Theoretical assessment of persistence and adaptation in weeds with complex life cycles

Dana Lauenroth[1,*] & Chaitanya S. Gokhale[1]

[1]Research Group for Theoretical Models of Eco-evolutionary Dynamics,

Department of Evolutionary Theory, Max Planck Institute for Evolutionary Biology,

August-Thienemann-Straße 2, 24306 Plön, Germany

[*]Correspondence: lauenroth@evolbio.mpg.de.com

Abstract

Perennial weed species like *Sorghum halepense* are a bane of agriculture and lead to significant economic losses. Such weeds can spread quickly and are particularly hard to control due to their ability to reproduce sexually via seeds and asexually through rhizomes. The growing problem of herbicide resistance imposes an additional major challenge on the weed management. Our theoretical study of *Sorghum halepense* incorporates the complete complex life cycle along with control measures of herbicide application and tillage. We evaluate the extent of control that the different strategies provide and the evolution of herbicide resistance. Our analysis indicates that the natural frequency of target-site resistance mainly depends on the resistance cost and less on its dominance. The sexual phase of the life cycle, including self-pollination and seed bank dynamics, contributes substantially to the persistence and rapid adaptation of the weed. For example, the seed bank significantly increases the probability of escape from control while maintaining genetic variation. We also extend our analysis to study herbicide mixtures, rotations and treatment combinations and how they contribute to reducing control failure. Our model is rooted in observational and experimental data on *Sorghum halepense*. Still, the general methodology developed herein is extendable to other perennial weeds comprising a complex life cycle. This approach shows how an integrated interdisciplinary view can synergistically contribute to understanding and tackling the global threat to food production presented by resistant weeds.

Keywords: seed bank, dormancy, agricultural weeds, persistence, herbicides, Johnsongrass, resistance evolution

1 Introduction

Sorghum halepense (Johnsongrass) is infamous for being "a threat to native grasslands and agriculture" (Klein & Smith 2021, p. 413). Johnsongrass is classified as a weed in 53 countries (Klein & Smith 2021), and Holm et al. (1977) ranked it as the world's sixth-worst weed. It causes significant yield losses in 30 different crops, including essential cash crops such as corn, cotton, sugarcane and soybean (Klein & Smith 2021). The weed also out-competes the native grasses (Schwinning et al. 2017). In addition to resource competition, Johnsongrass affects crop production by serving as an alternative host for various agricultural pests (Peerzada et al. 2017, Klein & Smith 2021). Johnsongrass is a highly competitive and invasive perennial weed capable of sexual reproduction through seeds and asexual propagation via rhizomes (Warwick & Black 1983, Peerzada et al. 2017, Schwinning et al. 2017).

In cropped areas, typical control of Johnsongrass is by a postemergence application of herbicides, particularly glyphosate and acetyl-coenzyme-A carboxylase (ACCase)- and acetolactate synthase (ALS)-inhibitors (Peerzada et al. 2017, Klein & Smith 2021). However, the continuous use of such herbicides has caused Johnsongrass to develop resistances, including target-site resistance towards various ACCase- and ALS-inhibitors (Lorentz 2014, Peerzada et al. 2017). Furthermore, multiple types of resistance now exist due to the excessive worldwide glyphosate application over the last decades (Vila-Aiub et al. 2007). Integrated approaches, combining herbicide application with mechanical measures such as tillage and potentially crop rotation, are reported to provide efficient control of Johnsongrass, including its rhizomes (Warwick & Black 1983, Peerzada et al. 2017). Herbicide mixtures and rotations involving herbicides with different sites of action reportedly reduce weed population sizes more than the recurrent use of one herbicide class and delay resistance evolution (Diggle et al. 2003, Busi et al. 2020, Zeller et al. 2021).

Mathematical modelling has proven to be a valuable tool for studying long-term population dynamics of weeds (Holst et al. 2007, Freckleton & Stephens 2009). However, the majority of this models deals with annual weeds, the complex life cycle of perennial species has been addressed to a lesser extend (Holst et al., 2007, but see Liu et al., 2019; Holmes et al., 2022). To our knowledge, just two population dynamical models of *Sorghum halepense* exist so far, comprising the perennial life cycle (Liu et al. 2019, Holmes et al. 2022). Liu et al. (2019) presented an individual-based model, which captures the sexual reproduction, including self-pollination, but lacks a seed bank. The density-dependent discrete model of Holmes et al. (2022) comprises both, a seed bank and a bank of rhizome buds. However, it lacks the possibility of de novo resistance evolution, and pure outcrossing is assumed.

The goals of this study are (i) to explore how the standing genetic variation of a Johnsongrass population never exposed to the herbicides is influenced by the fitness cost associ-

ated with resistance and its dominance; (ii) to study how genetic details like the dominance of resistance and the associated fitness cost impact the population dynamics and spread of herbicide resistance under control; (iii) to examine the effects selfing and a seed bank have on the survival, genetic diversity, and resistance evolution of such seed and rhizome propagated species; (iv) to determine the potential of tillage or a second herbicide and herbicide rotations for controlling resistance evolution and weed control failure. To this aim, we implemented the life cycle of Johnsongrass, both mathematically and computationally. The sexual and asexual phases are incorporated differentially since the spread of resistance is explicitly genetic.

Even though the model is presented in the context of *Sorghum halepense*, it can capture the population dynamics and herbicide resistance adaptation of other perennial weed species. Our population-based model could, more generally, be adapted to model any species that comprises sexual reproduction, in the continuum of selfing and outcrossing, asexual propagation and potentially a seed bank component.

2 Methods

The seminal work by Sager & Mortimer (1976) has inspired several models of weed life cycles. The population dynamic is then commonly modelled deterministically using a series of time discrete equations corresponding with the included life stages (Holst et al. 2007). Often the population is assumed to be even-aged, with the system time-steps of one-year increments (Holst et al. 2007). We introduce a detailed population-based deterministic model with an annual time step describing the dynamics of the seed and rhizome propagated perennial weed *Sorghum halepense* and forecasting the evolution of target-site herbicide resistance. We implement stochastic simulations of this population-based model capturing the stochastic nature of mutations, genetic drift and extinctions.

2.1 Model description

Johnsongrass plants are dormant throughout the winter, overwintering as seeds or rhizomes in the ground (Warwick & Black 1983). At the beginning of a growing season, seeds germinate to produce seedlings, and shoots emerge from nodes on the rhizomes (Peerzada et al. 2017). Mature plants produce new rhizomes and, after flowering, viable seeds (Warwick & Black 1983, Peerzada et al. 2017).

For deriving a population dynamics model of Johnsograss growing in one field, we consider five life-history stages: seeds, seedlings, rhizomes, shoots and adult plants. One time step of the model corresponds to one year.

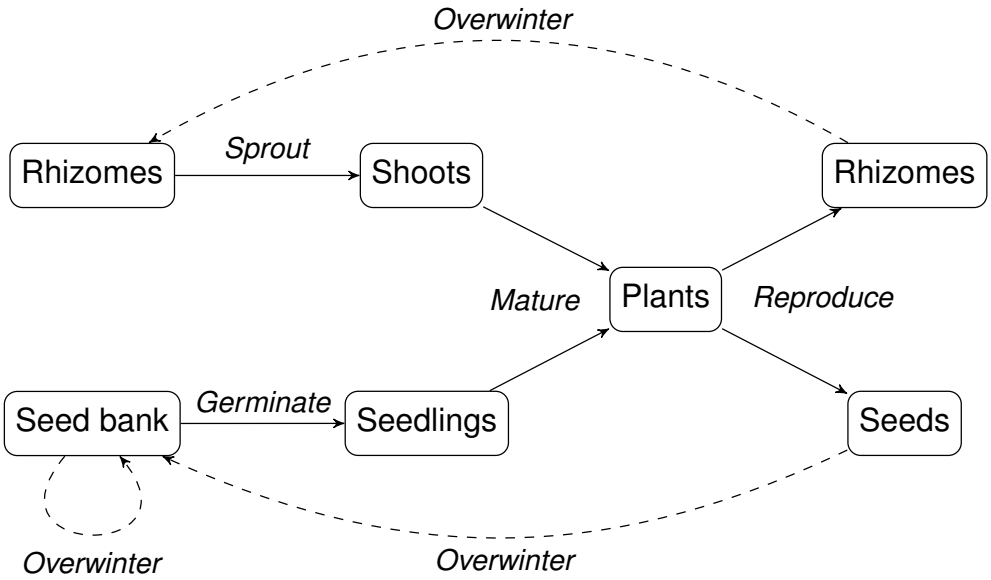

Figure 1: **Diagram of Johnsongrass life cycle as implemented in the population-based model.** Within-season dynamics are represented by solid arrows and dynamics between seasons by dashed arrows. The upper life cycle path depicts the asexual propagation whereas the bottom path corresponds to sexual reproduction of Johnsongrass (Liu et al. 2019).

Life cycle. For now, we neglect herbicide resistance and its genetics and concentrate solely on the plant life cycle. Figure 1 shows the corresponding life cycle of Johnsongrass as implemented in the model with both reproduction pathways, sexual via seeds and asexual via rhizomes. The rhizomes in the ground at the beginning of season t are denoted by $Z(t)$. Axillary buds formed on the nodes of rhizomes can develop as shoots or secondary rhizomes (Paterson et al. 2020). However, the apex of a rhizome exerts apical dominance over axillary buds inhibiting their growth (Beasley 1970). Let b be the number of nodes on a rhizome and g_Z the proportion of nodes producing shoots during a season. Then the number of shoots at time t is given by,

$$T(t) = g_Z \, b \, Z(t) \, . \tag{1}$$

For now, we assume a constant seed bank B present in each season. Within a season t, only a certain proportion g of the seeds in the seed bank germinates to produce seedlings (Legutzamón 1986),

$$L(t) = g \, B \, . \tag{2}$$

Seedlings and shoots grow up to adult plants. Even though rhizome shoots might emerge earlier than seedlings due to a lower temperature requirement for germination, similar growth

and development were observed for Johnsongrass plants originating from seeds and rhizomes (McWhorter 1961, Horowitz 1972). For this reason and simplicity, adult plants are not distinguished by their origin. Therefore,

$$P(t) = L(t) + T(t)\,, \tag{3}$$

gives the number of adult plants present in season t. New rhizomes are formed by the plants of which only a certain amount $(1 - d_Z)$ survives the winter to become the rhizomes of next season $t + 1$,

$$Z(t+1) = (1 - d_Z)\, P(t)\,. \tag{4}$$

Control and resistance. Numerous control strategies exist for curbing and eliminating weeds. They are typically classified via their primary mode of action, chemical, mechanical, biological, cultural or allelopathic (Peerzada et al. 2017). We consider tillage as a mechanical control measure and postemergence herbicide application as chemical control. As in the case of target-site resistance, e.g. against ACCase- or ALS-inhibitors, we consider a single allele R associated with herbicide resistance, (Scarabel et al. 2014, Hernndez et al. 2015, Kreiner et al. 2017). We assume a diploid genotype, but the model can readily be extended to reflect higher ploidy levels (see Holmes et al. (2022) for a comparison of the rate of herbicide resistance evolution for diploid and tetraploid genotypes). The susceptible phenotype results from the homozygous genotype WW, and genotype RR plants are resistant. The factor $0 \leq k_h \leq 1$ captures the variability in the dominance of resistance such that recessivity, partial dominance and complete dominance of the resistance trait can be captured in the model. Spontaneous mutations to the resistance allele R can occur during the sexual reproduction phase of the life cycle. We do not consider back mutations as they do not change the dynamics relevantly.

Herbicide application is expected to kill solely susceptible plants or partly resistant heterozygotes with an efficiency h or $(1 - k_h)\, h$, respectively. Due to inadequate translocation of herbicides into the rhizomes and their dormant buds, they are only partly controlled by the herbicide (Beasley 1970, Tuesca et al. 1999). Therefore when shoots are killed by herbicide application but not the parent rhizome, shoots may eventually resprout from the rhizome (Beasley 1970, Tuesca et al. 1999). This resprouting reduces the effective herbicide efficiency on rhizome shoots h_T compared to the herbicide efficiency on seedlings h_L (Vidrine 1989). During tillage, Johnsongrass rhizomes are cut into pieces and partly turned to the soil surface (Warwick & Black 1983). The fragmented rhizomes in shallow layers are exposed to low temperatures during winter, leading to increased mortality d_Z^* (Hull 1970, Warwick & Black 1983). Additionally, rhizome fragmentation is reported to maximize bud sprouting

(McWhorter & Hartwig 1965), allowing for an enhanced control h_T^* of Johnsongrass plants originating from rhizomes by the herbicide (Hull 1970, Tuesca et al. 1999).

Let $\mathbf{T}(t) = (T_{\text{WW}}(t), T_{\text{RW}}(t), T_{\text{RR}}(t))$, $\mathbf{L}(t) = (L_{\text{WW}}(t), L_{\text{RW}}(t), L_{\text{RR}}(t))$, $\mathbf{P}(t) = (P_{\text{WW}}(t), P_{\text{RW}}(t), P_{\text{RR}}(t))$ and $\mathbf{Z}(t) = (Z_{\text{WW}}(t), Z_{\text{RW}}(t), Z_{\text{RR}}(t))$ be the vectors of shoot, seedling, plant and rhizome numbers in growing season t, respectively, and $\mathbf{B} = (B_{\text{WW}}, B_{\text{RW}}, B_{\text{RR}})$ the vector describing the composition of the seed bank. The population dynamics under herbicide application are then described by,

$$\mathbf{T}(t) = g_Z\, b\, \mathbf{Z}(t)\,, \tag{5}$$

$$\mathbf{L}(t) = g\, \mathbf{B}\,, \tag{6}$$

$$\mathbf{P}(t) = \mathbf{L}(t) \begin{pmatrix} 1-h_L & 0 & 0 \\ 0 & 1-(1-k_h)\,h_L & 0 \\ 0 & 0 & 1 \end{pmatrix} + \mathbf{T}(t) \begin{pmatrix} 1-h_T & 0 & 0 \\ 0 & 1-(1-k_h)\,h_T & 0 \\ 0 & 0 & 1 \end{pmatrix}, \tag{7}$$

$$\mathbf{Z}(t+1) = (1-d_Z)\,\mathbf{P}(t)\,. \tag{8}$$

The herbicide efficiencies h_L and h_T are zero in a herbicide-free scenario. Enhanced winter mortality of rhizomes d_Z^* depicts the use of mechanical control. For the case of the combined treatment, an increased herbicide efficiency on rhizome shoots h_T^* is used additionally.

Second herbicide: To study the effect of binary herbicide mixtures and rotations, we consider two classes of herbicides used for Johnsongrass control, ACCase- and ALS-inhibitors (Peerzada et al. 2017). Due to the different sites of action, target-site resistance against herbicides of the one class is uncorrelated with resistance against the other herbicide class. However, in *Lolium rigidum* multiple and cross-resistance to ACCase- and ALS-inhibitors has been reported (Torra et al. 2021). This is caused by a combination of the target-site and non-target-site resistance (Torra et al. 2021). Since we do not consider off-target resistance, we omit cross-resistance. We assume that the herbicides act independently of each other. The combined efficiency of the two herbicides applied together in a mixture can then be calculated by $h_{mix} = 1-(1-h_1)(1-h_2) = h_1 + h_2 - h_1 h_2$, where h_1 and h_2 are the efficiencies of the herbicides in mono treatment (Bliss 1939). Likewise, we suppose the effects of the fitness costs associated with the respective target-site resistance to be independent such that the fitness cost of a plant fully resistant against both herbicides is given by $c_{\text{double resistant}} = 1-(1-c_1)(1-c_2) = c_1 + c_2 - c_1 c_2$, where c_1 and c_2 are the fitness costs of resistance towards the individual herbicides.

Intraspecific competition. Johnsongrass is a highly competitive weed, more strongly affected by intraspecific competition than by competition from other perennial weeds, like big bluestem (*Andropogon gerardii*), little bluestem (*Schizachyrium scoparius*) and switchgrass (*Panicum virgatum*) (Schwinning et al. 2017). Furthermore, as the crop density is assumed to remain constant throughout the seasons, competition from the crop is not explicitly modelled. We implement the hyperbolic self-thinning function introduced by Yoda et al. (1963) to model early plant survival. Thus the density-dependent mortality of Johnsongrass resulting from intraspecific competition is described by,

$$\widetilde{\mathbf{P}}(t) = \mathbf{P}(t) \left(1 + m \frac{n_P(t)}{A}\right)^{-1}, \tag{9}$$

where $\widetilde{\mathbf{P}}(t)$ is the vector of plants surviving till the end of season t, A the field size, $n_P(t) = \sum_{i \in \{\text{WW, RW, RR}\}} P_i(t)$ the total number of plants in the field without self-thinning and m^{-1} gives the highest possible density of Johnsongrass after self-thinning (Yoda et al. 1963). Therefore,

$$\mathbf{Z}(t+1) = (1 - d_Z)\, \widetilde{\mathbf{P}}(t)\,, \tag{10}$$

gives now the vector of rhizomes present at the start of season $t + 1$.

Moreover, Williams & Ingber (1977) reported a significant impact of intraspecific competition on the formation of reproductive structures in Johnsongrass. In their greenhouse experiment, high stand densities delayed or even prevented tillering and delayed the formation of rhizomes and panicles. Furthermore, the final dry weight of the reproductive structures was reduced in crowded stands. Due to lack of data, we assume that the total seed and rhizome yield per square meter approaches a constant value at moderate to high densities (Firbank & Watkinson 1985). Then the mean seed production and mean rhizome bud production per plant in season t can be described by,

$$f(t) = f \left(1 + a \frac{n_{\widetilde{P}}(t)}{A}\right)^{-1}, \tag{11}$$

$$b(t) = b \left(1 + a \frac{n_{\widetilde{P}}(t)}{A}\right)^{-1}, \tag{12}$$

respectively, where f and b are the mean yields of an isolated plant, a is the area required by a plant to produce f seeds and b rhizome buds and $n_{\widetilde{P}}(t) = \sum_{i \in \{\text{WW, RW, RR}\}} \widetilde{P}_i(t)$ gives the total number of plants at the end of season t (after self-thinning) (Watkinson 1980). As the number of buds produced on a rhizome in a given season determines the number of shoots that can

emerge from this rhizome in the next growing season,

$$\mathbf{T}(t) = g_Z\, b(t-1)\, \mathbf{Z}(t)\,, \tag{13}$$

gives the shoots emerging in season t.

Pollination and seed bank dynamics. Herbicide resistance can be associated with a fitness cost (Baucom 2019). In particular, a fitness cost on seed production associated with a mutation conferring resistance against ACCase-inhibitors has been reported in Johnsongrass (Panozzo & Sattin 2021). As for the herbicide resistance, the model allows for dominance variability regarding the fitness cost, implemented by a dominance factor $0 \leq k_c \leq 1$. Hence, for type RR and RS plants, a reduction by c and $k_c c$, respectively, in fecundity, i.e. the average number of seeds produced by one plant, is considered compared to the fecundity f of susceptible plants. Johnsongrass is primarily self-pollinated, which generally leads to a higher level of homozygosity and reduced genetic diversity in the produced seeds as opposed to cross-fertilization (Warwick & Black 1983). Let p_{self} denote the proportion of self-pollination. Then the number of seeds with genotype i produced during season t by self-pollinated plants can be calculated as,

$$S_i^{self}(t) = p_{self}\, f(t)\, \widetilde{\mathbf{P}}(t)\, \mathbf{m}^i \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1-k_c\,c & 0 \\ 0 & 0 & 1-c \end{pmatrix}\,, \tag{14}$$

with

$$\mathbf{m}^i = \left(M^i_{\text{WW WW}}, M^i_{\text{RW RW}}, M^i_{\text{RR RR}}\right)^T\,, \tag{15}$$

where M^i_{jk} gives the proportion of type i seeds produced by a plant of genotype j pollinated by type k pollen. This follows Mendelian inheritance and includes spontaneous mutations to the resistance allele R occurring with probability μ (see supporting information in Section 3.4). However, up to $5\,\%$ of cross-pollination has been observed in fields where Johnsongrass plants grow at sufficiently small distances (Warwick & Black 1983). Over the tth season a number of

$$S_i^{cross}(t) = \frac{(1-p_{self})\, f(t)}{n_{\widetilde{P}}(t)}\, \widetilde{\mathbf{P}}(t)\, \mathbf{M}^i\, \widetilde{\mathbf{P}}(t)^T \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1-k_c\,c & 0 \\ 0 & 0 & 1-c \end{pmatrix} \tag{16}$$

type i seeds are produced by cross-pollinated plants, where

$$\mathbf{M}^i = \begin{pmatrix} M^i_{\text{WW WW}} & M^i_{\text{WW RW}} & M^i_{\text{WW RR}} \\ M^i_{\text{RW WW}} & M^i_{\text{RW RW}} & M^i_{\text{RW RR}} \\ M^i_{\text{RR WW}} & M^i_{\text{RR RW}} & M^i_{\text{RR RR}} \end{pmatrix}, \tag{17}$$

contains the proportions of type i seeds produced by the different matings, M^i_{jk}, $j, k \in \{\text{WW, RW, RR}\}$. Adding up the seeds produced by self- and cross-pollinated plants gives the total number of genotype i seeds produced in season t,

$$\begin{aligned} S_i(t) &= S_i^{self}(t) + S_i^{cross}(t) \\ &= f(t)\,\widetilde{\mathbf{P}}(t) \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1-k_c\,c & 0 \\ 0 & 0 & 1-c \end{pmatrix} \left[\frac{1-p_{self}}{n_{\widetilde{P}}(t)}\,\mathbf{M}^i\,\widetilde{\mathbf{P}}(t)^T + p_{self}\,\mathbf{m}^i \right]. \end{aligned} \tag{18}$$

It is worth noting that the fitness cost associated with resistance is relevant regardless of the modelled control regime. Freshly produced Johnsongrass seeds are highly dormant throughout the season (Warwick & Black 1983). They germinate in the subsequent seasons and can stay viable in the ground for several years (Warwick & Black 1983, Peerzada et al. 2017). New seeds are assumed to enter the seed bank B. However, they are subject to post-dispersal predation and might lose viability or decay till the beginning of the next season with a probability of d_S (Bagavathiannan & Norsworthy 2013). Within a season t, only a certain proportion g of the seeds in the seed bank germinate to produce seedlings,

$$\mathbf{L}(t) = g\,\mathbf{B}(t)\,, \tag{19}$$

while non-germinated seeds may either be lost due to decay and viability loss, with a probability d_B, or survive to be part of next season's seed bank (Bagavathiannan & Norsworthy 2013). Therefore, the seed bank of season $t+1$ consists of non-germinated seeds from the previous seed bank and the seeds produced during season t,

$$\mathbf{B}(t+1) = (1-d_B)\,(1-g)\,\mathbf{B}(t) + (1-d_S)\,\mathbf{S}(t)\,. \tag{20}$$

Standing genetic variation. Standing genetic variation has been reported for herbicide resistance in plant populations never exposed to herbicides, among others, target-site resistance against ACCase-inhibitors (Délye et al. 2013). Moreover, it has been argued that herbicide resistance adaptation may primarily proceed from standing genetic variation as the initial fre-

quency of resistant individuals may be much higher than the spontaneous mutation rate of resistance alleles (Kreiner et al. 2017, Délye et al. 2013). We derive a matrix model approximation of the entire life cycle dynamics to investigate the long-term population dynamics. We calculate the expected standing genetic variation in an untreated field depending on the fitness cost associated with resistance and its dominance .

Let $\mathbf{P}(t) = (P_{\text{WW}}(t), P_{\text{RW}}(t), P_{\text{RR}}(t))$ and $\mathbf{B}(t) = (B_{\text{WW}}(t), B_{\text{RW}}(t), B_{\text{RR}}(t))$ be the vectors of plant and seed numbers in season t, respectively, and let $(\mathbf{P}(t), \mathbf{B}(t))$ be the population vector containing plant and seed numbers of all genotypes. Assuming an infinite density limit (no self-thinning) and maximum reproduction ($f(t) = f$, $b(t) = b$, $\forall t \geq 0$) and neglecting cross-pollination ($p_{self} = 1$), the population dynamics of *Sorghum halepense* can be approximated by

$$\begin{aligned}
&(\mathbf{P}(t+1), \mathbf{B}(t+1)) \\
&= (\mathbf{P}(t), \mathbf{B}(t)) \underbrace{\begin{pmatrix} g_Z\,(1-d_Z)\,b\,\mathbf{I} + g\,(1-d_S)\,f\,\mathbf{D}\,\widetilde{\mathbf{M}} & (1-d_S)\,f\,\mathbf{D}\,\widetilde{\mathbf{M}} \\ g\,(1-d_B)\,(1-g)\,\mathbf{I} & (1-d_B)\,(1-g)\,\mathbf{I} \end{pmatrix}}_{=:\mathbf{E}}, \qquad (21)
\end{aligned}$$

where $\mathbf{I}$ is the identity matrix of size 3,

$$\mathbf{D} = \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 - k_c\,c & 0 \\ 0 & 0 & 1-c \end{pmatrix},$$

is the diagonal matrix incorporating fitness cost on seed production and

$$\widetilde{\mathbf{M}} = \begin{pmatrix} (1-\mu)^2 & 2\,\mu\,(1-\mu) & \mu^2 \\ \frac{1}{4}\,(1-\mu)^2 & \frac{1}{2}\,(1-\mu^2) & \frac{1}{4}\,(1+\mu)^2 \\ 0 & 0 & 1 \end{pmatrix},$$

is the inheritance matrix assuming simple Mendelian inheritance.

We use the *Perron-Frobenius* theorem 1 (see supporting information in Section 3.4) to derive the long-term population dynamics

$$\lim_{t\to\infty} \frac{(\mathbf{P}(t+1), \mathbf{B}(t+1))}{\rho^t} = (\mathbf{P}(1), \mathbf{B}(1))\ \mathbf{u}^T\mathbf{v}\,, \qquad (22)$$

where ρ is the positive and simple eigenvalue of $\mathbf{E}$, that is in absolute value the largest eigenvalue, and $\mathbf{u}$ and $\mathbf{v}$ are the corresponding positive right and left eigenvectors (Harris 1963). Thus, in the long run, the population composition is approximately a real multiply of $\mathbf{v}$, regard-

less of the initial population. Therefore $\mathbf{v}$ gives us a rough estimate of the expected standing genetic variation in a Johnsongrass population that has never experienced selective pressure due to herbicide application before (see Fig. 2).

2.2 Stochastic simulations.

Deterministic models capture selection as an evolutionary force well, particularly for large population sizes. However, when the population is small, strong selection may lead to the extinction of non-favoured types. Moreover, genetic drift acquires more relevance in smaller populations. We implemented stochastic simulations of our population-based model to account for such natural stochasticity. This stochastic simulation model allows for random shifts in allele frequencies and extinction.

As in the underlying deterministic model, the population dynamics are modelled in discrete time steps of one growing season. The numbers of rhizome buds and seeds produced in season t by plants of a specific genotype i are drawn from Poisson distributions with mean $P_i(t)\, b(t)$ and $P_i(t)\, f(t)$, respectively. Probabilities, such as the germination probability, death probabilities, and herbicide efficiency, are realised on the population level for the different genotypes using binomial distributed random numbers. The number of sensitive seeds germinating in season t is, for example, given by a binomial random number with parameters $B_{\mathrm{WW}}(t)$ and g. Multinomial random numbers are used to derive the initial seeds and rhizomes and to model fertilisation and mutation. Seeds resulting from self-pollination in heterozygous plants, for instance, are obtained from the multinomial distribution with the total number of seeds produced via selfing by heterozygous plants and the inheritance vector $\left(\frac{1}{4}, \frac{1}{2}, \frac{1}{4}\right)$ as parameters.

3 Results and Discussion

3.1 Genetic complexities.

Standing genetic variation. The vector $\mathbf{v}$ from Eq. (22) approximates the genetic composition of a Johnsongrass population unexposed to the considered herbicide. We explored the impact of the fitness cost c associated with resistance and its degree of dominance on the proportion of resistance alleles R and the abundance of the resistant types.

The proportion of resistance allele R is controlled by the fitness cost on seed production (Fig. 2 (A)). It decreases with an increasing fitness cost. For a reduction of seed production in resistant homozygotes exceeding $12\ \%$, the proportion of resistance alleles is of the order of the considered de novo mutation rate $\mu = 10^{-8}$ (Haughn & Somerville 1987, Harms & DiMaio 1991). This indicates that competition with the sensitive type prevents the establishment

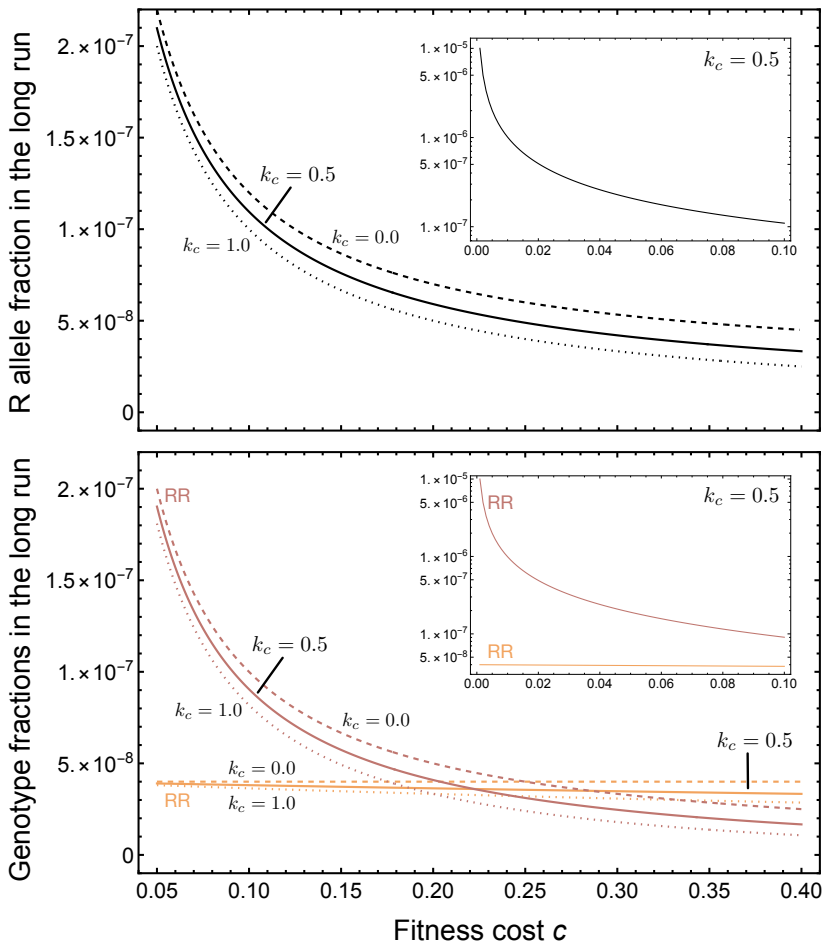

Figure 2: Frequency of the resistance allele R (A) and the resistant types (B) in the long-term population composition depending on the fitness cost c on seed production associated with resistance. The long-term population composition is obtained from Eq. (22) and shown for different degrees of dominance (k_c) of the resistance cost. Note that the y-axis of the smaller plots have a log-scale.

of resistant types. A higher degree of dominance regarding the fitness cost decreases the R allele fraction slightly, which is less visible for small fitness costs. Likewise, the frequency of both resistant types is slightly reduced by a higher dominance of the fitness cost (Fig. 2 (B)). However, the expected fraction of heterozygotes in the long-term is similar for varying fitness costs. In contrast, the proportion of resistant homozygotes increases with a decreasing fitness cost, most extreme for minimal fitness costs. This is because Johnsongrass is predominantly self-pollinated. Self-fertilized heterozygous plants ($95\ \%$) produce only one-half of heterozygous seeds and one-quarter each of the homozygotes. Therefore we see an increased level of homozygosity under lower fitness costs on seed production. Genotype RR is expected to be more abundant in the long-run than RW for costs lower than $20 - 25\ \%$, depending on the

degree of dominance.

In natural populations of *Lolium rigidum* plants with target-site resistance against an ALS-inhibitor were found at a frequency ranging from $1 \cdot 10^{-5}$ to $1.2 \cdot 10^{-4}$ (Preston & Powles 2002). Délye et al. (2013) detected one individual carrying a specific ACCase target-site resistance in a sample of 685 *Alopecurus myosuroides* specimens collected prior to the introduction of ACCase-inhibiting herbicides. This corresponds to a resistance allele frequency of $7.3 \cdot 10^{-4}$. In our model, a proportion of $1 \cdot 10^{-5}$ resistance alleles and resistant individuals is expected in an untreated field for a $0.1\ \%$ fitness cost on seed production. Such a low fitness cost might not be detectable. Indeed for two out of three mutant ACCase alleles investigated by Menchari et al. (2008) in blackgrass *Alopecurus myosuroides* no associated fitness cost could be detected. However, for a fitness cost on seed production of $30\ \%$, reported for a mutant ACCase allele endowing herbicide resistance in *Sorghum halepense* (Panozzo & Sattin 2021), the expected resistance allele frequency and frequency of resistant types range from $3.3 \cdot 10^{-8}$ to $5.3 \cdot 10^{-8}$ and $4.9 \cdot 10^{-8}$ to $7.3 \cdot 10^{-8}$, respectively, depending on the dominance of this fitness cost. The initial prevalence of target-site resistance is thus considerably lower and in the order of the assumed mutation rate (Haughn & Somerville 1987, Harms & DiMaio 1991).

For the deterministic population dynamics and associated stochastic simulations, the long-term population composition obtained from Eq. (22) provided the genetic composition of the initial seed bank and rhizomes.

Resistance allele dominance and fitness cost dominance. We investigated how the dominance of resistance and fitness cost change the dynamics of population composition under recurrent herbicide application. An increase in the dominance of the fitness cost on seed production leads to a relatively small reduction in the overall fitness of the heterozygous type. It thereby slightly reduces the eventual abundance of the heterozygotes and the sensitive homozygotes (Fig. 3). A reduced degree of resistance dominance drastically lowers heterozygotes' fitness under herbicide application. This reduced growth rate strongly decreases the number of heterozygous and homozygous sensitive plants in the population.

Fitness cost. We used stochastic simulations of our population-based model to investigate the impact of the fitness cost on seed production on population dynamics and the probability of Johnsongrass escaping herbicide control. It is worth noting that the fitness cost determines the frequency of standing genetic variants and substantially affects the escape probability. Presumed that the resistant types manage to get established, the change in the population composition is faster under lower fitness cost on seed production (Fig. 4 (A)). The resistant homozygotes outcompete the heterozygotes and the sensitive homozygotes (see also Fig. S1).

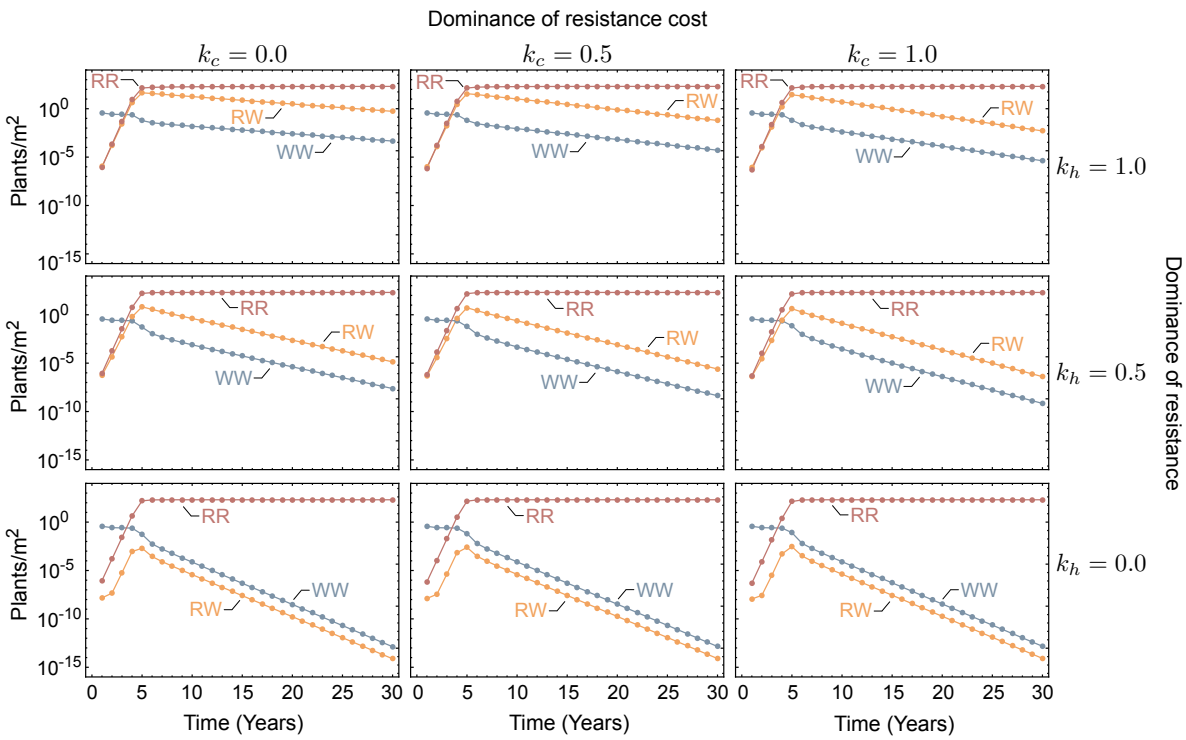

Figure 3: Deterministic changes in genotype composition of Johnsongrass plants (Eq. (7)) over 30 years of herbicide application for different degrees of dominance regarding resistance (k_h) and the fitness cost associated with resistance (k_c). Note that also the initial genotype composition differs according to the dominance of resistance and fitness cost.

Moreover, with a low fitness cost of $0.1\ \%$, the population escapes control by the herbicide in more than half of the 1000 simulation runs (Fig. 4 (B)). Most of these simulated populations start to grow again within the first years. This resurgence is due to a high initial frequency of resistance alleles for low fitness costs (compare Fig. 2). Thus, resistant individuals will likely be present in the field right from the start.

For a higher fitness cost of $30\ \%$, less than a quarter of the simulated populations regrow within 30 years. In such a case, we see a less skewed and more uniform distribution of escapes from control with almost no escapes in the first two years (Fig. 4 (B)). Due to a low initial frequency of resistant types (compare Fig. 2), the population rescue depends on the probability of a new mutation arising or resistant seeds in the seed bank germinating.

3.2 Sexual reproduction.

We investigated how the characteristics of Johnsongrass' sexual reproduction, namely self-pollination and seed bank formation, change the population dynamics under herbicide application.

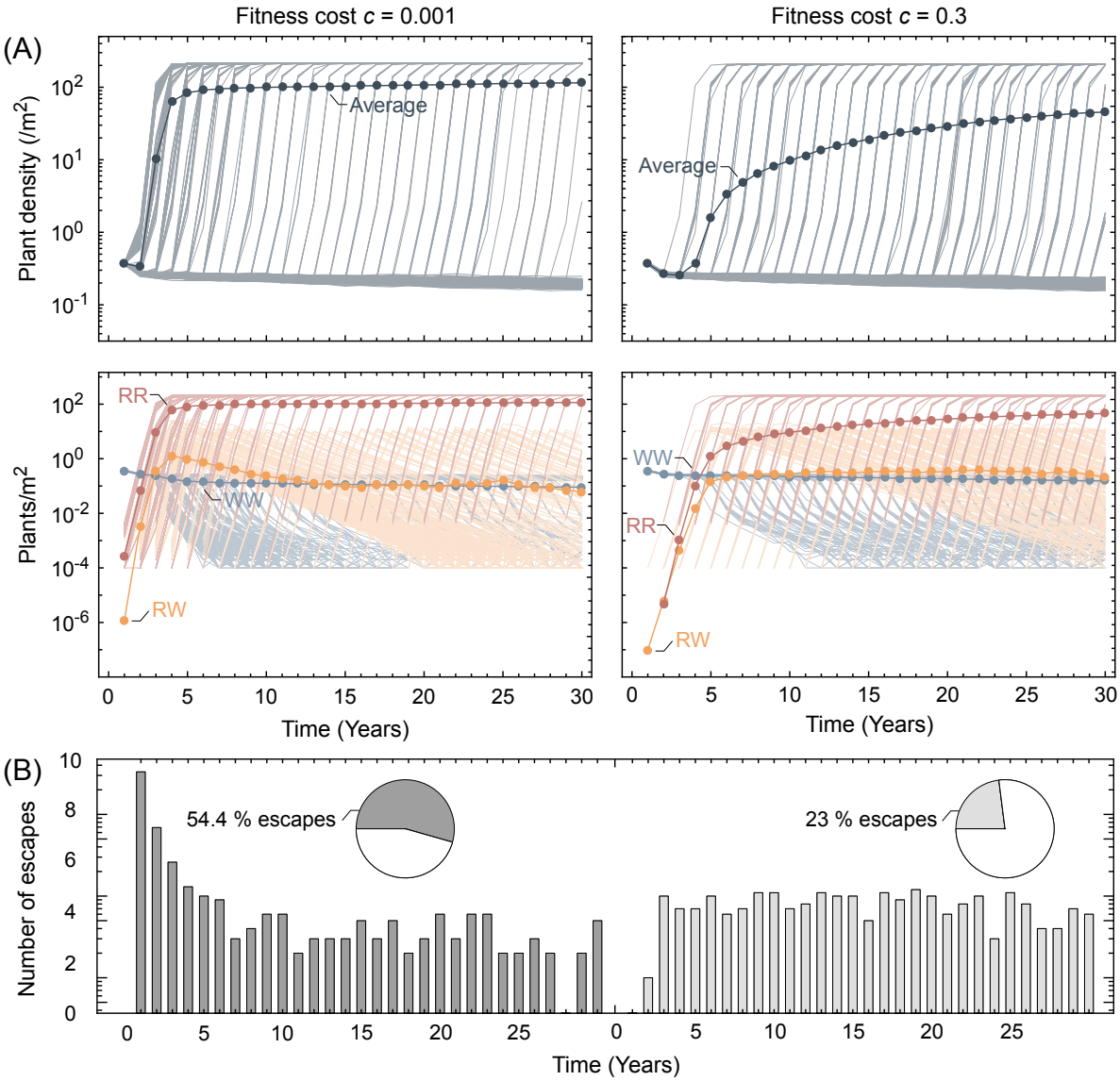

Figure 4: Simulated changes in Johnsongrass density per m^2 and genotype composition of plants (A) and distribution of escapes from control (B) over 30 years of herbicide application for different fitness costs c ($k_c = 0.5,\ k_h = 0.5$). Thick lines correspond to averages over 1000 simulation runs and thin lines depict individual runs (section 2.2). Note that also the initial genotype composition differs according to the fitness cost (compare Fig. 2 (B)).

Self-pollination. Selfing increases the level of homozygosity in the population. Hence the heterozygotes are significantly less abundant compared to a solely outcrossing population (Fig. 5 (A)). And the proportion of resistance alleles R increases faster under self-pollination (Fig. 5 (B)). Initially, it is surprising that with no self-pollination, the number of homozygous resistant seeds first decreases under herbicide application. This decrease is because the initial population composition derives under the assumption of no cross-pollination. The sensitive type dominates over the first years, producing a high fraction of non-resistant pollen. Therefore in a solely outcrossing population, pollination with non-resistant pollen prevails.

Seed bank. Most simulated populations that lack a seed bank go extinct within 30 under herbicide application and tillage (Fig. 6 (A)). At the same time, we see no extinctions within this time span when the simulated populations include a seed bank. In the latter case, some resistant seeds might be left in the seed bank, even if the resistant types do not establish during the early years of herbicide application. The weed population thus has the potential to escape from control even after being controlled at very low densities for many years. Moreover, since populations with a seed bank are larger, the probability of mutations arising increases. Therefore, the distribution of escapes from control over time, as observed in the stochastic simulations, is very wide for populations containing a seed bank (Fig. 6 (B)). The escapes aggregate to $7.6\ \%$ of the realizations. In contrast, in simulations without a seed bank, populations started to regrow solely in the first nine years accounting for $3\ \%$ of the runs. Without a seed bank, the sensitive type disappears in almost all cases (Fig. 6 (A)).

Contrary to this, most seed banks still contain some sensitive seeds after 30 years of recurrent herbicide application and tillage. The seed bank composition changes at a slower scale than the population structure in plants on the field, preserving genetic variation (see also Fig. 5).

3.3 Control strategies.

Combined control regimes. We examined the impact of integrating a second herbicide and tillage on Johnsongrass' population dynamics and resistance evolution. By combining herbicide and tillage, the population size is significantly reduced due to the higher winter mortality of rhizomes and increased herbicide efficiency on rhizome shoots (Fig. 7). Therefore mutations are less likely to arise, and the proportion of simulation runs in which the population regrows from resistant individuals also reduces. Nevertheless, the increased selective pressure with tillage causes the frequency of resistance alleles in the population to increase slightly faster than under herbicide application alone (Fig. 8). Our result contradicts the finding of Liu et al. (2019) that tillage delays the evolution of resistance. However, we could qualitatively

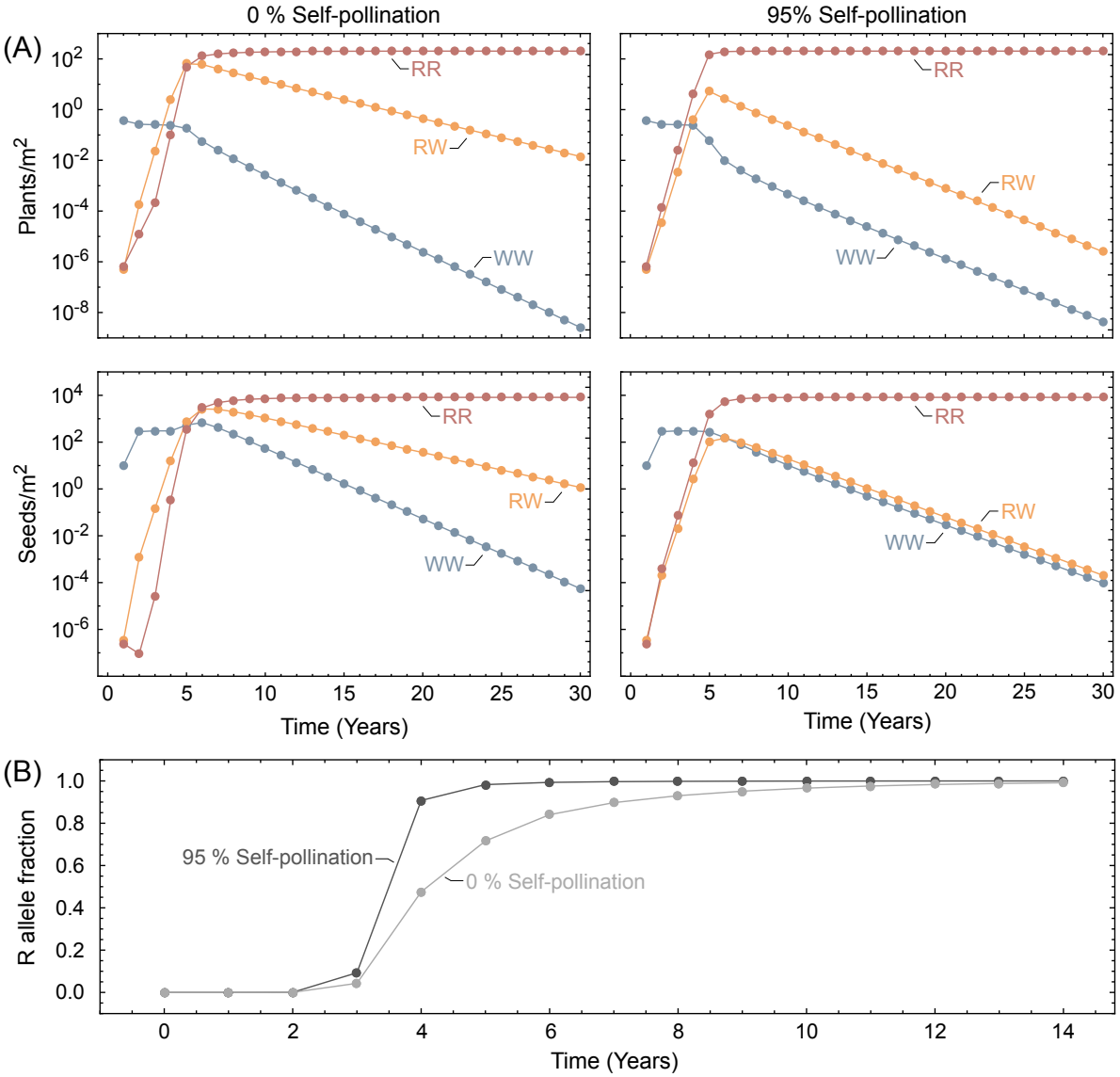

Figure 5: Deterministic changes in genotype composition of Johnsongrass plants (Eq. (7)) and seeds (Eq. (18)) (A) and change in the frequency of the resistance allele R in plants (Eq. (7)) (B) over 30 years of herbicide application depending on the ability of plants to self-pollinate (no selfing or 95 % selfing) ($k_c = 0.5,\ k_h = 0.5$).

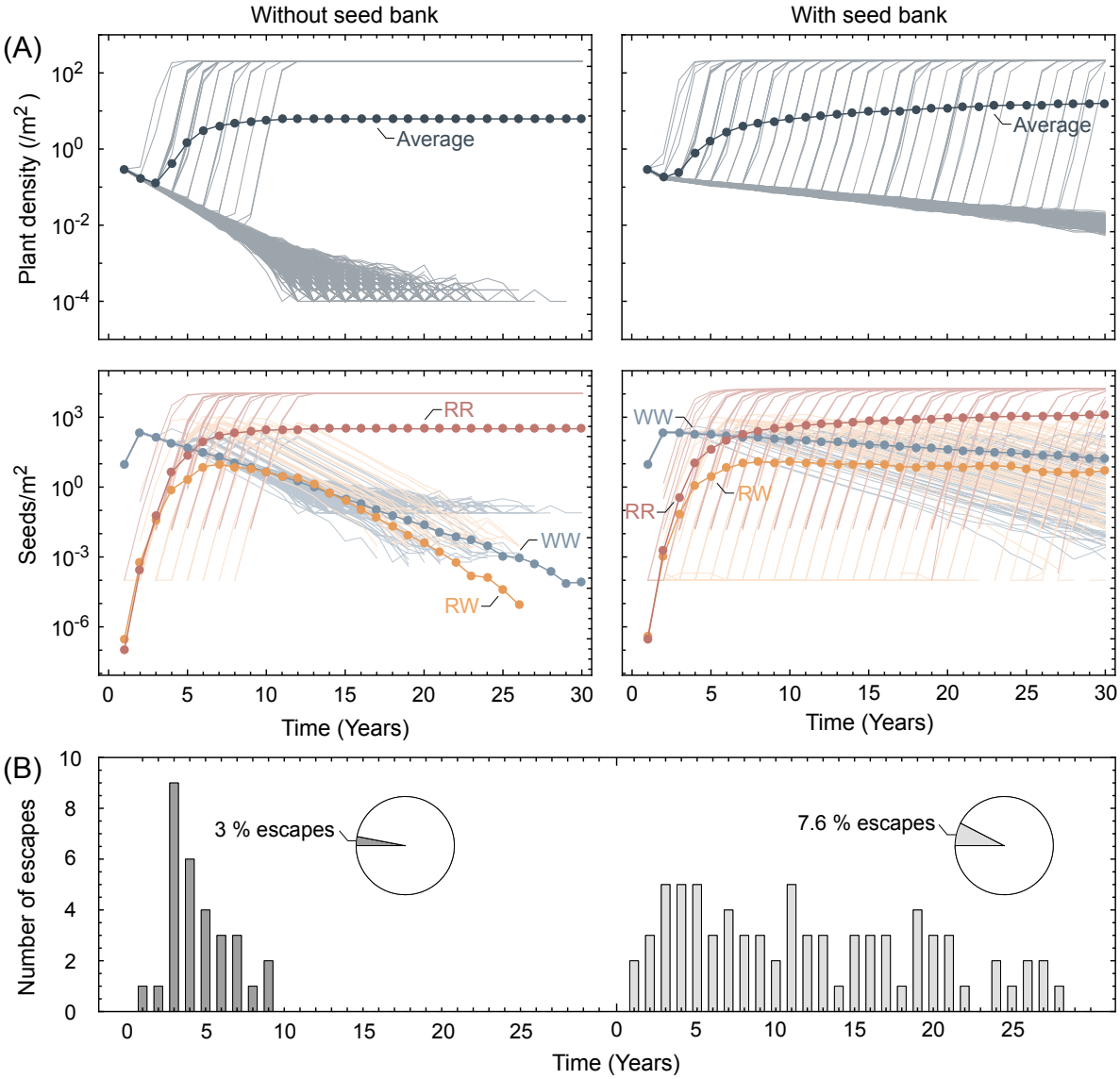

Figure 6: Simulated changes in Johnsongrass density per m^2 and genotype composition of seeds (A) and distribution of escapes from control (B) over 30 years of herbicide application and tillage depending on the formation of a seed bank ($k_c = 0.5,\ k_h = 0.5$). Thick lines correspond to averages over 1000 simulation runs and thin lines depict individual runs (section 2.2).

reproduce their result by using a parameter set similar to theirs (see Fig. S1). Therefore we believe the deviation is caused by the significantly lower reproductive capacity implemented by Liu et al. (2019). This assumption increases the effect of higher rhizome mortality, overcoming the increased selective pressure on shoots under low frequencies of the resistance allele.

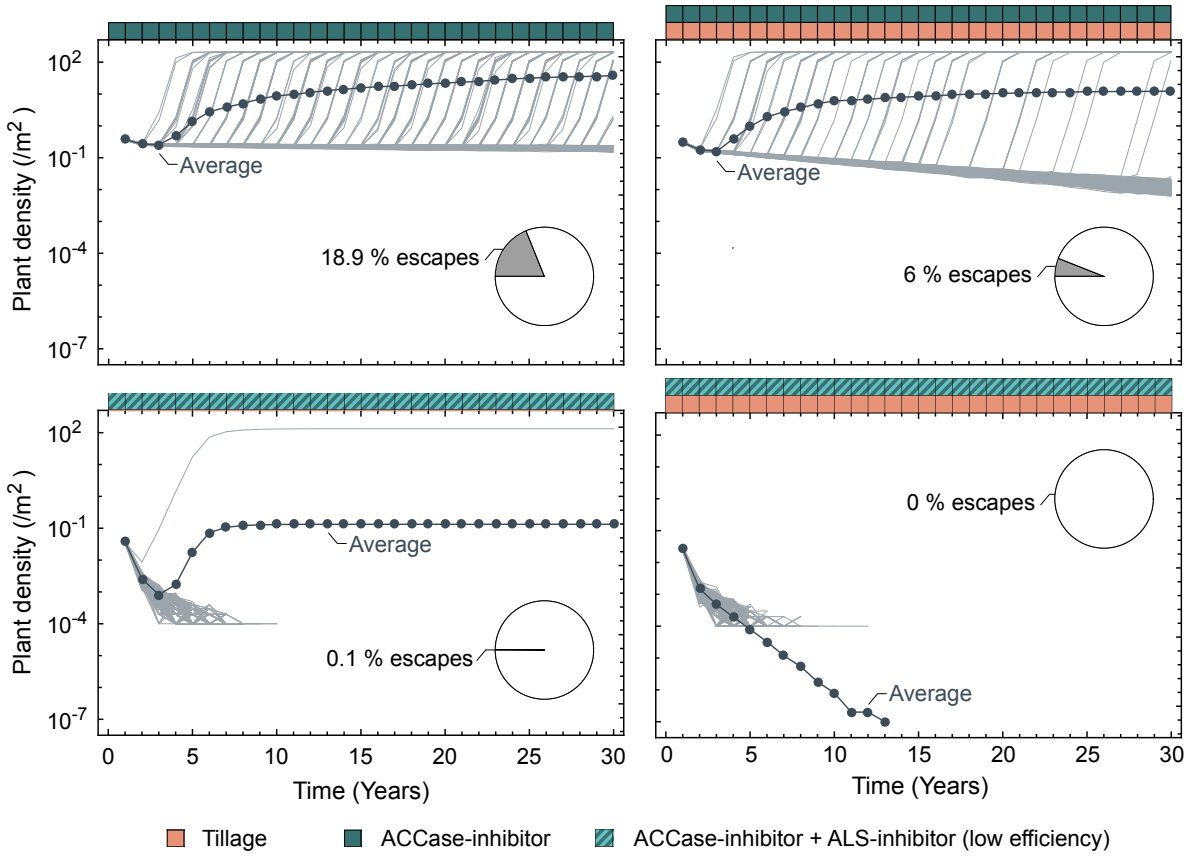

Figure 7: Simulated change in Johnsongrass density per m^2 and number of escapes from control for different control strategies over 30 years ($k_c = 0.5$, $k_h = 0.5$). Thick lines correspond to averages over 1000 simulation runs and thin lines depict individual runs (section 2.2). The distinct control strategies are from left to right, top to bottom: ACCase-inhibitor application, ACCase-inhibitor application combined with tillage, application of ACCase-inhibitor and ALS-inhibitor with low efficiency, application of ACCase-inhibitor and ALS-inhibitor with low efficiency combined with tillage.

Adding a second herbicide with a reduced efficiency makes the simulated populations go extinct within 15 years and escape very unlikely (Fig. 7). Furthermore, the second herbicide delays the resistance evolution against the first herbicide (Fig. 8). The reason is Johnsongrass' inherent ecology. As the herbicide efficiency is higher on seedlings than on rhizome shoots, the proportion of surviving plants that originate from rhizomes increases when a second herbicide

with a different mode of action is added. And the selective pressure acting on these plants is lower than on plants that emerge from seeds. This effect is even more substantial when the second herbicide acts with the same high efficiency as the first herbicide. However, suppose a herbicide with reduced efficiency is applied individually. In that case, the exceptionally high reproductive capacity of Johnsongrass allows the population to still grow (Fig. S2). The high density, in turn, leads to de novo mutations conferring herbicide resistance.

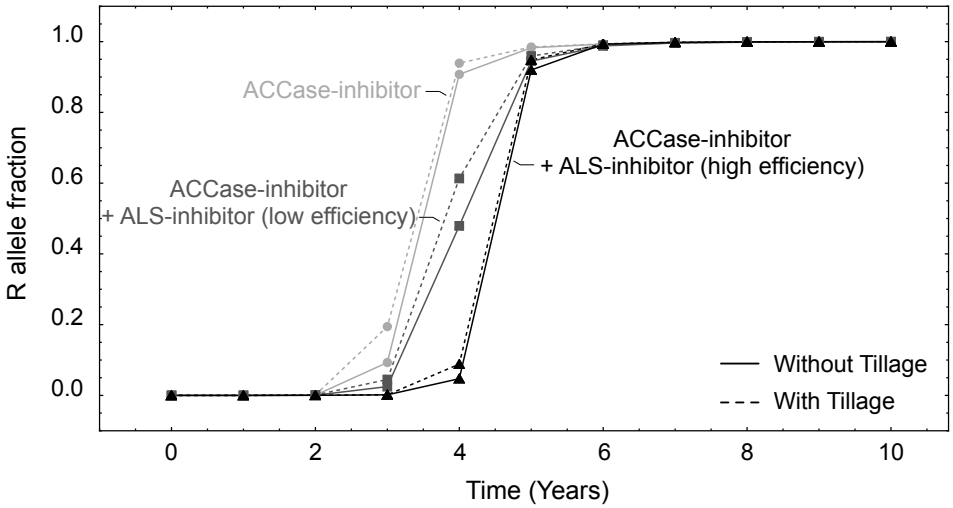

Figure 8: Deterministic change in the ACCase resistance allele fraction in plants (Eq. (7)) for different control strategies over 30 years ($k_c = 0.5$, $k_h = 0.5$). The distinct control strategies are: ACCase-inhibitor or ACCase-inhibitor and ALS-inhibitor with low or high efficiency applied solely or combined with tillage.

Herbicide rotation. We also investigated the effect of cycle length in a rotation of two herbicides on the number of escapes from control and the years of escape and resistance allele fixation (Fig. 9). The two herbicides differ in their mode of action and operate with equal efficiency. Cycle length refers here to the number of years one herbicide is applied recurrently before treatment switches to the other herbicide. Out of 10^5 simulation runs, the proportion of runs in which the population recovers ranges from 17.8 to 18.9 % without tillage and from 6.7 to 7.3 % with tillage. The lower values of these ranges are reached for rotations with cycle length greater than 4 years (Fig. 9 (A)). Overall the probabilities of escape from control are similar to that of the single herbicide simulations (compare Fig. 7). The average year of escape from control is the year in which the first plant fully resistant to at least one of the herbicides survives till reproduction. This average escape year shows a slight downward trend with increasing cycle length, varying between 15.2 and 15.9 years when the field is not tilled. Including tillage the range drops to $10.2 - 11.4$ years (Fig. 9 (A)). However, the expected year

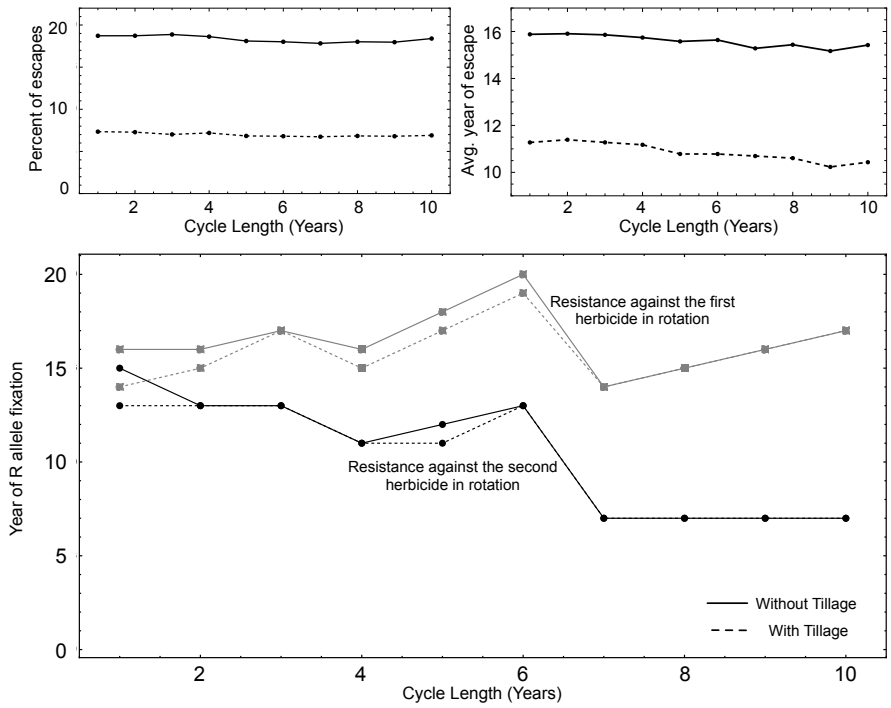

Figure 9: Average proportion of escapes from control, average year of escape and year of fixation of the resistance allele over 30 years depending on the cycle length in rotations of ACCase-inhibitor and ALS-inhibitor with equal efficiency ($k_c = 0.5$, $k_h = 0.5$). The cycle length corresponds to the number of years one herbicide is recurrently applied before the treatment switches to the other herbicide. (A) Presented values are the average over 10^5 stochastic simulation runs (section 2.2). (B) The year of resistance allele fixation is equivalent to the year in which the resistance allele frequency in plants (Eq. (7)), obtained from the deterministic model, reaches $99.5\ \%$.

of resistance allele fixation varies stronger with the speed of cycling (Fig. 9 (B)). For both, the herbicide applied first and the one applied second, the time till fixation is minimal with 7 and 14 years, respectively, for a cycle length of 7 years, regardless of tillage. This is the duration it takes for the resistance allele to fix in a mono treatment (compare Fig 8). The time till fixation of the resistance allele against the herbicide applied second is maximal for a cycle of length 6 years, with 20 years in the no-tillage regime and 19 years with tillage. Including tillage, the expected fixation time of the first resistance allele is at its maximum of 13 years for a cycle length of 1 to 3 or 6 years. In contrast, it reaches its maximum of 15 years for yearly rotation without tillage. This means the alternating selective pressure in herbicide rotations can delay the resistance evolution by more than the time individual herbicides are paused (compare Fig. 8). When a herbicide is not applied, plants sensitive to this herbicide have a higher reproductive capacity. Moreover, the average population density starts to increase later under fast herbicide rotation as compared to the application of a single herbicide (compare Fig. 8 and Fig. S3). The final average weed density reached after 30 years is 8.15 plants per m^2 for yearly rotation combined with tillage, considerably lower than 12.35 plants per m^2 for the combination of single herbicide application and tillage.

3.4 Conclusion and Outlook.

Our analysis suggests that target-site resistance with a frequency in untreated populations at least two orders of magnitude higher than the respective mutation rate is associated with a very low, probably undetectable, fitness cost. The resistance allele frequency in naive populations is expected to be similar to the magnitude of the mutation rate for fitness costs on seed production exceeding $12\ \%$. In other weed species, not all mutations endowing target-site resistance were reported to involve a fitness cost (Menchari et al. 2008). Furthermore, target-site resistance against ALS- and ACCase-inhibitors was found to have a natural frequency in the order of 10^{-5} to 10^{-4} in some weed species, which would, according to our results, correspond to an extremely low fitness cost (Preston & Powles 2002, Délye et al. 2013). These findings coincide with the conclusions of (Preston & Powles 2002). It is therefore likely that in Johnsongrass target-site mutations exist with very low or even no fitness cost. In this case, our simulations may underestimate the speed of resistance evolution and the risk of weed control failure.

Using simulations we show a more than doubled probability of escape from control under a low fitness cost of $0.1\ \%$ as compared to a fitness cost of $30\ \%$ on seed production. The latter cost was reported for a mutant ACCase allele endowing herbicide resistance in *Sorghum halepense* (Panozzo & Sattin 2021). Furthermore, the expected change in the population composition is faster under low fitness costs, and populations start to regrow earlier. Thus, if the fitness cost of target-site resistance is low, control of the weed population with a single

herbicide will likely fail quickly and should be avoided. In such a case, applying herbicide mixtures might be a good strategy, and integrating other control measures like tillage (Diggle et al. 2003, Peerzada et al. 2017).

Our results suggest that the fitness cost dominance has a minor impact on the prevalence of resistance and the population dynamics under control. Unlike the fitness cost of resistance, the herbicide resistance itself has no effect on a weed population in the absence of the herbicide. The dominance of resistance, therefore, does not affect the standing genetic variation. However, it determines the fitness of heterozygotes under herbicide application, their abundance, and the sensitive type's abundance. A high dominance of resistance initially increases the speed of resistance evolution. However, then the effect reverses, delaying resistance evolution. This effect is observed for diploids and tetraploid perennials (Holmes et al. 2022).

Holmes et al. (2022) found that target-site resistance alleles spread faster in haploids than in tetraploids, since tetraploid plants need to acquire twice as many mutations as diploids to reach the same proportion of resistance alleles. However, in Johnsongrass, the W2027C target-site mutation was not found to be present in more than two ACCase alleles (Lorentz 2014). The author argued that this might be caused by a fitness penalty or the absence of homologous recombination between Johnsongrass' two genomes. Since we use a diploid system, our model might overestimate the speed of resistance evolution if all four copies of ACCase and ALS genes acquire target-site mutations in Johnsongrass.

Our results indicate that resistance evolution is faster in a self-pollinated weed population than in a solely outcrossing population. Using a simulation study, Diggle et al. (2003) found that to successfully evolve resistance, populations of seed propagated annual weeds need to be larger if they are selfing compared to purely outcrossing populations They argue that this is due to the decreased number of heterozygous resistant individuals in the population. For calculations and simulations of the population dynamics, we used the initial composition of seeds and rhizomes derived from Eq. (22) under the assumption of pure selfing. However, we derived similar results with a control-free initialisation phase of 30 years for the population composition to stabilise under the assumed level of selfing. The simulations show that a seed bank significantly delays the extinction of a weed population under control. A seed bank preserves genetic variation and considerably increases the probability of establishing resistant mutants, leading to population regrowth. Therefore we emphasise the necessity of considering the details of sexual reproduction and seed bank formation when assessing the risk of weed control failure and resistance evolution. This caveat is valid even for weeds that can rapidly spread via asexual propagation. Other authors argued that resistance spreads mainly via asexual propagation when the first resistant plant gets established (Liu et al. 2019). While this might be partly true, considering the resistance cost on seed production and the possibility

that produced seeds are not resistant, resistance generally originates in the seeds. This is likely since mutations are most likely to arise during sexual reproduction. Even if standing genetic variation is present in the population, the number of seeds is considerably higher than the number of rhizomes. Therefore, resistant individuals are more likely to be in the seed bank. Overall, we conclude that controlling the seed bank is essential in managing seed and rhizome propagated perennial weeds like *Sorghum halepense*.

Furthermore, we show that the integration of soil tillage with herbicide application further reduces the Johnsongrass population in the field. We see no delay in the expected evolution of herbicide resistance, as observed by Liu et al. (2019). Nevertheless, the probability of Jonsongrass regrowth is more than halved by the reduction in population size. This result is in good agreement with field studies of Johnsongrass (McWhorter & Hartwig 1965) and blackgrass (Zeller et al. 2021). However, tillage increases the risk of soil erosion (Skøien et al. 2012) and diminishes soil water (Bescansa et al. 2006). Our study does not consider the risk of spreading resistant rhizomes and seeds with contaminated machinery.

According to our results, the binary herbicide mixture controls *Sorghum halepense* most effectively with minimal risk of the weed population escaping from control. We found that the resistance evolution is delayed by adding a second herbicide, with an increased effect for higher herbicide efficiencies. These results agree with other simulation studies demonstrating that herbicide mixtures effectively control resistance evolution and are superior to herbicide rotations (Diggle et al. 2003, Busi et al. 2020). These conclusions were derived assuming independent action of the herbicides, no cross-resistance and full application rates. Applying two herbicides at full rates not only includes a higher economic cost than a single herbicide but also increases the environmental impact. Furthermore, possible mechanisms of cross-resistance and synergistic or antagonistic interactions of the respective herbicides need to be explicitly considered. However, they are beyond the scope of the current study. A more comprehensive understanding of the effect of herbicide mixtures could be attained by incorporating herbicide interaction and cross-resistance into the present model and investigating different application rates and ratios.

Surprisingly, the probability of Johnsongrass regrowth was not reduced in our simulations for rotations of two herbicides with different target-sites compared to mono-treatments with these herbicides. Due to the tremendous reproductive capacity of Johnsongrass, subpopulations resistant to only one of the herbicides will, in our model, still grow over a rotation period with equal application of both herbicides. Nevertheless, the average population size starts increasing later under fast herbicide rotation. And it reaches a lower density within the 30 years simulated. For blackgrass, an average reduction in density of $59\ \%$ after seven years was reported for yearly rotation of herbicide site of action compared to mono-treatment (Zeller et al.

2021). This observation fits well with our finding of delayed onset of weed regrowth. Herbicide rotations have the potential to delay the evolution of target-site resistance. Regarding this we found no consistent advantage of fast or slow rotation. This result agrees with a simulation study by Diggle et al. (2003), in which yearly herbicide rotation was not superior to an 'expend and swap' approach. This aspect, however, only takes effect if a relevant cost of resistance induces a selective disadvantage of the resistant types in seasons the respective herbicide is not applied. Our findings agree with a simulation study of Busi et al. (2020) which found that binary rotations only delay resistance evolution if a fitness cost is assumed. However, they found more complex rotations, including four herbicides, to be more effective. One has to bear in mind here that target-site resistance is not necessarily associated with a considerable fitness cost (Menchari et al. 2008). Overall our results promise an advantage of herbicide rotations over mono-treatment in terms of a delayed population regrowth and resistance evolution, presuming a fitness penalty. Crop rotations, especially when they incorporate highly competitive crops like maize, can further reduce the population size of *Sorghum halepense* (Scopel et al. 1988, Weisberger et al. 2019, Zeller et al. 2021). Holmes et al. (2022) studied the effect of crop rotations along with rotations of herbicides. They found that the combination of crop rotation and a rotation of herbicide site of action can effectively control Johnsongrass and delay the evolution of herbicide resistance. However, they only included resistance against one of the herbicides, resulting from gene flow between resistant cultivated *Sorghum* and its wild relative. Our model can readily be extended to study the effect of crop rotation, as done in Bargués-Ribera & Gokhale (2020) in terms of pathogen evolution, and in general competition with the crop. This can be achieved by adding the corresponding competition terms in the equations for density dependant mortality (Eq. (9)) and reproduction (Eqs. (11) and (12)).

Besides standing genetic variation and de novo mutations, resistance can also arise from pollen-mediated gene flow from neighbouring resistant populations, the introduction of herbicide resistant rhizomes and seeds via contaminated equipment or gene flow from closely related resistant crops (Vrbničanin et al. 2017, Beckie et al. 2019). Maxwell et al. (1990) included gene flow from pollen and seed immigration in their population model of annual weeds. Holmes et al. (2022) modelled resistance evolution in Johnsongrass originating from gene flow between resistant *Sorghum* crops and its wild relative. Extending our model to a landscape-scale with intra- and inter-specific gene flow will provide a comprehensive assessment of resistance evolution in perennial weeds under control strategies. In general, as weed control measures always come with economic and ecological costs, further work is needed to evaluate the benefits of weed control together with the costs.

Acknowledgements. The authors thank the generous support from the Max Planck Society.

Data Availability and Analysis All code and preliminary plots are available on GitHub at `https://github.com/tecoevo/JohnsongrassDynamics`.

Code availability. `https://github.com/tecoevo/JohnsongrassDynamics`

References

Acciaresi, H. A. & Chidichimo, H. O. (2005), 'Ecophysiological response of Sorghum halepense populations to reduced rates of nicosulfuron', *Pesquisa Agropecuária Brasileira* **40**(6), 541–547.

Bagavathiannan, M. V. & Norsworthy, J. K. (2013), 'Postdispersal Loss of Important Arable Weed Seeds in the Midsouthern United States', *Weed Science* **61**(4), 570–579.

Bargués-Ribera, M. & Gokhale, C. S. (2020), 'Eco-evolutionary agriculture: Host-pathogen dynamics in crop rotations', *PLoS Computational Biology* **16**(1), e1007546.

Baucom, R. S. (2019), 'Evolutionary and ecological insights from herbicide-resistant weeds: what have we learned about plant adaptation, and what is left to uncover?', *New Phytologist* **223**(1), 68–82.

Beasley, C. A. (1970), 'Development of Axillary Buds from Johnsongrass Rhizomes', *Weed Science* **18**(2), 218–222.

Beckie, H. J., Busi, R., Bagavathiannan, M. V. & Martin, S. L. (2019), 'Herbicide resistance gene flow in weeds: Under-estimated and under-appreciated', *Agriculture, Ecosystems & Environment* **283**, 106566.

Bescansa, P., Imaz, M., Virto, I., Enrique, A. & Hoogmoed, W. (2006), 'Soil water retention as affected by tillage and residue management in semiarid spain', *Soil and Tillage Research* **87**(1), 19–27.

Bliss, C. I. (1939), 'The Toxicity of Poisons Applied Jointly', *Annals of Applied Biology* **26**(3), 585–615.

Busi, R., Powles, S. B., Beckie, H. J. & Renton, M. (2020), 'Rotations and mixtures of soil-applied herbicides delay resistance', *Pest Management Science* **76**(2), 487–496.

Délye, C., Deulvot, C. & Chauvel, B. (2013), 'Dna analysis of herbarium specimens of the grass weed alopecurus myosuroides reveals herbicide resistance pre-dated herbicides', *PLOS ONE* **8**(10).

Diggle, A. J., Neve, P. B. & Smith, F. P. (2003), 'Herbicides used in combination can reduce the probability of herbicide resistance in finite weed populations', *Weed Research* **43**(5), 371–382.

Dogan, M. N. & Boz, O. (2005), 'The concept of reduced herbicide rates for the control of johnsongrass (Sorghum halepense (L.) Pers.) in cotton during the critical period for weed control', *Journal of Plant Diseases and Protection* **112**, 71–79.

Egley, G. H. & Chandler, J. M. (1978), 'Germination and Viability of Weed Seeds After 2.5 Years in a 50-Year Buried Seed Study', *Weed Science* **26**(3), 230–239.

Firbank, L. G. & Watkinson, A. R. (1985), 'On the Analysis of Competition Within Two-Species Mixtures of Plants', *The Journal of Applied Ecology* **22**(2), 503.

Freckleton, R. P. & Stephens, P. A. (2009), 'Predictive models of weed population dynamics', *Weed Research* **49**(3), 225–232.

Ghersa, C., Satorre, E. & Esso, M. V. (1985), 'Seasonal Patterns of Johnsongrass Seed Production in Different Agricultural Systems', *Israel Journal of Plant Sciences* **34**(1), 24–30.

Harms, C. T. & DiMaio, J. J. (1991), 'Primisulfuron Herbicide-resistant Tobacco Cell Lines. Application of Fluctuation Test Design to in vitro Mutant Selection with Plant Cells', *Journal of Plant Physiology* **137**(5), 513–519.

Harris, T. E. (1963), *The Theory of Branching Processes*, Springer, Berlin.

Hartzler, R. G., Gover, A. & Stellingwerf, J. (1991), 'Factors Affecting Winter Survival of Johnsongrass (Sorghum halepense) Rhizomes', *Weed Technology* **5**(1), 108–110.

Haughn, G. & Somerville, C. R. (1987), *Selection for Herbicide Resistance at the Whole-Plant Level*, American Chemical Society, chapter 7, pp. 98–107.

Hernndez, M. J., Len, R., Fischer, A. J., Gebauer, M., Galdames, R. & Figueroa, R. (2015), 'Target-Site Resistance to Nicosulfuron in Johnsongrass (Sorghum halepense) from Chilean Corn Fields', *Weed Science* **63**(3), 631–640.

Holm, L. G., Plucknett, D. L., Pancho, J. V. & Herberger, J. P. (1977), *The World's Worst Weeds: Distribution and Biology*, East-West Center, University Press of Hawaii.

Holmes, K. H., Lindquist, J. L., Rebarber, R., Werle, R., Yerka, M. & Tenhumberg, B. (2022), 'Modeling the evolution of herbicide resistance in weed species with a complex life cycle', *Ecological Applications* **32**(1), e02473.

Holst, N., Rasmussen, I. A. & Bastians, L. (2007), 'Field weed population dynamics: a review of model approaches and applications', *Weed Research* **47**(1), 1–14.

Horowitz, M. (1972), 'Early Development of Johnsongrass', *Weed Science* **20**(3), 271–273.

Hull, R. J. (1970), 'Germination Control of Johnsongrass Rhizome Buds', *Weed Science* **18**(1), 118–121.

Johnson, D. B. & Norsworthy, J. K. (2014), 'Johnsongrass (Sorghum halepense) Management as Influenced by Herbicide Selection and Application Timing', *Weed Technology* **28**(1), 142–150.

Johnson, W. G. & Frans, R. E. (1991), 'Johnsongrass (Sorghum halepense) Control in Soybeans (Glycine max) with Postemergence Herbicides', *Weed Technology* **5**(1), 87–91.

Johnson, W. G., Li, J. & Wait, J. D. (2003), 'Johnsongrass Control, Total Nonstructural Carbohydrates in Rhizomes, and Regrowth After Application of Herbicides Used in Herbicide-Resistant Corn (Zea mays)1', *Weed Technology* **17**(1), 36–41.

Keeley, P. E. & Thullen, R. J. (1979), 'Influence of Planting Date on the Growth of Johnsongrass (Sorghum halepense) from Seed', *Weed Science* **27**(5), 554–558.

Klein, P. & Smith, C. M. (2021), 'Invasive Johnsongrass, a threat to native grasslands and agriculture', *Biologia* **76**(2), 413–420.

Kreiner, J. M., Stinchcombe, J. R. & Wright, S. I. (2017), 'Population Genomics of Herbicide Resistance: Adaptation via Evolutionary Rescue', *Annual Review of Plant Biology* **69**(1), 1–25.

Krenchinski, F., Albrecht, A., Albrecht, L., Villetti, H., Orso, G., Barroso, A. & Victoria, R. (2015), 'Germination and Dormancy in Seeds of Sorghum halepense and Sorghum arundinaceum', *Planta Daninha* **33**(2), 223–230.

Landry, R. L., Stephenson, D. O. & Woolam, B. C. (2016), 'Glufosinate Rate and Timing for Control of Glyphosate-Resistant Rhizomatous Johnsongrass (Sorghum halepense) in Glufosinate-Resistant Soybean', *International Journal of Agronomy* **2016**, 1–6.

Legutzamón, E. S. (1986), 'Seed survival and patterns of seedling emergence in Sorghum halepense (L.) Pers.', *Weed Research* **26**(6), 397–404.

Liu, C., Scursoni, J. A., Moreno, R., Zelaya, I. A., Muñoz, M. S. & Kaundun, S. S. (2019), 'An individual-based model of seed- and rhizome-propagated perennial plant species and sustainable management of Sorghum halepense in soybean production systems in Argentina', *Ecology and Evolution* **9**(17), 10017–10028.

Loddo, D., Masin, R., Otto, S. & Zanin, G. (2012), 'Estimation of base temperature for Sorghum halepense rhizome sprouting', *Weed Research* **52**(1), 42–49.

Lolas, P. C. & Coble, H. D. (1980), 'Johnsongrass (Sorghum halepense) growth characteristics as related to rhizome length', *Weed Research* **20**(4), 205–210.

Lorentz, L. (2014), Herbicide Resistance - Molecular and Physiological Characterization of the Glyphosate Resistant Weeds Amaranthus ssp. and Sorghum ssp., PhD thesis, Rheinische Friedrich-Wilhelms-Universität Bonn.

Maxwell, B. D., Roush, M. L. & Radosevich, S. R. (1990), 'Predicting the Evolution and Dynamics of Herbicide Resistance in Weed Populations', *Weed Technology* **4**(1), 2–13.

McWhorter, C. G. (1961), 'Morphology and Development of Johnsongrass Plants from Seeds and Rhizomes', *Weeds* **9**(4), 558.

McWhorter, C. G. (1971), 'Growth and Development of Johnsongrass Ecotypes', *Weed Science* **19**(2), 141–147.

McWhorter, C. G. & Hartwig, E. E. (1965), 'Effectiveness of Preplanting Tillage in Relation to Herbicides in Controlling Johnsongrass for Soybean Production1', *Agronomy Journal* **57**(4), 385–389.

Menchari, Y., Chauvel, B., Darmency, H. & Délye, C. (2008), 'Fitness costs associated with three mutant acetyl-coenzyme A carboxylase alleles endowing herbicide resistance in blackgrass Alopecurus myosuroides', *Journal of Applied Ecology* **45**(3), 939–947.

Ohadi, S., Hodnett, G., Rooney, W. & Bagavathiannan, M. (2018), 'Gene Flow and its Consequences in Sorghum spp.', *Critical Reviews in Plant Sciences* **36**(5-6), 1–19.

Panozzo, S. & Sattin, M. (2021), 'Fitness Costs Associated to an Ile2041Asn Mutation in the Geophyte Sorghum halepense Resistant to ACCase-Inhibiting Herbicides', *Frontiers in Agronomy* **3**, 711840.

Paterson, A. H., Kong, W., Johnston, R. M., Nabukalu, P., Wu, G., Poehlman, W. L., Goff, V. H., Isaacs, K., Lee, T.-H., Guo, H., Zhang, D., Sezen, U. U., Kennedy, M., Bauer, D.,

Feltus, F. A., Weltzien, E., Rattunde, H. F., Barney, J. N., Barry, K., Cox, T. S. & Scanlon, M. J. (2020), 'The Evolution of an Invasive Plant, Sorghum halepense L. ('Johnsongrass')', *Frontiers in Genetics* **11**, 317.

Peerzada, A. M., Ali, H. H., Hanif, Z., Bajwa, A. A., Kebaso, L., Frimpong, D., Iqbal, N., Namubiru, H., Hashim, S., Rasool, G., Manalil, S., Meulen, A. v. d. & Chauhan, B. S. (2017), 'Eco-biology, impact, and management of Sorghum halepense (L.) Pers.', *Biological Invasions* pp. 1–19.

Preston, C. & Powles, S. B. (2002), 'Evolution of herbicide resistance in weeds: initial frequency of target site-based resistance to acetolactate synthase-inhibiting herbicides in Lolium rigidum', *Heredity* **88**(1), 8–13.

Sager, G. R. & Mortimer, A. M. (1976), 'An approach to the study of the population dynamics of plants with special reference to weeds', *Applied Biology* **1**, 1–47.

Scarabel, L., Panozzo, S., Savoia, W. & Sattin, M. (2014), 'Target-Site ACCase-Resistant Johnsongrass (Sorghum halepense) Selected in Summer Dicot Crops', *Weed Technology* **28**(2), 307–315.

Schwinning, S., Meckel, H., Reichmann, L. G., Polley, H. W. & Fay, P. A. (2017), 'Accelerated development in Johnsongrass seedlings (Sorghum halepense) suppresses the growth of native grasses through size-asymmetric competition', *PLOS ONE* **12**(5), e0176042.

Scopel, A. L., Ballare, C. L. & Ghersa, C. M. (1988), 'Role of Seed Reproduction in the Population Ecology of Sorghum halepense in Maize Crops', *The Journal of Applied Ecology* **25**(3), 951.

Skøien, S. E., Børresen, T. & Bechmann, M. (2012), 'Effect of tillage methods on soil erosion in norway', *Acta Agriculturae Scandinavica, Section B — Soil & Plant Science* **62**(sup2), 191–198.

Stoller, E. (1977), 'Differential Cold Tolerance of Quackgrass and Johnsongrass Rhizomes', *Weed Science* **25**(4), 348–351.

Torra, J., Montull, J. M., Taberner, A., Onkokesung, N., Boonham, N. & Edwards, R. (2021), 'Target-Site and Non-target-Site Resistance Mechanisms Confer Multiple and Cross- Resistance to ALS and ACCase Inhibiting Herbicides in Lolium rigidum From Spain', *Frontiers in Plant Science* **12**, 625138.

Tóth, V. & Lehoczky, E. (2006), 'Investigations on the germination depth of Johnson grass (Sorghum halepense [L.] pers)', *Communications in Agricultural and Applied Biological Sciences* **71**(3), 803—808.

Tuesca, D., Puricelli, E., Nisensohn, L., Faccini, D. & Papa, J. (1999), 'Decision criteria for optimizing postemergence johnsongrass control in soybean crops in Argentina', *Pesquisa Agropecuária Brasileira* **34**(5), 749–753.

Vidrine, P. R. (1989), 'Johnsongrass (Sorghum halepense) Control in Soybeans (Glycine max) with Postemergence Herbicides', *Weed Technology* **3**(3), 455–458.

Vila-Aiub, M. M., Balbi, M. C., Gundel, P. E., Ghersa, C. M. & Powles, S. B. (2007), 'Evolution of Glyphosate-Resistant Johnsongrass (Sorghum halepense) in Glyphosate-Resistant Soybean', *Weed Science* **55**(6), 566–571.

Vrbničanin, S., Božić, D. & Pavlović, D. (2017), Gene flow from herbicide-resistant crops to wild relatives, *in* Z. Pacanoski, ed., 'Herbicide Resistance in Weeds and Crops', IntechOpen, Rijeka, chapter 3.

Warwick, S. I. & Black, L. (1983), 'The biology of Canadian weeds. 61. Sorghum halepense (L.) Pers.', *Canadian Journal of Plant Science* **63**, 997–1014.

Warwick, S. I., Phillips, D. & Andrews, C. (1986), 'Rhizome depth: the critical factor in winter survival of Sorghum halepense (L.) Pers. (Johnson grass)', *Weed Research* **26**(6), 381–388.

Watkinson, A. (1980), 'Density-dependence in single-species populations of plants', *Journal of Theoretical Biology* **83**(2), 345–357.

Weisberger, D., Nichols, V. & Liebman, M. (2019), 'Does diversifying crop rotations suppress weeds? A meta-analysis', *PLoS ONE* **14**(7), e0219847.

Williams, R. & Ingber, B. (1977), 'The Effect of Intraspecific Competition on the Growth and Development of Johnsongrass under Greenhouse Conditions', *Weed Science* **25**(4), 293–297.

Winton-Daniels, K., Frans, R. & McClelland, M. (1990), 'Herbicide Systems for Johnsongrass (Sorghum halepense) Control in Soybeans (Glycine max)', *Weed Technology* **4**(1), 115–122.

Yoda, K., Kira, T., Ogawa, H. & Hozumi, K. (1963), 'Self-Thinning in Overcrowded Pure Stands under Cultivated and Natural Conditions', *Journal of Biology* **14**, 107–129.

Zeller, A. K., Zeller, Y. I. & Gerhards, R. (2021), ‘A long-term study of crop rotations, herbicide strategies and tillage practices: Effects on Alopecurus myosuroides Huds. Abundance and contribution margins of the cropping systems’, *Crop Protection* **145**, 105613.

Supporting Information

Model parameters

Seed germination: Germination of Johnsongrass seeds was reported to be significantly influenced by temperature, light and burial depth (Horowitz 1972, Tóth & Lehoczky 2006, Krenchinski et al. 2015). Keeley & Thullen (1979) observed germination frequencies in the range of 24 to 91.5 % within 3 weeks for monthly sowing from March to October. In another study no seed germination was documented at temperatures under 19°C and a maximum of 64 % germination within 15 days was found at 39°C (Horowitz 1972). However, Krenchinski et al. (2015) reported considerably lower germination frequencies as well as a lower optimal germination temperature and reduced germination in the absence of light. They documented maximum germination at 30°C with 18.50 and 13.75 % in the presence and absence of light, respectively. Seed germination of Johnsongrass decreases with increasing burial depth from reported 64 % germination from 1 cm depth to 6 % from a sowing depth of 25 cm (Tóth & Lehoczky 2006).

However, the population level model does not account for these environmental factors. Warwick & Black (1983) found a mean seed germination frequency of 38 % in overwintering populations of *Sorghum halepense*. This observation agrees with a study by Legutzamón (1986). Therein, 37 % of seeds left on the soil surface germinated within the first year and 25 % when the seeds were transferred from the surface to 15 cm depth in autumn, simulating tillage practice. Our germination probability $g = 0.3$ is in between these values to account for seed burial due to natural causes or tillage. It is also consistent with the mean germination frequency documented by Egley & Chandler (1978).

Rhizome buds sprouting: In Johnsongrass, the development of rhizome buds is markedly controlled by apical dominance (Beasley 1970, Hull 1970). Rhizome apexes and emerged shoots partially suppress growth from adjacent buds (Beasley 1970, Hull 1970). Moreover, on branched rhizomes, secondary rhizomes influence the sprouting of axillary buds on the primary rhizome in an inverse manner (Beasley 1970). Bud sprouting and shoot growth in *Sorghum halepense* are reported to be highly temperature dependant (Hull 1970, Horowitz 1972), with a threshold temperature of 10°C needed, and to vary between populations (Loddo et al. 2012). In a study of Loddo et al. (2012) with 4-node rhizome segments shoot emergence was highest at 16°C, with a between population average of 48 %. Over the temperature range of $10 - 24$°C a total of 26 and 39 % of the rhizome nodes developed sprouts in the two studied populations. Hull (1970) documented an average bud germination on one-node rhizome pieces of 14 and 92 % at 15 and 30°C, respectively. However, Keeley & Thullen (1979) de-

tected no relevant influence of temperature on rhizome bud sprouting ($56\ \%$ at $18 - 35°$C) in their experiments, but a major impact on shoot development, with shoots emerged from 13 and $88\ \%$ of sprouted buds at 18 and $24 - 35°$C, respectively.

Our sprouting probability for buds on rhizomes cut into pieces by tillage practice is $30\ \%$, in agreement with the data of Loddo et al. (2012). Apical meristems, emerged shoots, and lateral rhizomes can inhibit axillary bud development on branched rhizomes (Hull 1970). Therefore, we assume a reduced sprouting probability of $20\ \%$ for no-tillage regimes.

Maximum plant density: *Sorghum halepense* produces tufts as a result of tillering, i.e. the development of secondary shoots from the crown area of existing plants (McWhorter 1971, Horowitz 1972). We do not explicitly model the production of tillers. However, the process of tillering is reflected in density-dependent reproduction. Lolas & Coble (1980) reported a considerable variation in the number of tillers produced by a plant between different growing seasons, ranging between 5 and 30 tillers per plant 60 days after planting, with an average of 19 tillers per plant grown from long rhizome pieces. Keeley & Thullen (1979) counted on average 5 to 20 tillers per plant, depending on the planting date. We assume that a plant not affected by intraspecific competition can, on average, produce 20 tillers.

In their experiments McWhorter (1971) observed an extreme variation between different Johnsongrass ecotypes in the number of culms growing in an area of one square meter, ranging from 65 up to 226 culms m^{-2}. In another study 200 Johnsongrass culms were counted on average per m^2 in an untreated soybean field (Landry et al. 2016). Therefore we assume a possible highest density of 220 plants per m^2 after self-thinning from very high densities. However, the ceiling density reached in populations growing from low densities is lower.

Tillers contribute to the total culm density on the field. However, high densities delay or even inhibit tillering in Johnsongrass (Williams & Ingber 1977). We assume that intraspecific competition becomes appreciable at a density of 10 plants per m^2, i.e. 200 culms m^{-2}. In the study of Keeley & Thullen (1979) Johnsongrass was planted at this density, meaning each plant had an area of 0.1 m^2 to produce the reported yields.

Seed survival: Egley & Chandler (1978) documented no significant impact of the burial depth on seed longevity and an average of $62\ \%$ of seeds that were still viable after burial for 2.5 years. However, seeds were placed in polypropylene screen envelopes which prevented predation by animals. It was not recorded if unrecovered seeds decayed or germinated. In another study, likewise excluding predation and not tracking germination, more prolonged seed survival in deep layers (15 and 22.5 cm) as compared to seeds in shallow layers (soil surface and 7.5 cm) was noticed (Legutzamón 1986). After 3 years they recovered less than

15 % of viable seeds from the shallow layers, while 34.5 to 53.8 % of seeds in the deeper layers were still viable. Nevertheless, regardless of the burial depth, virtually no viable seeds were found in their experiments after 6 years. Moreover, Legutzamón (1986) carried out one experiment in bare soil with different treatments. Some treatments comprised regular changes between the soil surface and 15 cm depth to examine the effect of cultivation on seed longevity. They found maximum seed survival to be less than 2.5 years. However, they discussed that the absence of vegetation in that experiment might have contributed to a faster loss of viable seeds. Bagavathiannan & Norsworthy (2013) reported 64 to 75 % of seed loss due to predation from dispersal in autumn till next spring, when seeds where left on the soil surface (no-tillage). Most recovered seeds ($78-91$ %) were found buried in the soil. In the same time-period, they documented 6 to 17 % seed decay. Also, a loss in viability of 23 % was observed that increased to 52 % (including germination) till the subsequent autumn. Overall the mean proportion of active seed bank still present in spring, after seed loss due to predation, decay and loss in viability, was 10 to 11 % and reduced to 7 to 8 % in autumn, after subsequent seed loss through decay, loss in viability and germination (Bagavathiannan & Norsworthy 2013). They documented no significant impact of residue cover or burial depth on seed decay and viability loss.

We consider only post dispersal seed predation, as most surviving seeds are buried in the soil and therefore subsequently inaccessible for predators, at a proportion of 75 % (Bagavathiannan & Norsworthy 2013). Our model includes 17 % seed decay and 23 % loss in viability from time of seed dispersal in autumn till next spring. We choose 24 % and 31 % yearly seed loss due to decay and loss in viability, respectively. With that seed loss in our model is in the range reported by Bagavathiannan & Norsworthy (2013), but higher than found in the undisturbed experiments of Egley & Chandler (1978) and Legutzamón (1986). However, our parameters are in good agreement with the simulated cultivation experiments conducted by Legutzamón (1986). Bagavathiannan & Norsworthy (2013) calculated the proportion of predation, correcting for the proportion of decayed seeds. Therefore, viable freshly produced seeds are lost to 0.94 % ($1-(1-0.75-0.17)(1-0.23) \approx 0.94$) in our model, and the yearly loss of viable seeds in the seed bank is 48 % ($1-(1-0.24)(1-0.31) \approx 0.48$).

Rhizome survival: Winter mortality of Johnsongrass rhizomes is highly dependent on the soil temperature determined by climatic conditions and burial depth (Stoller 1977, Warwick et al. 1986, Hartzler et al. 1991). Stoller (1977) documented about 5 % of rhizomes surviving at a burial depth of 20 cm and no survival above 20 cm depth in the first winter with a temperature record of -9°C at 20 cm. In the second milder winter, more than 60 % of the rhizomes survived at 20 cm and rhizome survival increased with increasing depth in soil

up to over 90 %. For overwintering populations Warwick et al. (1986) reported a dramatic reduction of rhizome survival at 20 cm depth to 20 % in more northern climate compared to 97 % survival under milder climatic conditions. They also recorded a survival dependency on burial depth, with only one out of 16 rhizomes surviving at a burial depth of 10 cm and 87.5 % winter survival at 30 and 50 cm. Hartzler et al. (1991) also found rhizome survival directly related to burial depth. With 5 and 25 % survival of 7.5 and 15 cm long rhizome pieces buried in soil 6 cm deep, respectively, in shallow layers survival of longer rhizome pieces was higher. Simultaneously, they did not notice a significant difference at 25 cm depth, where survival ranged between 85 and 87 %. Most rhizome biomass and nodes were found in the upper 30 cm, with the highest amount between 7.5 and 15 cm (Hartzler et al. 1991).

Our model does not account for the depth of rhizome burial or climatic conditions. We use the rhizome survival and distribution data published by Hartzler et al. (1991) to calculate the average winter mortality of long rhizome segments $d_Z = 0.35$, which is used in our model as the winter mortality in the no-tillage regime. Tillage cuts Johnsongrass rhizomes into smaller pieces and partly transports them to the soil surface. We assume that under control regimes comprising tillage, the share of rhizome pieces in the shallow layer is increased by 10 %. Together with the rhizome fragmentation this leads to higher average winter mortality of $d_Z = 0.6$.

All non-germinated rhizomes rotted in the experiments of Warwick et al. (1986). Furthermore, after sprouting, the rhizomes of Johnsongrass disintegrate within 19 to 24 days (McWhorter 1961). Therefore we only consider winter survival and assume that rhizomes can only give rise to shoots in the subsequent growing season.

Herbicide efficiency The herbicide efficiency on Johnsongrass aboveground material depends on the developmental stage of the plants, with small young plants killed with higher efficiency than larger older plants (Tuesca et al. 1999, Dogan & Boz 2005, Johnson & Norsworthy 2014). The efficiency also varies between years due to environmental factors and application timing (Vidrine 1989, Tuesca et al. 1999).

Johnson et al. (2003) and Johnson & Norsworthy (2014) report the investigated ACCase-inhibitors to have a higher efficiency on *Sorghum halepense* than ALS-inhibitors included in their studies. A single application of the ACCase-inhibitor Quizalofop led to 97 % reduction in Johnsgrass biomass 3 weeks after treatment. In contrast, single applications of the ALS-inhibitors Nicosulfuron and Primisulfuron reduced fresh weight biomass by only 88 % (Johnson et al. 2003). In the second study, a higher rate of the ACCase-inhibitor Clethodim controlled small plants (15 and 30 cm) at 97 % 2 weeks after treatment while Nicosulfuron provided 90 % control of 15 cm Johnsongrass and only 74 % control of 30 cm plants (Johnson

& Norsworthy 2014). Even though they noticed Johnsongrass regrowth after 2 weeks, stand reduction 4 weeks after treatment compared to an untreated control was similar to the initial weed control.

Due to insufficient translocation of active ingredients and the inactive state of rhizome nodes, rhizomes are controlled inadequately (Beasley 1970). Regrowth from the latter may occur, resulting in a reduced herbicide efficiency on rhizome shoots (Beasley 1970, Tuesca et al. 1999). In a study of Vidrine (1989) the average control of Johnsongrass seedlings provided by different ACCase-inhibiting herbicides was $99\ \%$ 3 weeks after treatment and $95\ \%$ for rhizome shoots. Sethoxidim failed to effectively control Johnsongrass shoots in one year of the experiment,though. Excluding sethoxidim, the other three ACCase-inhibitors controlled seedlings and shoots at 99.8 and $98.5\ \%$, respectively. Johnsongrass control was reduced 12 weeks after treatment. Nevertheless, with one exception, control was still at least $86\ \%$ and in most cases, even above $90\ \%$. Winton-Daniels et al. (1990) reported that regrowth occurred after all treatments with ACCase-inhibitors, but that sequential treatments typically control Johnsongrass better than single applications. A second herbicide application later in the season reduces the time for Johnsongrass plants to regrow. Therefore, regrown plants might not be able to produce new rhizome and viable seeds till the end of the growing season.

Given the above points, it is reasonable to assume that the final control provided by a sequential treatment is similar to the initial control a single application gives. As per Vidrine (1989) we choose the efficiency of ACCase-inhibiting herbicides to be $99.8\ \%$ on seedlings and to be reduced on shoots with $98.5\ \%$. As ALS-inhibiting herbicides are reported to control *Sorghum halepense* with a lower efficiency (Johnson et al. 2003, Johnson & Norsworthy 2014), we assumed an ALS-inhibitor efficiency of 92 and $90\ \%$ on seedlings and rhizome shoots, respectively. Mechanical practices inducing increased bud activity as a response to rhizome fragmentation allow for improved control of rhizomes and shoots by herbicide application (Hull 1970, Tuesca et al. 1999). Therefore herbicide efficiency on shoots of fragmented rhizomes is assumed to be intermediate between seedlings and shoots on intact rhizomes.

Inheritance: We suppose simple Mendelian inheritance. Mutations to the resistance allele R are assumed to arise with probability $\mu = 10^{-8}$, but back mutations are not considered (Haughn & Somerville 1987, Harms & DiMaio 1991). Therefore the inheritance matrices (17) used to calculate the number of seeds arising from cross-pollination (16) are given by,

$$\mathbf{M}^{\mathrm{WW}} = (1-\mu)^2 \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \tag{A.1}$$

$$\mathbf{M}^{\mathrm{RW}} = 2\,\mu\,(1-\mu) \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix} + (1-\mu) \begin{pmatrix} 0 & 0.5 & 1 \\ 0.5 & 0.5 & 0.5 \\ 1 & 0.5 & 0 \end{pmatrix}, \tag{A.2}$$

$$\mathbf{M}^{\mathrm{RR}} = \mu^2 \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix} + \mu \begin{pmatrix} 0 & 0.5 & 1 \\ 0.5 & 0.5 & 0.5 \\ 1 & 0.5 & 0 \end{pmatrix} + \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0.25 & 0.5 \\ 0 & 0.5 & 1 \end{pmatrix}. \tag{A.3}$$

Standing genetic variation

We make use of a well known result from the theory of matrices.

Theorem 1 (Perron-Frobenius (Harris 1963)) *Let $\mathbf{A}$ be a nonnegative k x k matrix, such that $\mathbf{A}^N$ is positive for some $N \in \mathbb{N}$. Then $\mathbf{A}$ has a positive and simple eigenvalue ρ that is in absolute value greater than any other eigenvalue of $\mathbf{A}$. ρ corresponds to positive right and left eigenvectors $\mathbf{u}$ and $\mathbf{v}$ which are the only nonnegative eigenvectors of $\mathbf{A}$. Moreover, for $n \in \mathbb{N}$ we have*

$$\mathbf{A}^n = \rho^n\,\mathbf{u}^T\mathbf{v} \,+\, \mathbf{R}^n\,, \tag{A.4}$$

with the normalisation $\mathbf{u}\,\mathbf{v}^T = 1$. Furthermore, $|\mathbf{R}^n| := \sum_{i,j=1}^{k} (R^n)_{ij} = \mathcal{O}(\alpha^n)$ for some α, $0 < \alpha < \rho$.

From Eq. (A.4) it follows that in the limit

$$\lim_{n\to\infty} \frac{\mathbf{A}^n}{\rho^n} = \mathbf{u}^T\mathbf{v}\,, \tag{A.5}$$

holds, i.e. $\mathbf{A}^n$ is dominated by ρ^n for $n \to \infty$.

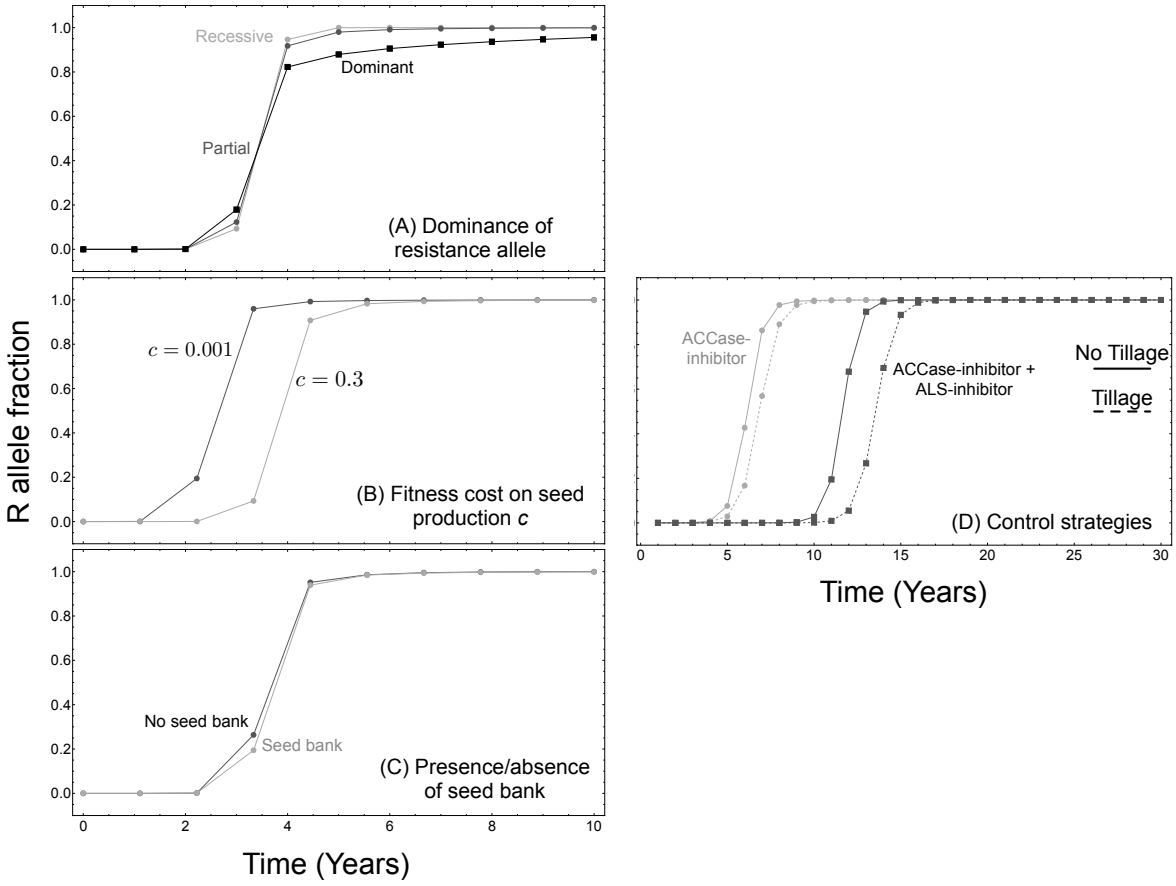

Fig. S1: Deterministic change in the resistance allele fraction in plants (Eq. (7)) over 30 years of herbicide application (A) for different degrees of dominance of resistance k_h ($k_c = 0.5$). Where recessive corresponds to $k_h = 0$, partial dominant to $k_h = 0.5$ and dominant to $k_h = 1$. (B) for different fitness costs c ($k_c = 0.5$, $k_h = 0.5$). (C) depending on the formation of a seed bank ($k_c = 0.5$, $k_h = 0.5$). (D) Deterministic change in the ACCase resistance allele fraction in plants for different control strategies over 30 years ($k_c = 0.5$, $k_h = 0.5$). The distinct control strategies are: ACCase-inhibitor or ACCase-inhibitor and ALS-inhibitor with equal efficiency applied solely or combined with tillage. This dynamics where obtained with a different version of our population-based model. Compared to the model presented inhere, this model does not include density dependence in the reproduction and no mortality of seeds before entering the seed bank. Furthermore, the parameter set is based on the one used by Liu et al. (2019) along with a yearly seed bank mortality of 20 %.

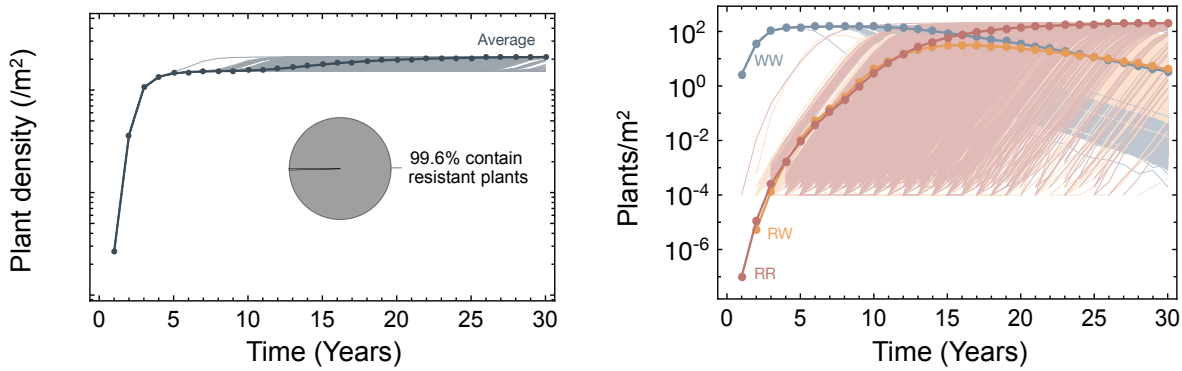

Fig. S2: Simulated change in Johnsongrass density per m^2 and genotype composition of plants over 30 years of ALS-inhibitor application with low efficiency ($k_c = 0.5$, $k_h = 0.5$). Thick lines correspond to averages over 1000 simulation runs and thin lines depict individual runs (section 2.2).

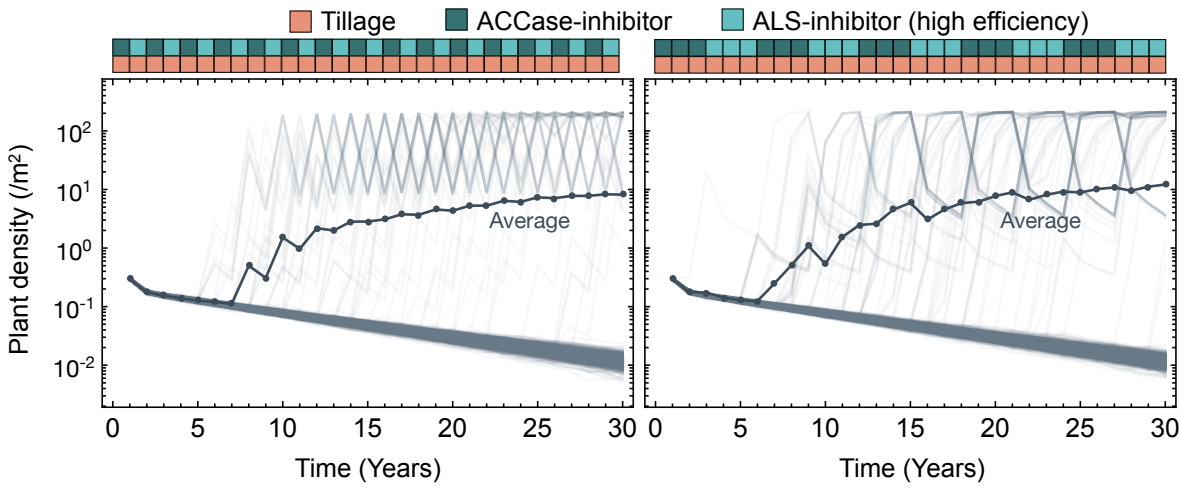

Fig. S3: Simulated change in Johnsongrass density per m^2 for different control strategies over 30 years ($k_c = 0.5$, $k_h = 0.5$). Thick lines correspond to averages over 1000 simulation runs and thin lines depict individual runs (section 2.2). The distinct control patterns are repeated sequentially; left: one year of ACCase-inhibitor application followed by one year of ALS-inhibitor application combined with yearly tillage; right: three years of ACCase-inhibitor application followed by three years of ALS-inhibitor application combined with yearly tillage.

Table 1: Model parameters, values and references.

Model parameters	Value	Comments	References
Sprouting probability		influenced by apical dominance and temperature; wide variation in reported values (Hull 1970, Horowitz 1972, Keeley & Thullen 1979)	Loddo et al. (2012)
without tillage	$g_Z = 0.2$		
after tillage	$g_Z^* = 0.3$		
Germination probability	$g = 0.3$	influenced by environmental factors not considered in the model, e.g. temperature, light and burial depth (Horowitz 1972, Tóth & Lehoczky 2006, Krenchinski et al. 2015)	Egley & Chandler (1978), Warwick & Black (1983), Legutzamón (1986)
Fitness cost on seed production	$c = 0.3$	in blackgrass only one mutation conferring resistance to ACCase-inhibitors was found to be associated with a fitness cost (Menchari et al. 2008)	Panozzo & Sattin (2021)
Proportion of self-pollination	$p_{self} = 0.95$	there are arguments for higher levels of outcrossing (Ohadi et al. 2018)	Warwick & Black (1983)
Mutation rate	$\mu = 10^{-8}$	estimates of spontaneous mutation rates of ALS-resistance alleles in *Arabidopsis thaliana* and *Nicotiana tabacum*	Haughn & Somerville (1987), Harms & DiMaio (1991)
Possible highest limit of plant density (m^{-2})	$m^{-1} = 220$	a lower ceiling density is reached for populations growing from low densities	McWhorter (1971), Williams & Ingber (1977), Landry et al. (2016)
Herbicide efficiency			
ACCase-inhibitor (high efficacy ALS-inhibitor)			
on seedlings	$h_L = 0.998$	influenced by environmental factors and application timing (Tuesca et al. 1999, Dogan & Boz 2005, Johnson & Norsworthy 2014); wide variation in reported regrowth (Vidrine 1989, Johnson & Frans 1991)	Vidrine (1989)
on shoots	$h_T = 0.985$		
on shoots after tillage	$h_T^* = 0.992$		
ALS-inhibitor			
on seedlings	$h_L = 0.92$		Johnson et al. (2003)
on shoots	$h_T = 0.90$		
on shoots after tillage	$h_T^* = 0.91$		

(continues)

Table 1: (continued)

Model parameters	Value	Comments	References
Rhizome loss winter mortality without tillage after tillage	 $d_Z = 0.35$ $d_Z^* = 0.6$	influenced by climatic conditions and burial depth not considered in the model (Stoller 1977, Warwick et al. 1986, Hartzler et al. 1991)	Hartzler et al. (1991)
Number of viable seeds produced per isolated plant	$f = 13,000$	wide variation between ecotypes and in reported numbers (McWhorter 1971, Ghersa et al. 1985, Acciaresi & Chidichimo 2005)	Keeley & Thullen (1979)
Seed loss fresh seeds old seeds in seed bank	 $d_S = 0.94$ $d_B = 0.48$	conflicting information on impact of burial depth and wide variation in reported values (Egley & Chandler 1978, Legutzamón 1986, Bagavathiannan & Norsworthy 2013)	Bagavathiannan & Norsworthy (2013)
area (m^2) required by a plant to produce f seeds and b rhizome buds	$a = 0.1$	assuming intraspecific competition becomes appreciable at a density of 10 plants m^{-2}	Keeley & Thullen (1979)
Initial density (m^{-2}) Seeds Rhizomes	 10 1		Liu et al. (2019)
Field size (m^2)	$A = 10,000$		Liu et al. (2019)