors, environmental parameters such as soil properties, emergence probabilities of the seed (plant specific but dependent on climatic conditions), dispersion parameters such as the Moore radius and management parameters comprising application rates, weed density threshold for spraying, size of the subareas and crop rotation. In addition, the initial distribution of the seedbank in relation to biotypes is essential. General answers to the question posed in the headline therefore must be backed up by a large number of simulations with parameters out of a feasible parameter space. It should be kept in mind that the following results obtained by this model are only examples however interesting but cannot at this stage be generalized.

Results and discussion

The following examples refer to weeds exhibiting metabolic resistance which is assumed to be coded at three loci, giving rise to $n=27$ biotypes. Resistance factors were obtained as described above (eq. 11). Weed parameters were taken from the paper of SANDT et al. (2008). The cellular automaton generates spray maps based on the actual weed density in a subarea. Figure 2 shows an example.

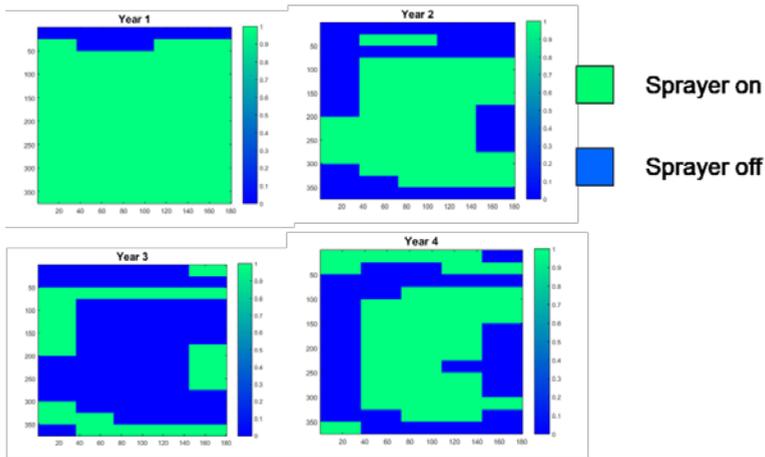


Fig. 2 Herbicide Spray maps generated by the model.

Abb. 2 Durch das Modell erzeugte Herbizid-Applikationskarten.

The next figure shows what might happen, if the management scheme favours the emergence of resistant biotypes. In this example, there was no crop rotation and weed density threshold was high and application rate low.

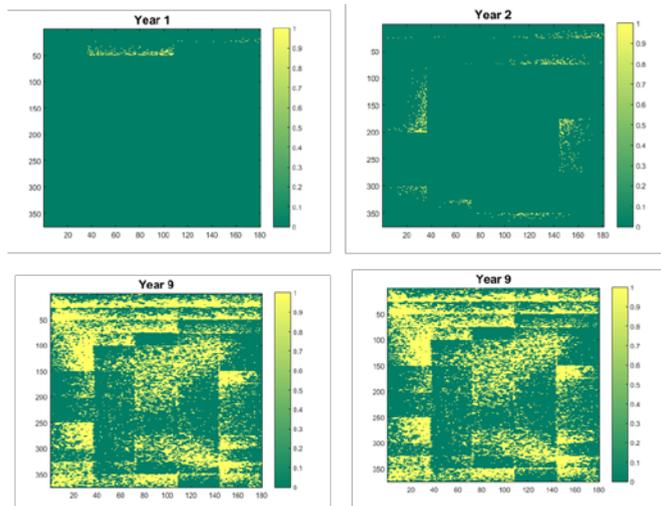


Fig. 3 Example for mismanagement. The number of plots with densities above threshold (in yellow) increases.

Abb. 3 Beispiel für missglücktes Management. Die Anzahl der Parzellen mit Unkrautdichten über dem Schwellenwert nimmt mit der Zeit zu.

Weed density thresholds in combination with application rates have an important influence of the emergence of resistant biotypes. Figure 4 shows the development in time of the seedbanks of biotypes 1,5,12 and 27 for a high (Fig. 4a) and for a low threshold (Fig. 4b). The biotypes presented are ordered with respect to their resistance factors. Biotype 1 has the lowest, biotype 27 the highest resistance factor. These simulations were run with 40% of the possible application rates. Low thresholds favour the emergence of resistant biotype 27. In the case of a low threshold (10 plants/m²) the development of resistance biotypes immediately sets in. At high threshold

(50 plants/m²) the development of resistant biotypes is suppressed albeit at a cost of a high seedbank of sensitive biotypes.

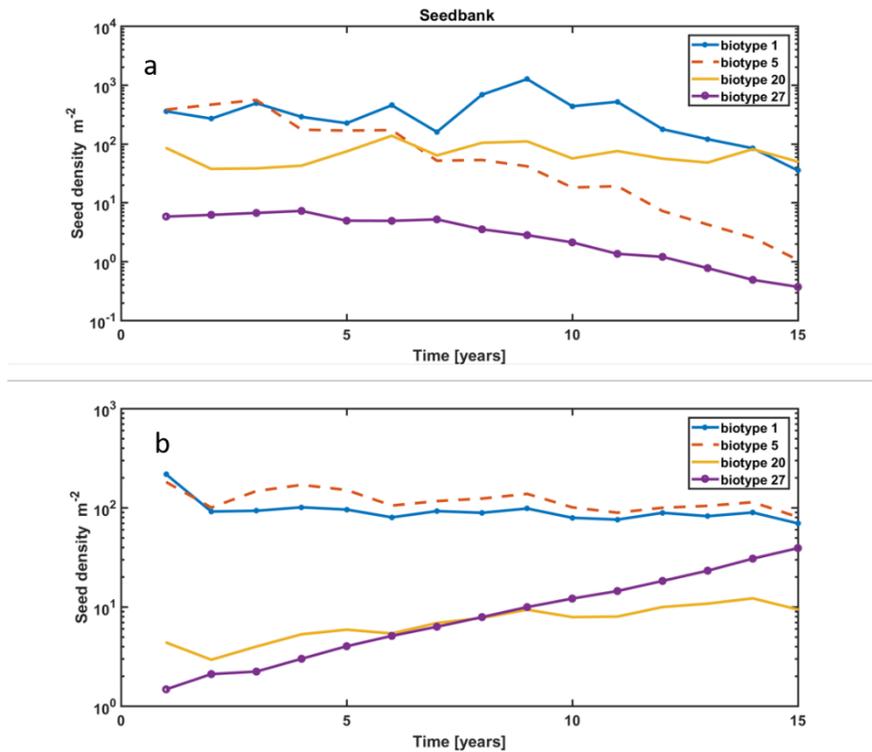


Fig. 4 Influence of weed control thresholds on the emergence of resistance. Fig 4a: 50 plant /m², Fig 4b: 15 plants/m². Biotype 1 has the lowest; biotype 27 has the highest resistance factor. Note that the plot is semilogarithmic.

Abb. 4 Einfluss der Bekämpfungsschwelle auf das Aufkommen resistenter Biotypen. Fig. 4a: Bekämpfungsschwelle 50 Pflanzen /m², Fig. 4b: 15 Pflanzen/m². Biotyp 1 hat den niedrigsten, Biotyp 27 den höchsten Resistenzfaktor. Man beachte die semilogarithmische Darstellung.

Figure 5 demonstrates the influence of the application rate on the dynamics of resistance. If the application rate is high (Fig. 5a), under high thresholds (here 50 plants/m²) no outbreaks of resistance occurs. Under low doses (here 40% of the maximum feasible dose) the seedbank of resistant biotypes is increasing from the beginning (Fig. 5b).

The simulation examples support (tentatively) the formulation of the following hypotheses:

- i) Weed control by precision spray technology favours the emergence of resistance at low application rates.
- ii) At high application rates even high control thresholds are tolerable.

These findings are only preliminary and have to be substantiated by exploration of the model behaviour in the feasible parameter space together with the exploration of a large body of field data.

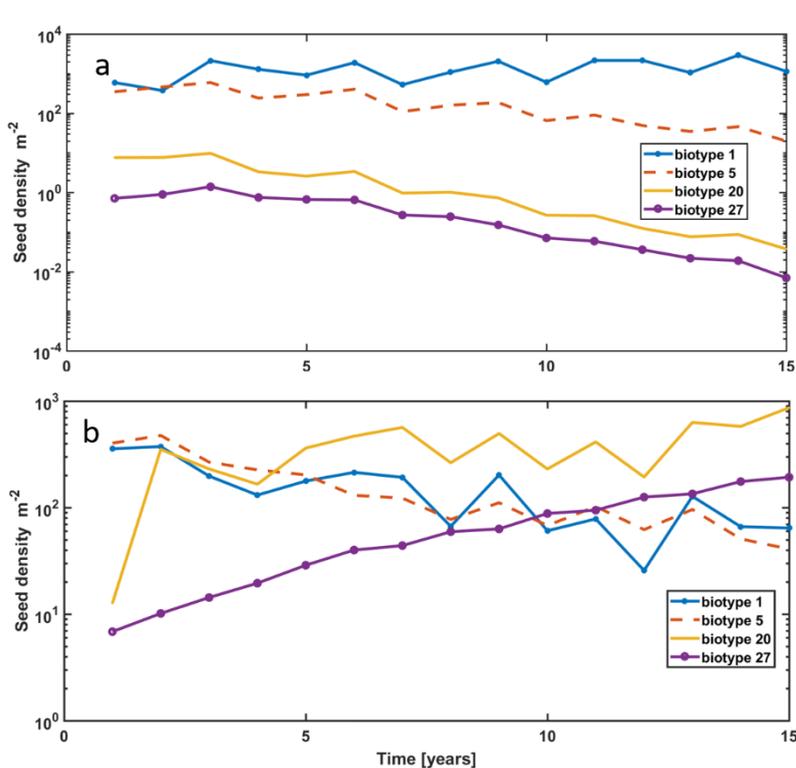


Fig. 5 Influence of application rates on the emergence of resistance. Fig 5a: threshold 50 plant /m² and maximum application rate, Fig 5b: threshold 50 plants/m² and 50% of maximum application rate. Biotype 1 has the lowest; biotype 27 has the highest resistance factor. Note that the plot is semilogarithmic.

Abb. 5 Einfluss der Aufwandmenge auf das Aufkommen resistenter Biotypen. Fig 5a: Bekämpfungsschwelle 50 Pflanze /m² und maximale Aufwandmenge. Fig 5b: Bekämpfungsschwelle 50 Pflanzen/m² und 40% der maximal möglichen Aufwandmenge. Biotyp 1 hat den niedrigsten, Biotyp 27 den höchsten Resistenzfaktor. Man beachte die semilogarithmische Darstellung.

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