

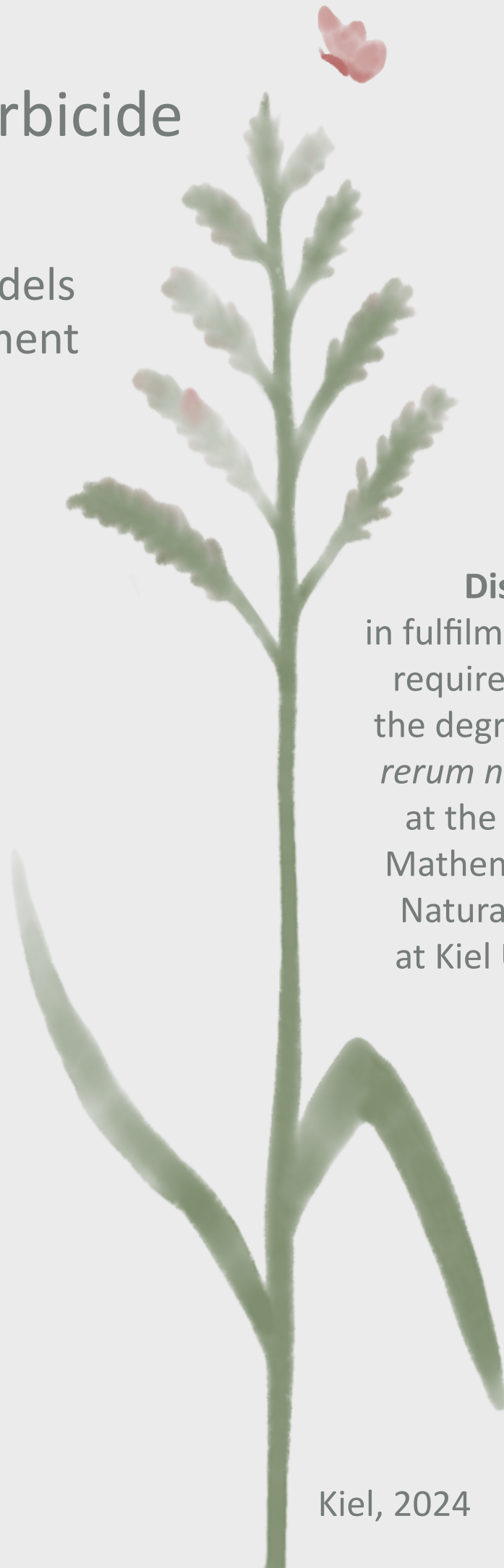
Assessing herbicide resistance

Mathematical models
of weed management
and resistance
evolution

Dissertation
in fulfilment of the
requirements for
the degree *Doctor
rerum naturalium*
at the Faculty of
Mathematics and
Natural Sciences
at Kiel University

submitted by
Dana Lauenroth

Kiel, 2024



First referee: Prof. Dr. Chaitanya S. Gokhale
Second referee: Prof. Dr. Eva H. Stukenbrock

Date of oral examination: 24th of September 2024

Kurzfassung

Seit den Anfängen der Landwirtschaft wird der Nutzpflanzenanbau durch schädliche Organismen – insbesondere Unkräuter, Schädlinge und Krankheitserreger – beeinträchtigt, die das Überleben der Pflanzen sowie die Menge und Qualität der erntefähigen Erzeugnisse mindern. Unkräuter sind die Organismen, welche die höchsten potenziellen Ertragsverluste verursachen. Seit jeher haben Landwirte nach Strategien zum Schutz ihrer Nutzpflanzen gesucht, wie das Unkrautjäten von Hand und später mechanische Bekämpfungsmaßnahmen. Die Kommerzialisierung selektiver Herbizide nach dem Zweiten Weltkrieg hat den Nutzpflanzenanbau revolutioniert, da sie eine einfache und hocheffiziente Lösung für die Unkrautbekämpfung boten und die maschinelle Ernte ermöglichten. Heute bedrohen weitverbreitete Resistenzen die Nachhaltigkeit von Herbiziden und Anbausystemen. Herbizidresistenz ist ein häufig vorkommendes Beispiel für eine rasche Anpassung an menschenvermittelte Selektion, das seit mehr als 60 Jahren extensiv untersucht wird, vor allem im Hinblick auf Methoden zur Begrenzung. Die genauen Determinanten von Geschwindigkeit und Art der Herbizidresistenzentwicklung und die Art und Weise, wie Bekämpfungsmethoden die zugrundeliegenden öko-evolutionären Prozesse verändern, sind jedoch nach wie vor kaum bekannt – insbesondere bei mehrjährigen Arten. In dieser Arbeit setze ich populationsdynamische Modelle ein, um kritische Faktoren zu identifizieren, die die Wahrscheinlichkeit, den Zeitpunkt und die Quelle der Resistenzanpassung bei einem mehrjährigen Unkraut bestimmen, und um Strategien zur Begrenzung der Herbizidresistenzentwicklung zu evaluieren.

Die in den einzelnen Kapiteln entwickelten Methoden sind auf spezifische Aspekte der Herbizidresistenzentwicklung und des Managements von sich über Samen und Rhizomen ausbreitenden mehrjährigen Pflanzen zugeschnitten. Im Kern analysiere ich, wie sich bestimmte Merkmale des Lebenszyklus – wie die Art der Reproduktion – und Details des Resistenzmechanismus – wie damit verbundenen Fitnesskosten – auf die Anpassung auswirken und bewerte die Nachhaltigkeit von Bekämpfungsmaßnahmen. Durch die Erfassung der Dynamik in der aktiven Unkrautpopulation und der inaktiven Samenbank auf Populationsebene ermöglichen mir die Modelle, die doppelte Wirkung einer Samenbank auf die Herbizidresistenzentwicklung zu untersuchen. Diese Arbeit zielt darauf ab, mechanistische Einblicke in die öko-evolutionären Prozesse zu liefern, die die Herbizidresistenzanpassung vorantreiben, und hebt den Wert populationsdynamischer Modelle für das Erlangen eines umfassenden Verständnisses dieser Dynamiken hervor.

Kapitel 2 konzentriert sich auf die Demographie einer mehrjährigen Unkrautpopulation und darauf, wie inhärente Unkrautmerkmale und -management ihre Dynamik bestimmen. In **Kapitel 3** wende ich Konzepte der Theorie evolutionärer Rettung an, um Fragen nach dem Ursprung adaptiver Variation und der Wahrscheinlichkeit und dem Zeitpunkt des Behandlungsversagens durch herbizidresistente Unkräuter zu beantworten.

Ich stelle fest, dass die Integration von Bodenbearbeitung bei Herbizideinsatz das Risiko eines Bekämpfungsversagens durch Minderung der Unkrautdichte wirksam verringert und dass Mixturen verschiedener Herbizidklassen Rotationen und Einzelanwendungen bei der Kontrolle des mehrjährigen Unkrauts und der Resistenzentwicklung überlegen sind. Unter Verwendung einer Matrixapproximation des Lebenszyklusmodells leite ich Schätzungen der vorhandenen genetischen Variation für Herbizidresistenz ab, die durch genetische Details des Resistenzmechanismus reguliert wird, wobei die assoziierten Fitnesskosten als primäre Determinante identifiziert werden. Durch die Erhöhung der Homozygotie beschleunigt Selbstbestäubung die Entwicklung von Wirkortresistenzen

bei deren Auftreten, verringert jedoch die Anpassung ausgehend von bestehenden Varianten, wenn die Resistenz (unvollständig) dominant vererbt wird. Die Dominanz eines Resistenzallels bestimmt, wie stark es der Selektion ausgesetzt ist; eine hohe Dominanz beschleunigt die initiale Ausbreitung, verzögert aber die Fixierung aufgrund der Maskierung sensitiver Allele bei Heterozygoten. Samenbanken bewahren genetische Informationen vorangegangener Selektion, was eine schnelle Reaktion auf wiederkehrende Selektionsregime ermöglicht, und verlängern die Zeit für Anpassung, da sie durch Unkrautmanagement verursachte Änderungen der Populationsgröße abpuffern. Indem sie einen wesentlichen Teil der Population vor den Selektionskräften schützen, verzögern Samenbanken die Fixierung von Allelen und erhalten die genetische Variation.

Zusammenfassend wird in dieser Arbeit untersucht, welche Faktoren für Entstehung von Herbizidresistenzen in mehrjährigen Unkräutern entscheidend sind. Sie schlägt zwei unterschiedliche Methoden vor, welche entworfen wurden, um verschiedene Aspekte dieses öko-evolutionären Prozesses zu studieren und Kommunikation über wissenschaftliche Disziplinen hinweg zu fördern.

Abstract

Since the dawn of agriculture, crop production has been challenged by pests – especially weeds, animal pests and pathogens – reducing crop survival and the amount and quality of harvestable produce. Weeds are the organisms causing the highest potential yield losses. Ever since, farmers have been searching for strategies to protect their crops, such as hand weeding and later mechanical control measures. The commercialisation of selective herbicides after the Second World War has revolutionised crop production, providing an easy and highly effective solution to weed management and enabling machine harvesting. Today, widespread resistance threatens the sustainability of herbicides and cropping systems. Herbicide resistance is a highly replicated example of rapid adaptation to human-mediated selection, extensively studied for more than 60 years, primarily in terms of mitigation practices. Yet, the precise determinants of the rate and nature of herbicide resistance evolution and how management practices alter the underlying eco-evolutionary processes remain poorly understood – especially in perennial species. In this thesis, I employ population dynamic models to identify critical factors defining the probability, timing, and source of resistance adaptation in a perennial weed and evaluate strategies to limit herbicide resistance evolution.

The approaches developed in the individual chapters are tailored to address specific aspects of herbicide resistance evolution and the management of seed- and rhizome-propagated perennials. In essence, I analyse how specific life history traits – like the mode of reproduction – and details of the resistance mechanism – like an associated fitness cost – affect the adaptation and assess the sustainability of management interventions. Capturing population-level dynamics in the active weed population and the dormant seed bank, the models allow me to study the twofold effect of a seed bank in herbicide resistance evolution. This work aims to provide mechanistic insights into the eco-evolutionary processes driving herbicide resistance adaptation, highlighting the value of weed population dynamic models for gaining a holistic understanding of these dynamics.

Chapter 2 focuses on the demography of a perennial weed population and how inherent weed characteristics and management govern its dynamics. In **Chapter 3**, I employ concepts of rescue theory to address questions concerning the source of adaptive variation and the probability and timing of treatment failure resulting from herbicide-resistant weeds.

I find that the integration of tillage with herbicide applications effectively reduces the risk of control failure by lowering weed densities and that mixtures of different herbicide classes are superior to rotations and mono-treatment in controlling the perennial weed and resistance evolution. Using a matrix approximation of the life cycle model, I derive estimates of the standing genetic variation for herbicide resistance modulated by the genetic details of the resistance mechanism, identifying the associated fitness cost as the primary determinant. Increasing the level of homozygosity, self-pollination accelerates target-site resistance evolution upon occurrence but diminishes adaptation from standing variants if resistance inheritance is (incompletely) dominant. The dominance of a resistance allele determines its exposure to selection; high dominance hastens the initial spread but delays fixation due to the masking of sensitive alleles in heterozygotes. Seed banks preserve genetic information of forgone selection, allowing fast responses to recurrent selection regimes, and prolong the time for adaptation by buffering changes in population size induced by weed management. Sheltering a substantial part of the population from the selective force, seed banks delay allele fixation and maintain genetic variation.

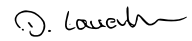
In summary, this thesis investigates pivotal factors determining herbicide resistance adaptation

in perennial weeds. It proposes two distinct approaches designed to study different aspects of the eco-evolutionary process and facilitate communication across scientific fields.

Declaration

I hereby declare that apart from my supervisor's guidance, this thesis is in content and form my own work. The thesis has not been submitted in part or entirety as part of an examination procedure before. Whether materials are published or submitted for publication is indicated in the thesis. This thesis has been prepared in compliance with the Principles of Good Scientific Practice of the German Research Foundation. I did not have any academic degree revoked.

Plön, 2024



Dana Lauenroth

Contents

1	Introduction	1
2	Persistence and adaptation in weeds with complex life cycles	9
	Introduction	9
	Model summary	10
	Results	14
	Discussion	21
	Methods	24
3	Weed control failure due to herbicide resistance evolution	29
	Introduction	29
	Methods	30
	Results	36
	Discussion	41
4	Summary and Outlook	45
	Bibliography	51
	Appendix	61
A	Appendix—Persistence and adaptation in weeds with complex life cycles	61
	Model parameters	61
	Matrix theory	66
	Supplementary figures	67
B	Appendix—Weed control failure due to herbicide resistance evolution	73
	Supplementary figures	73
	Supplementary methods	75
	Parameterisation	80

CHAPTER 1

Introduction

Pests – mainly weeds, animal pests, and pathogens – have challenged crop production since agriculture’s origins nearly 10,000 years ago. These harmful organisms reduce crop survival and the amount and quality of harvestable produce, leading to economic losses for the farmers and potentially threatening global food security. Farmers have always been searching for strategies to protect their crops. First, they relied solely on manual and mechanical measures for controlling weed populations and animal pests, while diseases caused by pathogens were essentially unidentifiable, with no effective treatments available (OERKE, 2006). The introduction of synthetic crop protection chemicals, specifically herbicides, insecticides and fungicides, has vastly improved pest control and enabled intense farming (CARVALHO, 2017; OERKE, 2006; TUDI et al., 2021). In combination with other factors, like high-yielding crop varieties and the use of fertilisers, chemical pest control has contributed to more than doubling food production during the last century (CARVALHO, 2017; OERKE, 2006). The global population growth, involving an increase in food demand – predicted to range between 35 and 56 % from 2010 to 2050 (DIJK et al., 2021) – necessitates further enhancement of crop production. However, the overreliance of modern agriculture on pesticides has caused the evolution of resistance in various pest species globally (HAWKINS et al., 2019; HEAP, 2024). The potential loss of pesticide efficacy imposes a significant challenge to agriculture and threatens food security (HAWKINS et al., 2019).

Weeds are the organisms causing the highest potential yield losses in crop production systems, followed by animal pests and pathogens (OERKE, 2006). While inorganic copper salts were used as the first chemicals for weed control as early as the beginning of the 20th century, it was not until the Second World War that the first selective herbicide was synthesised (HAMILL et al., 2004; OERKE, 2006). Subsequently, numerous herbicides targeting specific plant proteins – involved in vital processes like photosynthesis, cell growth and division, and cellular metabolism – have been developed (DUKE, 2012; QU et al., 2021). Simplifying weed control tremendously and enabling machine harvesting of crops, target-based herbicides have replaced labour-intensive mechanical weed control and become the primary – often exclusive – tool for weed management within less than 30 years (PEROTTI et al., 2020; POWLES et al., 2010). This development towards cropping systems relying heavily on herbicides was even enhanced with the introduction of transgenic herbicide-resistant crops in the 1990s (DUKE, 2012; OERKE, 2006; QU et al., 2021). Herbicides are marketed together with resistant crops, providing a simple weed management system with high crop safety (POWLES et al., 2010; QU et al., 2021).

Although their commercialisation has vastly improved weed management and considerably contributed to the increases in food productivity, herbicides have not resulted in a reduction of the weed burden (OERKE, 2006; POWLES et al., 2010). Natural selection acting on genetic diversity enables biological organisms to adapt to unfavourable conditions and facilitates their persistence under changing circumstances (ORR, 2005). While environmental conditions often change gradually over long periods, catastrophic events might result in abrupt alterations. Human interventions are characterised by sudden and drastic shifts in the environmental conditions of affected species (PALUMBI, 2001). Frequent herbicide application in agricultural fields – removing up to 99 % of sensitive weeds – exerts strong selection on weed populations, forcing the weeds to adapt rapidly,

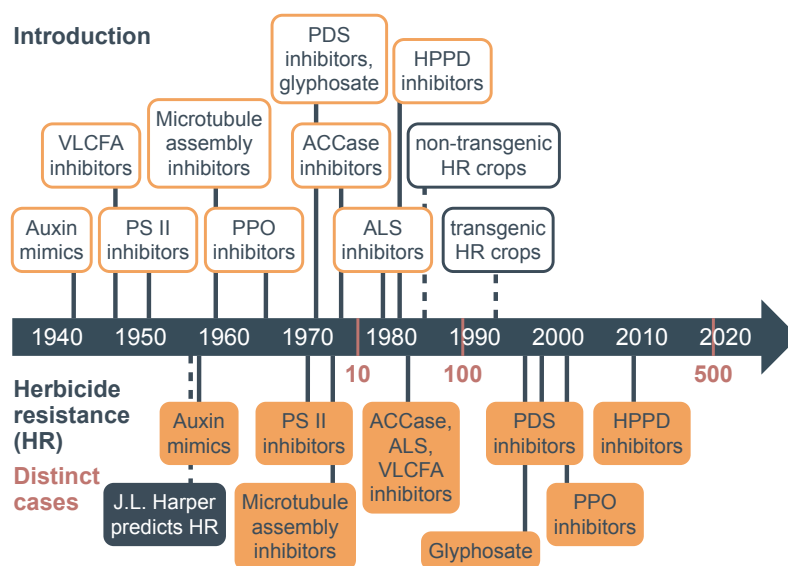


Figure 1.1 | Timeline showing the introduction and first case of resistance for the most important herbicide groups. The thresholds of 10, 100 and 500 distinct cases of herbicide resistance (by herbicide group and weed species) are marked on the timeline in red. Additionally, the first prediction of herbicide resistance and the introduction of non-transgenic and transgenic herbicide-resistant crops (HR crops) are depicted. The data was taken from BAUCOM (2019), HEAP (2024), and REDDY et al. (2012).

racing against extinction (BAUCOM, 2016; DÉLYE, 2013; JOHNSON et al., 2014; KREINER et al., 2017). The ecologist J.L. Harper was the first one to predict the evolution of resistance in response to the application of herbicides in 1956, verified just one year later by case reports of herbicide resistance in two weed species (BAUCOM, 2019; HEAP, 2024; PEROTTI et al., 2020). Since this first incidence, herbicide resistance has evolved multiple times in a variety of plants. Currently, 272 weed species are known to exhibit resistance against at least one of 168 affected herbicides (HEAP, 2024), and these numbers continue to increase steadily. Over the last decades, barely any new herbicides with novel sites of action have been released, primarily due to several economic reasons and high regulatory safety standards (DUKE, 2012; QU et al., 2021; SELBY et al., 2023). The overdue supply of novel herbicides aggravates the threat ever-increasing cases of herbicide resistance pose to the sustainability of chemical weed management. The history of herbicides is illustrated briefly in Figure 1.1.

Herbicide-resistance genes are able to migrate between weed populations via pollen, seed or vegetative propagule dispersal, facilitating a fast spread and rapid increase in the frequency of herbicide resistance traits among weed populations once they arise (BECKIE et al., 2019; MAITY et al., 2022). Owing to the herbicide resistance gene flow, herbicide sensitivity is a common resource shared among farmers and societies that we need to preserve in a collective attempt. Within the last 60 years, researchers have investigated herbicide resistance at the phenotypic, physiological, and genetic levels to ascertain the extent of resistance and mitigate its spread and further resistance evolution (BAUCOM, 2019). The integration of mathematical models in studies of population dynamics in arable weeds was initiated in the 1970s with the seminal work of SAGER et al. (1976), building the framework for many subsequent models to this day (HOLST et al., 2007). Mathematical and computer simulation modelling are nowadays essential tools in weed science and management for predicting long-term responses of weed populations to different management regimes – more

general, changes in their environment – and identifying key factors affecting evolutionary and population dynamics of herbicide resistance (BAGAVATHIANNAN et al., 2020; FRECKLETON et al., 2009; HOLST et al., 2007; RENTON et al., 2014).

Where field and greenhouse experiments are limited in time, space and management scenarios that can be explored, mathematical models allow the *in silico* study of long-term weed population dynamics under various management scenarios (BAGAVATHIANNAN et al., 2020; HOLST et al., 2007; RENTON et al., 2014). This is particularly helpful for first predictions of the outcome of novel weed management strategies yet to be implemented, where a high number of different options is to be compared or in cases where field studies would produce a substantial quantity of resistant weeds (FRECKLETON et al., 2009; RENTON et al., 2014). However, even if the management options can be readily tested in the field, the ecological and evolutionary processes proceed over several years, involving very high numbers of individuals and potentially spanning large areas. Long-term and large-scale evolutionary and population dynamics in weeds are impractical to be fully captured in an experimental setup, necessitating a model description that provides a fast and safe framework for their investigation (BAGAVATHIANNAN et al., 2020; HOLST et al., 2007; RENTON et al., 2014). Further, modelling advances our understanding of the ecological, evolutionary and anthropogenic drivers of herbicide resistance adaption and their interplay (BAGAVATHIANNAN et al., 2020; RENTON et al., 2014). Mathematical models allow the manipulation of individual factors and interactions, providing insights into specific life history processes – including their effect on resistance evolution – and revealing causal relationships. The seeds of most weed species get preserved in a persistent seed bank, enabling populations to survive periods of unfavourable conditions – like the presence of herbicides (MAQSOOD et al., 2020). This dormancy causes a considerable time lag in the response of such weed populations to alterations in the environment and management regime, impeding the assessment of weed dynamics by field experiments (STORKEY et al., 2022). Mathematical models can capture this usually hidden aspect of the weed life cycle. Capturing the change in seed bank composition over time, models thus yield information about how management strategies affect the weed seed bank and how the seed bank interacts with ecological factors to impact population dynamics and resistance evolution (STORKEY et al., 2022).

Compared to the emergence of resistance in cancer therapy and bacterial resistance to antibiotics, the evolution of herbicide resistance received little attention among theoretical biologists and, more generally, in broader ecology and evolution (BAUCOM, 2019). Mathematical and simulation modelling approaches dealing with dynamics of herbicide resistance adaption are typically found within the weed science literature (BAUCOM, 2019; HOLST et al., 2007). Nevertheless, as weed science benefits from ecological and evolutionary models and insights, the fields of ecology and evolution can profit from the discoveries made in herbicide resistance research (BAUCOM, 2019; KREINER et al., 2017). The largely replicated evolution of herbicide resistance in weeds presents an excellent model for studying phenomena at the heart of evolutionary biology, like rapid adaption to anthropogenic selection regimes – characterised by extreme selective pressure – and phenotypic convergence – the independent evolution of one trait in several distinct evolutionary lineages (BAUCOM, 2016; BAUCOM, 2019; KREINER et al., 2017).

In this thesis, I employ population dynamical models to study the factors determining the probability and timing of herbicide resistance evolution in a perennial weed species and strategies to mitigate or delay its occurrence. In the following, I will briefly describe the concepts and methods used in this thesis and outline the structure.

Complex life cycle. The life history of an organism plays an integral role in shaping evolutionary and population dynamics. Dormant life history stages like weed seed banks are expected to slow evolution by shielding a substantial proportion of the population from the impact of the selective force (HAIRSTON et al., 1988; LENNON et al., 2021). Under unfavourable or fluctuating environmental conditions, seed banking – a bet-hedging strategy – decreases the probability of population extinction and prolongs the time until extinction (EVANS et al., 2007; KALISZ et al., 1993; LENNON et al., 2021). Theoretical results suggest that asexual reproduction generally slows the evolution towards an optimum but potentially leads to higher survival after a sudden environmental change (ORIVE et al., 2017).

Past modelling studies of herbicide resistance have mainly dealt with annual weeds (HOLST et al., 2007, but see LIU et al., 2019; HOLMES et al., 2022) while a substantial fraction of the most problematic arable weed species is rhizomatous perennials (HOLM et al., 1977). The work in this thesis focuses on the dynamics of herbicide resistance evolution in seed- and rhizome-propagated weeds with a persistent seed bank. Being one of the world's most destructive and economically important perennial weed species, *Sorghum halepense* (Johnsongrass) and its management in an agricultural setting serves us as a model system (KLEIN et al., 2021). In [Chapter 2](#), I present a population-based model of Johnsongrass comprising the whole perennial life cycle. Capturing the genetics of herbicide resistance, the model allows me to forecast the weed population dynamics and evolution of herbicide resistance as governed by different management strategies and influenced by specific life history traits and genetic details of the resistance mechanism.

Sorghum halepense (Johnsongrass). Johnsongrass is a highly competitive and invasive perennial, capable of sexual reproduction via seeds and asexual propagation through rhizomes (KLEIN et al., 2021; PEERZADA et al., 2023; SCHWINNING et al., 2017). The grassy weed – classified as such in 53 countries – out-competes native grasses and causes significant yield losses in essential food and fodder crops (KLEIN et al., 2021; SCHWINNING et al., 2017). During winter, Johnsongrass enters dormancy, surviving as seeds and rhizomes underground (WARWICK et al., 1983). In spring, seeds germinate into seedlings and shoots sprout from buds on the rhizomes (PEERZADA et al., 2023). Maturing over the season, the plants compete for resources to survive and to produce new rhizomes and seeds (SCHWINNING et al., 2017; WARWICK et al., 1983; WILLIAMS et al., 1977). In the monoecious species, sexual reproduction is ensured even at very low densities by a high level of self-pollination (WARWICK et al., 1983). While rhizomes only give rise to shoots in the first spring after their production, the seeds of Johnsongrass are able to stay viable in a dormant state for several years, forming a seed bank in the soil (PEERZADA et al., 2023; WARWICK et al., 1986). I illustrate this cyclical life pattern of Johnsongrass and its life stages in [Figure 1.2 a and b](#).

In cropped areas, Johnsongrass is typically controlled by post-emergence herbicide application (KLEIN et al., 2021; PEERZADA et al., 2023). Acetyl-coenzyme-A carboxylase (ACCase)-inhibitors target the biosynthesis of fatty acid (DÉLYE et al., 2013b; PEERZADA et al., 2023). Glyphosate and acetolactate synthase (ALS)-inhibitors both target amino acid biosynthesis (DÉLYE et al., 2013b; KLEIN et al., 2021). The continuous use of such herbicides has caused Johnsongrass to develop resistance mechanisms against herbicides of all the three classes in use (LORENTZ, 2014; PEERZADA et al., 2023; VILA-AIUB et al., 2007).

Herbicide resistance. On the molecular level, herbicide resistance can be categorised into two mechanisms – target-site resistance and non-target-site resistance. Target-site resistance is characterised by alterations of the target site limiting the effect of a herbicide, even though it is reaching the target site at a normally lethal dose (PEROTTI et al., 2020; POWLES et al., 2010). Common are amino-acid substitutions in the targeted protein conferring changes in the 3-dimensional structure that prevent conformational binding of the inhibiting herbicide; less often, target-site resistance results from

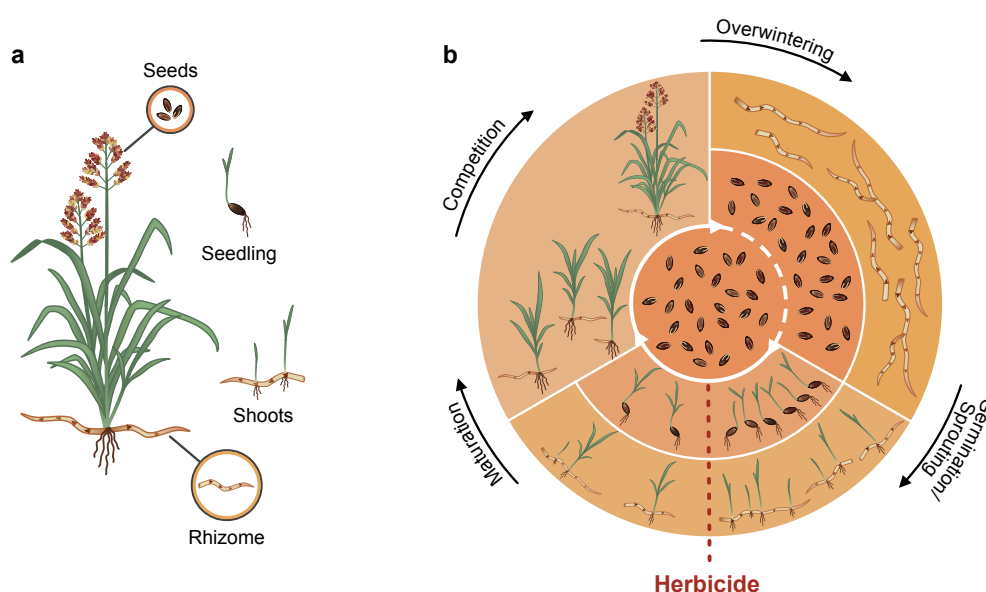


Figure 1.2 | Schematic illustration of Johnsongrass' life history stages and life cycle.

a, Life history stages of Johnsongrass. Shown is an adult weed plant, which produced fresh seeds and a rhizome, next to a seedling and two shoots emerging from a rhizome. **b**, Life cycle of Johnsongrass. Johnsongrass reproduces sexually via seeds (inner ring) and asexually through rhizomes (outer ring). Seeds can stay dormant in the ground for several years, forming a seed bank (central circle). New seeds and seeds from the seed bank might germinate in spring or stay dormant as part of the seed bank (expressed by the dashed line). Rhizomes give rise to shoots in the first spring after their production. Herbicide application (red dashed line) can kill susceptible seedlings and shoots. The plants that survive then compete for resources as they mature. The aboveground plant material dies in winter, and Johnsongrass overwinters as seeds and rhizomes in the ground.

the overexpression of the target proteins (DÉLYE et al., 2013b; POWLES et al., 2010; QU et al., 2021). Target-site mutations confer resistance to herbicides targeting the protein concerned; the efficacy of herbicides with a different mode of action is not impacted (DÉLYE, 2013; QU et al., 2021). The inheritance is monogenic, with most cases of target-site resistance conferred by dominant or semi-dominant alleles (DÉLYE et al., 2013b). Non-target-site resistance, on the contrary, is conferred by mechanisms that minimise the exposure of target proteins to active herbicidal ingredients – including the overexpression of transporter proteins, an increase in metabolism or a reduction in herbicide penetration or translocation (POWLES et al., 2010; QU et al., 2021). Cross-resistance is not restricted to herbicides sharing the same mode of action and can even include herbicides yet to be introduced (DÉLYE et al., 2013b). While rare cases of monogenic inheritance exist, non-target-site resistance is typically a complex polygenic trait (DÉLYE et al., 2013b). The reported cases of herbicide resistance in Johnsongrass biotypes include target-site resistance towards various ACCase- and ALS-inhibitors (LORENTZ, 2014; PEERZADA et al., 2023) and non-target-site resistance to glyphosate (VILA-AIUB et al., 2007). In this thesis, I cover target-site resistance in Johnsongrass.

Resistance mechanisms confer a fitness advantage in the presence of herbicide, but weeds might suffer adverse effects on fitness in its absence (PURRINGTON, 2000). These resistance costs control the rate of resistance evolution under heterogeneous selection regimes as constituted by herbicide rotations, catalyse the loss of resistance alleles in the absence of the herbicide and moderate the role of preexisting variation (NEVE et al., 2014; VIGUEIRA et al., 2013). However, while some resistance-endowing mutations are associated with deleterious pleiotropic effects resulting in fitness

costs for the plant, others have no detectable effect on fitness or are even beneficial in the absence of herbicides (DÉLYE et al., 2013b; VILA-AIUB et al., 2009). Fitness costs related to target-site alterations are a consequence of impaired enzyme functionality, whereas altered metabolic pathways might cause a shift in resource allocation from plant growth and reproduction to defence (POWLES et al., 2010; VILA-AIUB et al., 2009). In Johnsongrass, a target-site mutation conferring resistance to ACCase-inhibitors has been linked to a fitness cost on seed production (PANOZZO et al., 2021); I study its impact on the dynamics of herbicide resistance evolution and the primary source of adaptive variation.

Evolutionary rescue and source of adaptive variation. Populations confronted with a drastic environmental change that causes maladaptation and population decline might escape extinction through evolutionary adaptation occurring on the same time scale (ALEXANDER et al., 2014; BELL, 2017; CARLSON et al., 2014). This phenomenon – termed evolutionary rescue – has been studied in the past 30 years mainly in the context of conservation biology and medicine (ALEXANDER et al., 2014; BELL, 2017; GOMULKIEWICZ et al., 1995). The rapid adaptation of arable weeds to the strong selection induced by herbicide application constitutes an example of evolutionary rescue (BELL, 2017), which has – despite calls from the weed research community – not previously been treated in the rescue literature (KREINER et al., 2017; NEVE et al., 2014). Weeds exhibit an astonishing variation and complexity in their reproductive system. A few studies investigated in a general context how the mode of reproduction – outcrossing versus selfing and sexual versus asexual reproduction – influences fast adaptation (GLÉMIN et al., 2013; ORIVE et al., 2017; UECKER, 2017). Yet another critical factor shaping eco-evolutionary dynamics in weed populations is their persistent seed bank, providing “migration from the past” (YAMAMICHI et al., 2019). To the best of my knowledge, a comprehensive analysis of how the seed bank and the mode of reproduction interact to shape the dynamics of herbicide resistance evolution is missing in the literature (but see NEVE, 2008 for a simulation study considering selfing and varying seed germination rates under constant seed bank mortality).

The probability and rate of a population successfully adapting to environmental stress and avoiding extinction depend on the origin of the adaptive type – which might have been already present in the population as part of the standing genetic variation or might arise subsequently by *de novo* mutations (BELL, 2017; KREINER et al., 2017). Adaptation is typically faster from standing variants available in the population at higher copy numbers with the onset of stress (BARRETT et al., 2008). Whether evolutionary rescue is more likely to result from preexisting variants or novel mutations is determined by their absolute frequencies and can affect the suitability of measures aiming to mitigate undesired adaptation, like the evolution of herbicide and antibiotic resistance (BELL, 2017; KREINER et al., 2017). There are theoretical arguments suggesting that the selection of mechanisms conferring high levels of resistance – like target-site mutations – proceeds mainly from novel mutation (HAWKINS et al., 2019; NEVE et al., 2014). While genomic data of several waterhemp (*Amaranthus tuberculatus*) populations support a prevailing role of *de novo* mutations in this species (KREINER et al., 2022), genomic analysis and simulations identified a prevalent role of standing genetic variation in target-site resistance adaptation of the annual weed blackgrass (*Alopecurus myosuroides*) (KERSTEN et al., 2023). The precise determinants of the primary source of adaptive variation in herbicide resistance remain poorly understood. In [Chapter 3](#), I present a branching process model of rapid adaptation in response to strong selection, capturing complex life cycles of sexual and asexual reproduction and dormancy in a perennial weed. Using rescue and branching process theory, I study the probability of herbicide resistance adaptation and the waiting time distribution until the first appearance of resistant plants as determined by seed bank dynamics and details of the reproductive system. I analyse key factors – including the cost of resistance and mode of pollination – determining the primary source of adaptive variation.

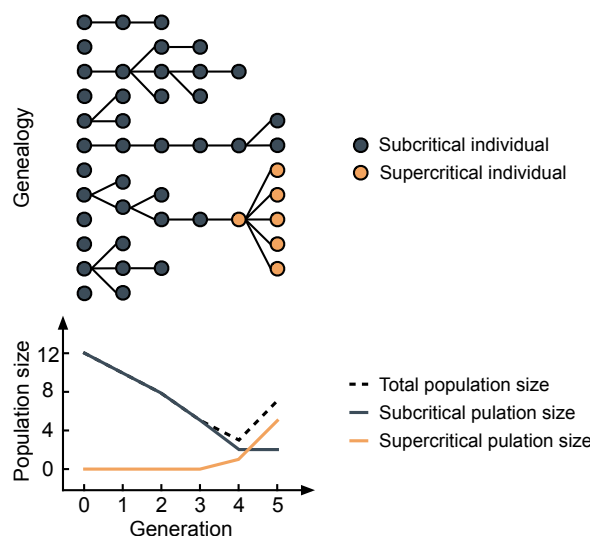


Figure 1.3 | Realisation of a multitype Galton-Watson process with subcritical and supercritical types. The genealogy of the process is illustrated, and below is the development of the population size shown – the total population size (dashed line) and the size of the subpopulations (solid lines). A grey individual’s expected number of offspring is lower than one; we denote this type by subcritical type. Orange individuals have, on average, more than one offspring, enabling positive population growth; we term this a supercritical type. A population of subcritical individuals (grey) is doomed to extinction unless a type with higher fitness (orange) appears.

Branching processes. Branching processes are well-established in biological studies for modelling population dynamics, often concerned with the probability of ultimate extinction (ATHREYA et al., 1972; HARRIS, 1963; KIMMEL et al., 2002; SANTER et al., 2020). They have been used to model populations at risk of extinction due to low reproduction rates after an environmental change, investigating how the emergence of an adapted mutant – arising *de novo* or as part of the preexisting variation – could prevent population extinction via evolutionary rescue (AZEVEDO et al., 2021; ORR et al., 2008; UECKER et al., 2014). The declining maladapted population is often treated deterministically while a single-type branching process captures the stochasticity in the number of adapted mutants (ORR et al., 2008; UECKER et al., 2016; UECKER et al., 2014). However, multitype branching processes, jointly describing the dynamics of maladapted and adapted types, can yield further information about the waiting time until such an adapted mutant appears (ALEXANDER, 2016; AZEVEDO et al., 2021; SERRA et al., 2007). In [Chapter 3](#), I use a multitype discrete-time branching process model, precisely a multitype Galton-Watson process model, to study the probability and timing of herbicide resistance adaptation from novel mutations and standing variation in Johnsongrass. Homogeneous subpopulations that produce on average less than one offspring – termed subcritical types and said to have negative fitness – are doomed to extinction, while lineages of types yielding an expected number of offspring greater than one – termed supercritical types and said to have positive fitness – have a positive survival probability. Upon appearance, an adapted type with positive fitness potentially restores positive growth in a population declining in response to an environmental change, as illustrated in [Figure 1.3](#).

This thesis aims to show how modelling the complex system of agricultural weed management facilitates a more complete understanding of underlying mechanisms and exposes open questions in ecology and evolution. Mathematical and computer simulation modelling provide a means to inform the development of management strategies with an understanding of the ecologic and evolutionary processes driving the evolution of herbicide resistance in weed populations. Herbicide-resistant weeds, at the same time, constitute uniquely suited models for studying rapid adaptation to human-mediated regimes of strong selection.

CHAPTER 2

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

This chapter is published in a peer-reviewed journal. Lauenroth, D., and Gokhale, C.S. *Theoretical assessment of persistence and adaptation in weeds with complex life cycles*. Nature Plants 9, 1267–1279 (2023). DOI: [10.1038/s41477-023-01482-1](https://doi.org/10.1038/s41477-023-01482-1).

Abstract

Herbicide-resistant weeds pose a significant threat to global food security. Perennial weed species are particularly troublesome. Such perennials as *Sorghum halepense* spread quickly and are difficult to manage due to their ability to reproduce sexually via seeds and asexually through rhizomes. Our theoretical study of *Sorghum halepense* incorporates this complex life cycle with control measures of herbicide application and tillage. Rooted in the biology and experimental data of *Sorghum halepense*, our population-based model predicts population dynamics and target-site resistance evolution in this perennial weed. We found that the resistance cost determines the standing genetic variation for herbicide resistance. The sexual phase of the life cycle, including self-pollination and seed bank dynamics, contributes substantially to the persistence and rapid adaptation of *Sorghum halepense*. While self-pollination accelerates target-site resistance evolution, seed banks significantly increase the probability of escape from control strategies and maintain genetic variation. Combining tillage and herbicide application effectively reduces weed densities and the risk of control failure without delaying resistance adaptation. We also show how mixtures of different herbicide classes are superior to rotations and mono-treatment in controlling perennial weeds and resistance evolution. Thus, by integrating experimental data and agronomic views, our theoretical study synergistically contributes to understanding and tackling the global threat to food security from resistant weeds.

Introduction

Sorghum halepense (Johnsongrass) is infamous for being “a threat to native grasslands and agriculture” (KLEIN et al., 2021, p. 413). Johnsongrass is classified as a weed in 53 countries (KLEIN et al., 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 et al., 2021). The grassy weed out-competes native grasses like big bluestem (*Andropogon gerardii*), little bluestem (*Schizachyrium scoparius*) and switchgrass (*Panicum virgatum*) in grasslands (SCHWINNING et al., 2017). Johnsongrass is a highly competitive and invasive perennial, capable of sexual reproduction via seeds and asexual propagation through rhizomes (PEERZADA et al., 2023; SCHWINNING et al., 2017; WARWICK et al., 1983). In addition to resource competition, Johnsongrass affects crop production by serving as an alternative host for various agricultural pests (KLEIN et al., 2021; PEERZADA et al., 2023).

In cropped areas, typical control of Johnsongrass is by a post-emergence application of herbicides, particularly glyphosate, acetyl-coenzyme-A carboxylase (ACCCase)- and acetolactate synthase (ALS)-inhibitors (KLEIN et al., 2021; PEERZADA et al., 2023). 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., 2023) and non-target-site resistance to glyphosate (VILA-AIUB et al., 2007). Herbicide resistance imposes a considerable challenge on weed management. There is an urgent need for weed management strategies that reduce herbicide resistance risk while effectively controlling the weed population. Integrated approaches, combining herbicide application with mechanical measures, such as tillage, and potentially crop rotations, are reported to provide efficient control of Johnsongrass, including its rhizomes (PEERZADA et al., 2023; WARWICK et al., 1983). Mixtures and rotations of herbicides with different sites of action reportedly reduce weed densities more efficiently than recurrent use of one herbicide class and delay resistance evolution (BUSI et al., 2020; DIGGLE et al., 2003; ZELLER et al., 2021).

To develop and select sustainable control strategies, we must improve our understanding of eco-evolutionary processes underlying the population dynamics and how inherent plant characteristics shape the evolution of herbicide resistance in perennial weeds. Mathematical modelling has proven valuable in studying long-term population dynamics and herbicide resistance evolution in weeds (FRECKLETON et al., 2009; HOLST et al., 2007). However, the majority of existing models deal with annual weeds; the complex life cycle of perennial species has been addressed to a lesser extent (HOLST et al., 2007, but see LIU et al., 2019; HOLMES et al., 2022). To our knowledge, only two population dynamical models exist for Johnsongrass, and more general for perennials comprising the whole life cycle (HOLMES et al., 2022; LIU et al., 2019). LIU et al. (2019) presented an individual-based model, which captures self-pollination but lacks a seed bank. The stage-structured model of HOLMES et al. (2022) comprises both a seed bank and a bank of rhizome buds. However, the model lacks the possibility of *de novo* resistance evolution, and pure cross-pollination is assumed.

The goals of this study are (i) to explore how the resistance cost and its dominance affect the standing genetic variation for target-site resistance; (ii) to study how genetic details, like the dominance of resistance and the associated fitness cost, impact population dynamics and target-site resistance evolution in herbicide-treated weeds; (iii) to examine the effects of self-pollination and seed bank formation on genetic diversity, target-site resistance evolution, and risk of control failure in perennial species; (iv) to determine the potential of tillage, herbicide mixtures and 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 Johnsongrass, it can capture the population dynamics and herbicide resistance adaptation of general perennial weeds. More generally, our population-based approach can be adapted to any species that comprise sexual reproduction in the continuum of selfing and outcrossing, asexual propagation, and potentially a seed bank component.

Model summary

The seminal work by SAGER et al. (1976) has inspired several models of weed life cycles. The population dynamic is typically captured deterministically using several discrete-time equations, each corresponding to 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 model describing the dynamics of seed- and rhizome-propagated Johnsongrass. Our deterministic model with an annual time step comprises the whole perennial life cycle. We then use the model to forecast population dynamics and the evolution of target-site resistance. In this section, we summarise the main features of our theoretical model: life history stages and eco-biology, control measures and resistance, and stochastic simulations. For a detailed exposition, we refer to the Methods. We inferred all model parameters carefully from field trials in the literature. In the Supplementary Information, we provide [Table B.1](#) with all parameter values and the derivation.

Life cycle

Johnsongrass is dormant throughout the winter, overwintering as seeds or rhizomes in the ground (WARWICK et al., 1983). At the beginning of a growing season, seeds germinate to produce seedlings, and shoots emerge from nodes on the rhizomes (PEERZADA et al., 2023). Mature plants produce new rhizomes and, after flowering, viable seeds (PEERZADA et al., 2023; WARWICK et al., 1983).

Figure 2.1 outlines the life cycle of Johnsongrass with both reproduction pathways, sexual via seeds and asexual through rhizomes (a) and its representation in our model (b). The figure further illustrates the implementation of self-thinning and density-dependent reproduction resulting from intraspecific competition (c). Only rhizomes and seeds are present in early spring. 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). The number of shoots in any given season can be determined from the number of rhizomes, the number of nodes on rhizomes and the proportion of nodes producing shoots.

Freshly produced Johnsongrass seeds are highly dormant throughout the season (WARWICK et al., 1983). They germinate in the subsequent seasons and can stay viable in the ground for several years (PEERZADA et al., 2023; WARWICK et al., 1983). New seeds enter the seed bank but are subject to post-dispersal predation and might lose viability or decay until the beginning of the next season (BAGAVATHIANNAN et al., 2013). Each spring, only a certain proportion of seeds in the seed bank germinates to produce seedlings (LEGUIZAMÓN, 1986). Non-germinated seeds may either be lost due to decay and viability loss or stay dormant (BAGAVATHIANNAN et al., 2013). At all times, the seed bank consists of non-germinated seeds from the previous seed bank and seeds produced in the preceding season.

Seedlings and shoots grow into 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 originating from seeds and rhizomes (HOROWITZ, 1972; MCWHORTER, 1961). Hence, we do not distinguish the adult plants by their origin. The cycle continues as the adult plants produce new seeds and rhizomes, of which only a certain amount survive the winter.

Intraspecific competition

Johnsongrass is more strongly affected by intraspecific competition than by competition from other perennial grasses, like big bluestem, little bluestem and switchgrass (SCHWINNING et al., 2017). In addition, since the crop density is assumed to remain constant throughout the seasons, we do not explicitly model competition from the crop and other weeds. We implement the hyperbolic self-thinning function introduced by YODA et al. (1963) to model early plant survival (illustrated in Figure 2.1 c).

Moreover, WILLIAMS et al. (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. Also, the final dry weight of reproductive structures was reduced in crowded stands. Due to a 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 et al., 1985) (the density-dependent fertility is illustrated in Figure 2.1 c).

Control and resistance

Numerous control strategies exist for managing weed populations. They are typically classified according to their primary mode of action: chemical, mechanical, biological, cultural or allelopathic (PEERZADA et al., 2023). We focus on tillage (mechanical control) and post-emergence herbicide application (chemical control).

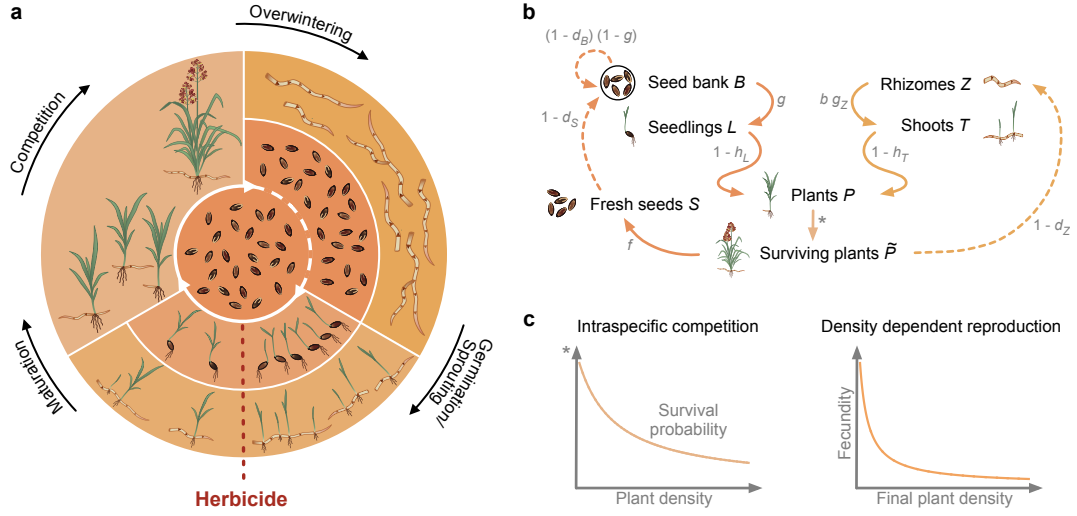


Figure 2.1 | Schematic illustration of Johnsongrass' life cycle and its representation in our model. **a**, Diagram of Johnsongrass' life cycle. Johnsongrass reproduces sexually via seeds (inner ring) and asexually through rhizomes (outer ring). Seeds can stay dormant in the ground for several years, forming a seed bank (central circle). New seeds and seeds from the seed bank might germinate in spring or stay dormant as part of the seed bank (expressed by the dashed line). Rhizomes give rise to shoots in the first spring after their production. Herbicide application (red dashed line) can kill susceptible seedlings and shoots. The plants that survive then compete for resources as they mature. The aboveground plant material dies in winter, and Johnsongrass overwinters as seeds and rhizomes in the ground. **b**, Schematic representation of our model. The left part of (b) corresponds to the sexual reproduction of Johnsongrass, and the right part represents the asexual propagation. Solid arrows depict within-season dynamics, and dashed arrows show dynamics between seasons. Survival probabilities and fecundity are shown in grey next to the corresponding arrows. **c**, Intraspecific competition. Intraspecific resource competition leads to self-thinning and density-dependent fecundity reduction. The left panel displays the probability of intraspecific competition survival in young plants (P) as a function of their density. The density-dependent reduction in fecundity, i.e. the number of seeds (f) and rhizome buds (b) produced by mature plants, is illustrated on the right.

As for target-site resistance, e.g. against ACCase- or ALS-inhibitors, we consider a single allele R associated with herbicide resistance (HERNANDEZ et al., 2015; KREINER et al., 2017; SCARABEL et al., 2014). We assume a diploid genome, but the model can be extended to reflect higher ploidy levels (see HOLMES et al. (2022) for a comparison of the rate of herbicide resistance evolution in diploid and tetraploid weeds). 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 we can account for recessivity ($k_h = 0$), partial dominance ($0 < k_h < 1$) and complete dominance ($k_h = 1$) of the resistance trait. Spontaneous mutations to the resistance allele R can occur during sexual reproduction. We do not consider back mutations as they do not change the dynamics relevantly. Johnsongrass is primarily self-pollinated, leading to higher homozygosity and reduced genetic diversity in the produced seeds compared to cross-fertilization (WARWICK et al., 1983). The sexual reproduction in our model follows Mendelian inheritance and includes spontaneous mutations to the resistance allele R occurring with probability μ (see Supplementary Information).

Standing genetic variation. Indeed, while mutations can emerge due to the selection pressure of the herbicides, resistance may already exist in the natural variation in a population. Standing genetic variation for herbicide resistance has been found in plant populations never exposed to herbicides, among others, target-site resistance against ACCase-inhibitors (DÉLYE et al., 2013a). Moreover, herbicide resistance adaptation may primarily proceed from standing genetic variants as the initial frequency of resistant individuals may be much higher than the spontaneous mutation rate of resistance alleles (DÉLYE et al., 2013a; KREINER et al., 2017). Thus we also include standing genetic variation in our model and derive an analytic approximation of the expected variation (\mathbf{v}) in an untreated field.

Resistance cost. 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 et al., 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 RW plants, a reduction in the average number of seeds produced by c and $k_c c$, respectively, is considered compared to the fecundity of susceptible plants. The resistance cost is always relevant, irrespective of the control regime.

Herbicide efficacy. In our model, herbicide resistance is complete for homozygous-resistant plants. Herbicide application can kill susceptible plants and partly resistant heterozygotes with an efficacy h and $(1 - k_h)h$, respectively. Due to inadequate translocation of herbicides into the rhizomes and dormant buds, their control is only partial (BEASLEY, 1970; TUESCA et al., 1999). Thus even when the herbicide kills shoots, new shoots may eventually resprout from the parent rhizome (BEASLEY, 1970; TUESCA et al., 1999). This resprouting reduces the herbicide efficacy on rhizome shoots compared to seedlings (VIDRINE, 1989). Johnsongrass rhizomes are cut into pieces during tillage and partly turned to the soil surface (WARWICK et al., 1983). The fragmented rhizomes in shallow layers are exposed to low temperatures during winter, leading to increased mortality (HULL, 1970; WARWICK et al., 1983). Additionally, rhizome fragmentation can maximize bud sprouting (MCWHORTER et al., 1965), allowing for enhanced control of Johnsongrass plants originating from rhizomes by the herbicide (HULL, 1970; TUESCA et al., 1999).

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., 2023). Due to the different sites of action, target-site resistance against herbicides of one class is uncorrelated with resistance against the other. However, in annual ryegrass (*Lolium rigidum*), multiple and cross-resistance to ACCase- and ALS-inhibitors has been reported (TORRA et al., 2021) due to a combination of target-site and non-target-site resistance (TORRA et al., 2021). Since we do not consider non-target-site resistance, we omit cross-resistance. The combined efficacy of the two herbicides applied as a mixture is calculated as $h_{mix} = 1 - (1 - h_1)(1 - h_2) = h_1 + h_2 - h_1 h_2$, where h_1 and h_2 are the efficacies of the herbicides in mono-treatment (BLISS, 1939). Likewise, suppose the effects of the fitness costs associated with the respective target-site resistance are independent. Then the fitness cost of a plant fully resistant against both herbicides is $c_{\text{double resistant}} = 1 - (1 - c_1)(1 - c_2) = c_1 + c_2 - c_1 c_2$. Here c_1 and c_2 are the fitness costs of resistance towards the individual herbicides.

Stochastic simulations

Deterministic models capture selection as an evolutionary force, particularly for large populations. However, for small populations, strong selection may lead to the extinction of non-favoured types. Moreover, genetic drift acquires more relevance in small populations. We implemented stochastic simulations of our population-based model to account for such natural stochasticity. This approach

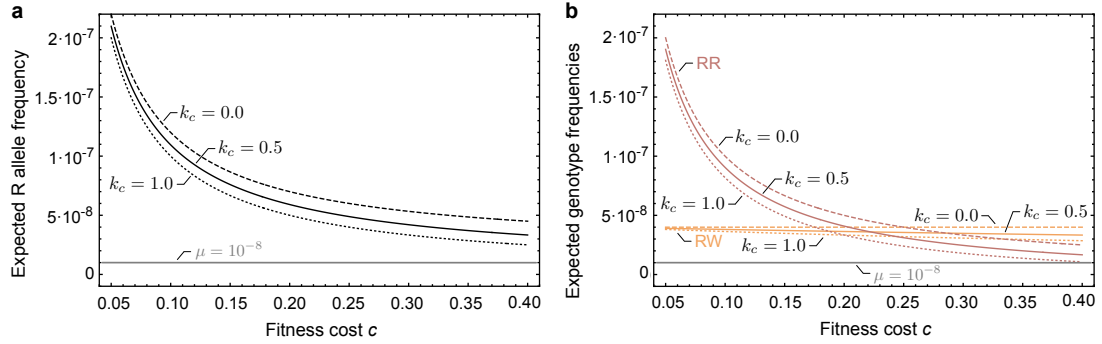


Figure 2.2 | Variation of the approximated standing genetic variation for target-site resistance with the resistance cost. The genetic composition of untreated Johnsongrass populations is approximated on the basis of Eq. (2.22) and shown for different degrees of dominance (k_c) of the resistance cost. Dashed lines correspond to a recessive resistance cost ($k_c = 0$), solid lines indicate partial dominance ($k_c = 0.5$) and dotted lines complete dominance ($k_c = 1$). The grey line marks the spontaneous mutation rate of resistance alleles ($\mu = 10^{-8}$). **a**, Expected frequency of the resistance allele R in an untreated population depending on the resistance cost (c). **b**, Expected frequencies of the resistant genotypes in an untreated population as a function of the resistance cost (c). The frequency of resistant heterozygotes (RW) is shown in yellow and resistant homozygotes (RR) are represented in red.

allows for random shifts in allele frequencies and extinction. We provide the details of the algorithm in the Methods section, and the complete code is available on [GitHub](#) for reproducibility.

Results

Genetic complexities

Standing genetic variation. We estimate the standing genetic variation for target-site resistance in untreated Johnsongrass by vector \mathbf{v} in Equation (2.22). The obtained population composition provided the initial seed bank and rhizomes composition for our deterministic population dynamics and corresponding stochastic simulations. We explore how the standing genetic variation for target-site resistance depends on the associated fitness cost (c) and its dominance (k_c).

The frequency of resistance alleles R is controlled by the fitness cost on seed production (Figure 2.2 a). It decreases with an increasing fitness cost. For fecundity reductions in resistant homozygotes exceeding 12 %, we expect a resistance allele frequency in the order of the considered de novo mutation rate $\mu = 10^{-8}$ (HARMS et al., 1991; HAUGHN et al., 1987); indicating that competition with the sensitive type prevents the establishment of the resistant mutants. 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 (Figure 2.2 b). Remarkably, the expected frequency of heterozygotes shows little variation with the fitness cost. However, the expected frequency of resistant homozygotes increases with a decreasing fitness cost, most extreme for minimal fitness costs. This differential behaviour is because Johnsongrass is predominantly self-pollinated. Self-fertilised heterozygous plants (95 %) produce only one-half of heterozygous seeds and one-quarter of each homozygote. Therefore, we see an increased level of homozygosity under low resistance costs. For costs lower than 20 – 25 % (depending on the degree of dominance), we expect homozygous resistant plants to be more abundant than heterozygotes.

In natural populations of annual ryegrass, 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 et al., 2002). DÉLYE et al. (2013a) detected one individual carrying a specific ACCase target-site resistance in a

sample of 685 blackgrass (*Alopecurus myosuroides*) specimens collected prior to the introduction of ACCase-inhibiting herbicides. This abundance corresponds to a resistance allele frequency of $7.3 \cdot 10^{-4}$. According to our model, we expect resistance alleles and resistant individuals in untreated fields at a frequency of $1 \cdot 10^{-5}$ for a 0.1 % fitness cost on seed production (Data available on [GitHub](#)). Such a low fitness cost might not be detectable. Indeed, no associated fitness cost was detected for two out of three mutant ACCase alleles investigated by MENCHARI et al. (2008) in blackgrass. However, for a fitness cost on seed production of 30 %, reported for a mutant ACCase allele endowing herbicide resistance in Johnsongrass (PANOZZO et al., 2021), the expected resistance allele frequency and cumulative 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. This initial prevalence of target-site resistance is in the order of the spontaneous mutation rate (HARMS et al., 1991; HAUGHN et al., 1987) and considerably lower than what is reported in the literature (DÉLYE et al., 2013a; PRESTON et al., 2002).

Resistance allele dominance and fitness cost dominance. An increase in the dominance of the resistance cost leads to a relatively small reduction in the overall fitness of heterozygotes, resulting in a minor decrease in the eventual abundance of heterozygotes and sensitive homozygotes (Figure A.1). Sensitive plants decrease in abundance with heterozygous plants due to the prevalence of self-pollination. Reduced resistance dominance drastically lowers heterozygotes' fitness under herbicide application. The reduced survival strongly decreases the number of heterozygous and homozygous sensitive plants in the population.

A high dominance of resistance accelerates target-site resistance evolution when resistance is rare while hindering fixation of the resistance allele (Figure A.3 a). At low frequencies of target-site resistance, it is advantageous if heterozygotes have a high fitness and produce many offspring, of which most are resistant again. This, in turn, is a disadvantage for allele fixation since the sensitive allele is masked in heterozygotes.

Fitness cost. Using stochastic simulations, we investigate the impact of resistance cost on target-site resistance evolution and resulting population regrowth in herbicide-treated Johnsongrass. It is worth noting that the fitness cost determines the frequency of standing genetic variants, substantially affecting the probability of Johnsongrass escaping from control and regrowing.

Presumed that the resistant types manage to establish, the change in population composition is slightly faster under a low resistance cost on seed production ($c = 0.001$) compared to a high cost ($c = 0.3$) (Figure 2.3 a). The resistant homozygotes outcompete other types more easily (see also Figure A.3 b). Moreover, with a low fitness cost of 0.1 %, the population escapes control by the herbicide in over half of the simulation runs (Figure 2.3 b). Most of these simulated populations start to grow again within the first years (78.5 % within the first three years and 84.4 % within six years), where the year of escape from control and the start of regrowth is defined as the year in which the first homozygous resistant plants survive till reproduction. This resurgence is due to a high initial resistance allele frequency under low fitness costs (compare Figure 2.2). Thus, resistant individuals will likely be in the field at the start of treatment. The impact of the fitness cost itself is comparably low. Under herbicide application, resistant plants have a major selective advantage over sensitive plants, even with a high fitness cost on seed production. For a higher fitness cost of 30 %, less than a quarter of simulated populations escape from control by the herbicide and regrow within 30 years (Figure 2.3 b). The distribution of escapes from control is less skewed and more uniform, with almost no escapes in the first two years. Due to a low initial frequency of resistant types (compare Figure 2.2), the population rescue depends on the probability of a new mutation arising or resistant seeds in the seed bank germinating.

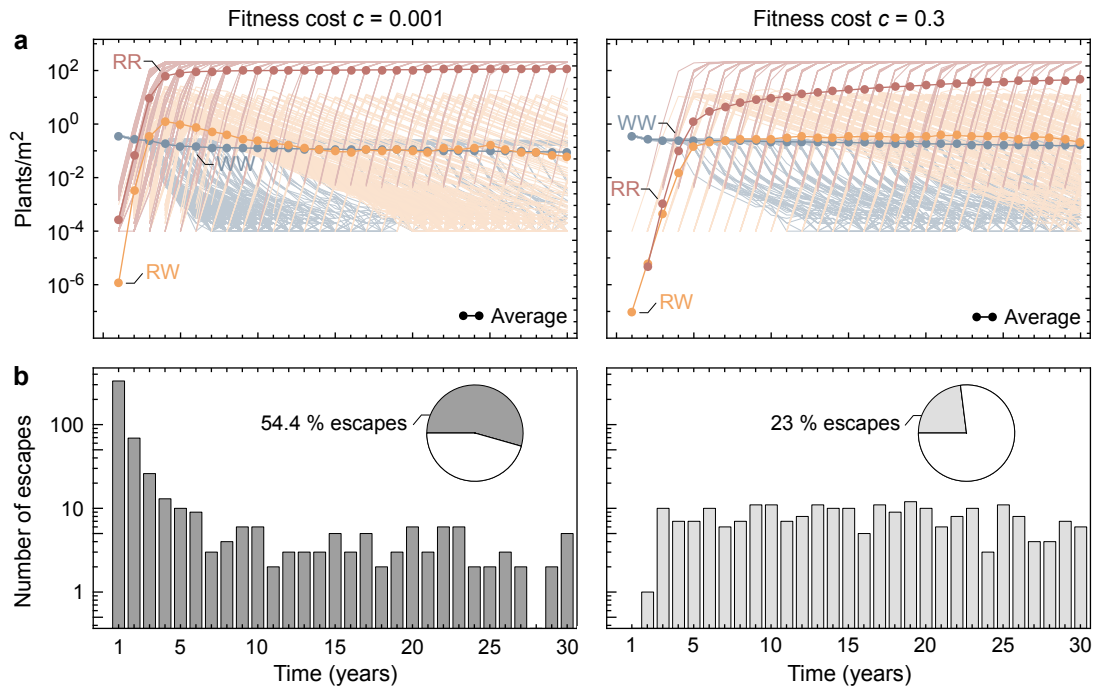


Figure 2.3 | Simulated target-site resistance evolution and resulting population regrowth in herbicide-treated Johnsongrass for low and high resistance cost. Shown are the results of 1000 simulation runs obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). The initial genotype composition differs between the low ($c = 0.001$) and high ($c = 0.3$) fitness cost (compare Figure 2.2 b). **a**, Changes in genotype composition of plants (\bar{P}) over 30 years of herbicide application for low and high resistance cost. The frequency of sensitive plants (WW) is shown in blue, resistant heterozygotes (RW) in yellow and resistant homozygotes (RR) are represented in red. The thick lines with closed circles correspond to the average of all simulation runs, and the thin lines represent the individual realisations. **b**, Distribution of escapes from control over 30 years of herbicide application for low and high resistance cost. Weed populations can regrow under herbicide treatment if a resistant plant establishes on the field and reproduces. The year of escape from control is the year in which the first homozygous resistant plant survives till reproduction. The pie charts display the proportion of simulation runs where the weed population escapes from control and regrows.

Sexual reproduction

Genetic diversity mainly results from sexual reproduction, while perennials of a well-adapted type rapidly spread through asexual propagation. Two characteristics of Johnsongrass' sexual reproduction, namely self-pollination and seed bank formation, seem particularly relevant for the population dynamics and target-site resistance evolution.

Self-pollination. Selfing increases the level of homozygosity in the population. Hence, the heterozygotes are significantly less abundant compared to a solely cross-pollinated population (Figure A.2), and the proportion of resistance alleles R increases faster under self-pollination (Figure 2.4 b). The interplay of a higher generation of homozygous-resistant individuals and the selective pressure exerted by the herbicide causes the accelerated target-site resistance evolution in self-pollinated weed populations. It might seem surprising that our model predicts an initial decline in homozygous-resistant seeds under herbicide application if the weed population is cross-pollinating. This decline is because, in our model, 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

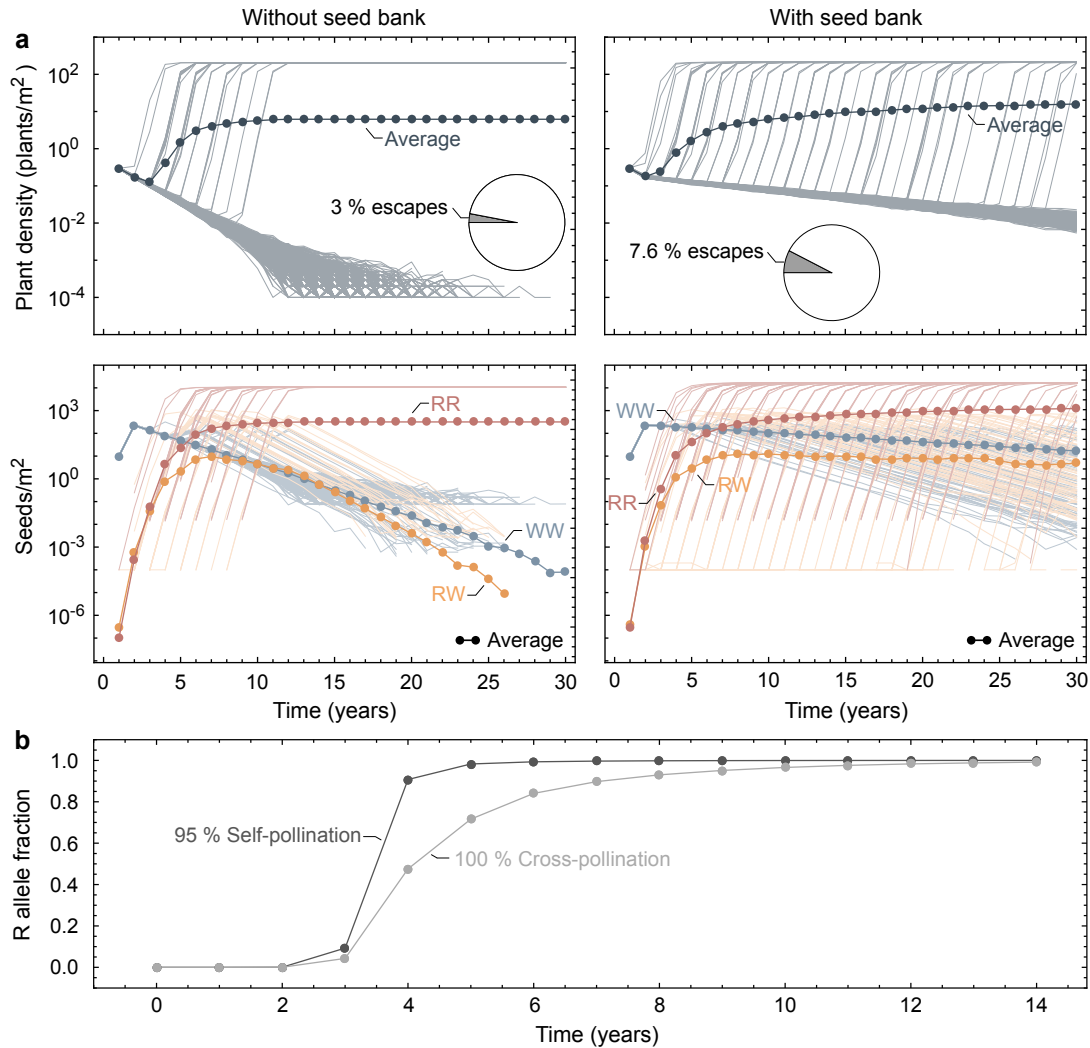


Figure 2.4 | Predicted target-site resistance evolution in herbicide-treated Johnsongrass depending on seed bank formation and self-pollination. The results are obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). **a**, Simulated changes in Johnsongrass density (\tilde{P}/A) and genotype composition of seeds (in the seed bank (B) if formed, otherwise produced seeds (S) that survived the winter) over 30 years of herbicide application and tillage depending on the formation of a seed bank. Shown are the results of 1000 simulation runs. The thick lines with closed circles correspond to the average of all simulation runs, and the thin lines represent the individual realisations. The frequency of sensitive seeds (WW) is shown in blue, resistant heterozygotes (RW) in yellow and resistant homozygotes (RR) are represented in red. The pie charts display the proportion of simulation runs in which the weed population escapes from control and regrows due to herbicide resistance evolution. **b**, Predicted changes in the frequency of the resistance allele R in Johnsongrass plants (\tilde{P}) under herbicide application for pure cross-pollination and 95 % self-pollination. Shown are predictions of our deterministic model.

sensitive pollen. Therefore, in a solely cross-pollinated population, pollination with sensitive pollen prevails.

Seed bank. Most simulated populations that lack a seed bank go extinct within 30 years under herbicide application and tillage (Figure 2.4 a). At the same time, we see no extinctions within this period for simulated populations that 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, observed in the stochastic simulations, is very wide for populations containing a seed bank. The escapes aggregate to 7.6 % of the realisations. In contrast, in simulations without a seed bank, populations started regrowing solely in the first nine years, accounting for 3 % of the runs.

Without a seed bank, the sensitive type disappears from almost all simulated populations, while most seed banks still contain some sensitive seeds after 30 years of recurrent herbicide application and tillage. The seed bank composition changes more slowly than the genotype composition of plants on the field, preserving genetic variation (see also Figure A.2). The seed bank only slightly delays the rise of resistance alleles in plants over several years of herbicide treatment (Figure A.3 c).

Control strategies

Combined control regimes. We examined the impact of a binary herbicide mixture and the integration of soil tillage on population dynamics and target-site resistance evolution in Johnsongrass. The integration of tillage with herbicide application significantly reduces the weed population size (Figure 2.5 a). This control improvement is caused by increased winter mortality of rhizomes and enhanced herbicide efficacy on rhizome shoots. Therefore, mutations are less likely to arise, and the proportion of simulation runs where the weed 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 (Figure 2.5 b). Our result contradicts the finding of LIU et al. (2019) that tillage delays the evolution of target-site resistance. However, we could qualitatively reproduce their result using a similar parameter set (see Figure A.4). Therefore, we acknowledge the deviation due to the significantly lower reproductive capacity implemented by LIU et al. (2019). The limited propagation increases the effect of higher rhizome mortality, overcoming the increased selective pressure on shoots under low frequencies of the resistance allele.

Adding a second herbicide with a reduced efficacy makes the simulated populations go extinct within 15 years and escapes very unlikely (Figure 2.5 a). Furthermore, the second herbicide delays the resistance evolution against the first herbicide (Figure 2.5 b). The reason is Johnsongrasses' inherent ecology. As the herbicide efficacy is higher on seedlings than on rhizome shoots, the proportion of surviving plants originating from rhizomes increases when a second herbicide with a different mode of action is added. The selective pressure on these rhizome plants is lower than on plants that emerge from seeds. This effect is even more substantial for higher efficacies of the second herbicide. However, suppose a herbicide with reduced efficacy is applied individually. In that case, Johnsongrass' exceptionally high reproductive capacity still allows the population to grow (Figure A.5). The high density, in turn, leads to de novo mutations conferring herbicide resistance.

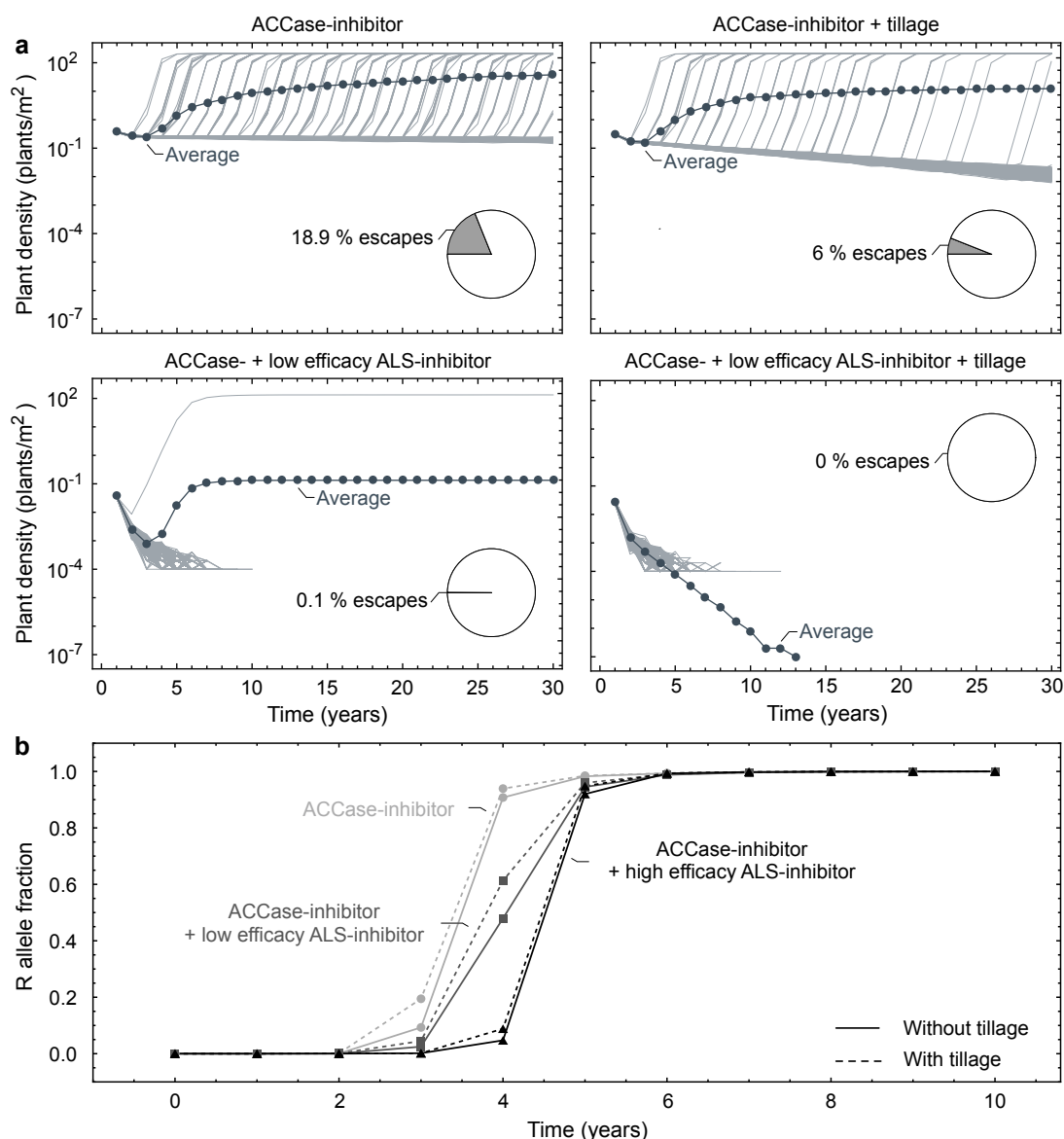


Figure 2.5 | Predicted population dynamics and target-site resistance evolution in Johnsongrass under different control regimes. The results are obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). **a**, Simulated changes in Johnsongrass density (\bar{P}/A) and proportion of populations escaping from control over 30 years of different control regimes. Shown are the results of 1000 simulation runs. The thick lines with closed circles correspond to the average of all simulation runs, and the thin lines represent the individual realisations. The pie charts display the proportion of simulation runs in which the weed population escapes from control and regrows due to herbicide resistance evolution. 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 efficacy, application of ACCase-inhibitor and ALS-inhibitor with low efficacy combined with tillage. **b**, Predicted changes in the frequency of the ACCase resistance allele R in Johnsongrass plants (\bar{P}) under different control regimes. Shown are predictions of our deterministic model. The distinct control strategies are: ACCase-inhibitor (light grey line with closed circles) or ACCase-inhibitor and ALS-inhibitor with low (dark grey line with closed squares) or high (black line with closed triangles) efficacy applied solely (solid line) or combined with tillage (dashed line).

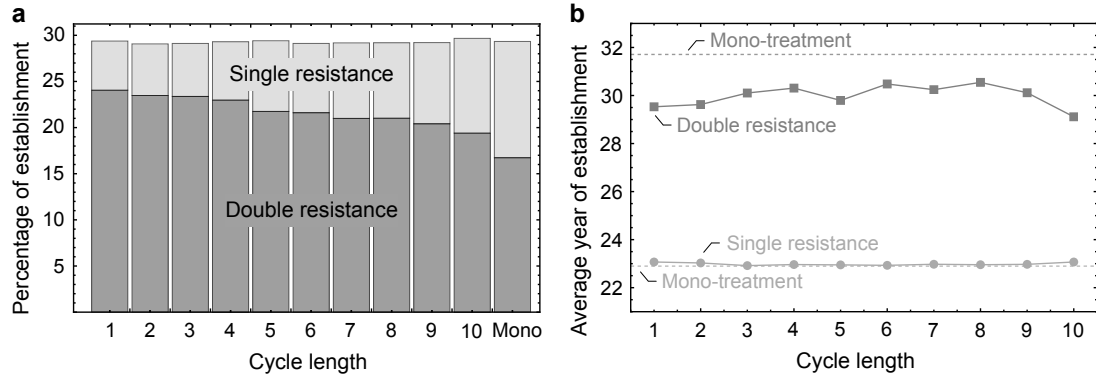


Figure 2.6 | Simulated evolution of target-site resistance in Johnsongrass under binary herbicide rotations and mono-treatment. Shown are results of 10^5 simulation runs obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). Considered are rotations of two herbicides with distinct target sites, ACCase- and ALS-inhibitors, Johnsongrass is known to develop target-site resistance towards. We assume equal efficacy here. The cycle length refers to the number of years one herbicide is recurrently applied before the treatment switches to the other herbicide. **a**, Proportion of simulated populations with resistant plants establishing on the field within 30 years of binary herbicide rotations depending on the cycle length and compared to mono-treatment. Bars show the percentage of simulation runs in which the plants (\tilde{P}) evolve resistance, with the light grey part referring to the population with single resistance, i.e. target-site resistance against only one of the herbicides, and the dark grey part reflecting double resistance. **b**, Average year of resistant plants establishing on the field within 30 years of binary herbicide rotations depending on the cycle length and compared to mono-treatment. The light grey line with closed circles displays the average year the first resistant plant survives till reproduction, and the dark grey line with closed squares depicts the average year of double resistant plants establishing on the field. The dashed lines show the reflective year of resistance occurrence under mono-treatment.

Herbicide rotation. We further investigated the effect of the cycle length in binary herbicide rotations on the establishment of resistant plants on the field (Figure 2.6). We consider rotations of two herbicides with different target sites and equal efficacies, where mixture and mono-treatment can be viewed as the two extremes. Cycle length refers to the number of years one herbicide is applied recurrently before treatment switches to the other herbicide.

Herbicide rotations fail to reduce the probability of resistant plants establishing on the field. Varying slightly around 29.3 % observed in mono-treatment, the proportion of simulated populations with resistant plants is not dependent on the cycle length (Figure 2.6 a). Due to the rapid population decrease, resistance evolves only in 0.02 % of the plants treated with a mixture of the two herbicides. In contrast, the emergence of double resistance decreases with increasing cycle length from 24 % in annual to 19.4 % in decennial rotation. In mono-treatment, we observe double resistant plants in 16.7 % of the simulated populations and none under the application of herbicide mixture. Resistance against one of the herbicides in a binary rotation is sufficient for the weed population to grow in the long run (compare Figure A.6). Further, herbicide rotations control sensitive plants with the same efficacy as a mono-treatment, leading to a similar probability of resistance adaptation. However, double resistance is costly for the plants, increasing the fitness cost on seed production in our model to 51 % compared to 30 % in single-resistant plants. When a herbicide is not applied, plants sensitive to this herbicide have a higher reproductive capacity. Therefore less frequent switching of herbicides reduces the risk of double resistance.

In our simulations, plants with single resistance established before the potential occurrence of double resistance. On average, the first resistant plants establish on the field after about 23 years of herbicide application, unaffected by cycling (Figure 2.6 b). Under herbicide mixtures, double resistance occurs on average six to seven years afterwards, about two years earlier than under mono-treatment. Nevertheless, rotations of the herbicides can delay the resistance allele fixation due to the alternating selective pressure and the fitness cost associated with resistance (compare Figure A.7). Moreover, herbicide rotations delay weed population regrowth as compared to the application of a single herbicide (compare Figure 2.5 b and Figure A.6). The average density reached by resistant populations after 50 years is 124 to 174 plants per m² for herbicide rotations, considerably lower than 203 plants per m² for mono-treatment.

Discussion

Through developing a comprehensive life cycle model of Johnsongrass, we have addressed the goals set out in the Introduction: we showed, (i) that standing genetic variation for target-site resistance is determined by the resistance cost, while its dominance has a minor impact; (ii) how high resistance dominance hastens the initial spread of resistance alleles but delays fixation due to the masking of sensitive alleles in heterozygotes; and the resistance cost affects probability and time of control escapes mainly by determining the standing genetic variation, with little effect of its dominance; (iii) that target-site resistance evolution is faster under self-pollination, and a seed bank can increase the probability of escape from control while maintaining genetic variation; (iv) that the integration of tillage with herbicide applications effectively reduces weed density and, thereby, the probability of escape from the control without delaying resistance evolution; how herbicide mixtures not only control Johnsongrass more effectively but also delay target-site resistance evolution due to the weeds inherent ecology; and that herbicide rotations do not delay the onset of resistance, but can slow resistance evolution and delay weed population regrowth. Beyond the specific implications for Johnsongrass control, our results have significance in the broader context of the essential concepts in controlling weeds: life cycle details, chemical and physical control methods, resistance evolution and the prospects of ensuring food security. Below we draw out these conclusions.

Our analysis suggests that target-site resistance is associated with a meagre, probably undetectable, fitness cost when found in untreated populations at frequencies two orders of magnitude higher than the spontaneous mutation rate. For fitness costs exceeding 12 % on seed production, the resistance allele frequency in populations that never encountered the herbicide before is expected to be similar in magnitude to the mutation rate. Not all mutations endowing target-site resistance in weeds are known to involve a fitness cost (MENCHARI et al., 2008). Furthermore, target-site resistance against ALS- and ACCase-inhibitors were found with a natural frequency in the order of 10^{-5} to 10^{-4} (DÉLYE et al., 2013a; PRESTON et al., 2002). According to our results, this would correspond to a meagre fitness cost. These findings coincide with the conclusions of PRESTON et al. (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.

We show a more than doubled probability of weed populations escaping herbicide control under a low fitness cost of 0.1 % compared to a fitness cost of 30 % on seed production. The latter cost was reported for a mutant ACCase allele endowing resistance in Johnsongrass (PANOZZO et al., 2021). The expected change in population composition is faster under low resistance costs, and resistant plants establish earlier on the field, leading to population recovery. Thus, if the fitness cost of target-site resistance is low, control of the weed population with a single herbicide might fail quickly and should be avoided. Applying herbicide mixtures might be a good strategy in such a case, and integrating other control measures like tillage (DIGGLE et al., 2003; PEERZADA et al., 2023).

Our results suggest that the fitness cost dominance has a minor impact on the prevalence of

resistance and population dynamics under control. Unlike the fitness cost of resistance, herbicide resistance itself has no effect on the weed population in the absence of herbicides. 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, later the effect reverses due to the masking of sensitive alleles in heterozygotes, delaying resistance evolution. This effect is observed for diploids and tetraploid perennials (HOLMES et al., 2022).

Especially, HOLMES et al. (2022) found that target-site resistance alleles spread faster in diploids than in tetraploids since tetraploid plants need to acquire twice as many mutations to reach the same proportion of resistance alleles. However, in Johnsongrass, the W2027C target-site mutation was not found to be present on more than two ACCase alleles (LORENTZ, 2014). The cause might be a fitness penalty or the absence of homologous recombination between Johnsongrass' two genomes (LORENTZ, 2014).

Our results indicate that target-site resistance evolution is faster in self-pollinating weed populations than in cross-pollinating populations. The increased homozygosity, combined with the herbicide's selective pressure, causes a more rapid increase in the relative frequency of resistance alleles given resistant plants established. The higher number of homozygous-resistant individuals in the standing genetic variation of self-pollinated weeds comes at the cost of a lower number of resistance-carrying individuals, decreasing the probability of resistance adaptation for dominant target-site resistance. In another simulation study, populations of seed-propagated annual weeds needed to be larger for evolving resistance if they were self-pollinated compared to purely cross-pollinated populations (DIGGLE et al., 2003). However, the opposite is true for recessive target-site resistance. As heterozygotes are fully susceptible recessive target-site resistance is more likely to establish in a herbicide-treated field if the weed is self-pollinated. We derived our initial composition of seeds and rhizomes from Equation (2.22) under the assumption of pure self-pollination. This genotype composition was used to calculate the population dynamics in an out-crossing population. However, we obtained similar results with a control-free initialisation phase of 30 years, allowing the population composition to stabilise under the assumed level of self-pollination.

Our 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 establishment probability of resistant mutants, leading to population regrowth. Therefore, we emphasise the necessity of considering the details of sexual reproduction and seed bank during risk assessment of control measures. It has been argued that herbicide resistance spreads mainly through asexual propagation (LIU et al., 2019). Once resistant plants have established, more resistant offspring might be generated via asexual propagation than seed production, considering the resistance cost on seed production and the possibility that produced seeds are not resistant. Resistance, however, generally originates in the seeds since mutations are most likely to arise during sexual reproduction. Even if standing genetic variants are present in the population, the number of seeds is considerably higher than that of rhizomes. Therefore, resistant individuals are more likely to be in the seed bank. Moreover, sexual reproduction is needed to generate homozygous-resistant offspring from heterozygous plants. We conclude that controlling the seed bank is essential in managing seed- and rhizome-propagated perennial weeds like Johnsongrass.

Integrating soil tillage with herbicide application further reduces Johnsongrass populations. We see no delay in the expected evolution of target-site resistance, as observed by LIU et al. (2019). Nevertheless, the probability of Johnsongrass regrowth is more than halved by the reduction in population size. This result agrees with field studies of Johnsongrass (MCWHORTER et al., 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.

Our results show herbicide mixtures control Johnsongrass most effectively, with minimal risk of weed populations escaping control. Including a second herbicide delays resistance evolution, with an increased effect for higher herbicide efficacies. These results agree with other simulation studies, demonstrating that herbicide mixtures effectively control resistance evolution and are superior to rotations (BUSI et al., 2020; DIGGLE et al., 2003). These conclusions were derived assuming independent action of the herbicides, no cross-resistance and full application rates. Applying two herbicides at full rates includes a higher economic cost than a mono-treatment and increases the environmental impact. Also, possible mechanisms of cross-resistance and synergistic or antagonistic interactions of the respective herbicides need to be explicitly considered. A comprehensive understanding of the effect of herbicide mixtures could be attained by incorporating herbicide interactions and cross-resistance into the present model and investigating different application rates and ratios.

In our simulations, binary herbicide rotations failed to reduce the risk of target-site resistance compared to mono-treatments. Frequent herbicide switching even favours the evolution of double resistance. 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. Though not delaying the onset of resistance, herbicide mixtures can slow resistance adaptation. 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. Also, target-site resistance is not necessarily associated with a considerable fitness cost (MENCHARI et al., 2008). Moreover, rotations of two herbicides delay weed population regrowth. A field experiment on blackgrass found an average reduction in density of 59 % after seven years for yearly rotation of herbicide site of action compared to mono-treatment (ZELLER et al., 2021). Our results show agreement with a simulation study by DIGGLE et al. (2003), in which rotations of herbicides were by far inferior to mixtures in controlling regrowth and herbicide resistance evolution. Overall our results promise an advantage of herbicide rotations over mono-treatment in terms of slowing population regrowth and target-site resistance evolution, presuming a fitness penalty, with no consistent advantage of fast or slow rotation. However, rotations do not reduce the resistance risk. Fast rotations even favour the occurrence of double resistance. In general, combinations of herbicides are far more effective in reducing herbicide resistance evolution and control failure.

Crop rotations, especially when they incorporate highly competitive crops like maize, can further reduce the population size of Johnsongrass (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 in a theoretical attempt. They highlight the effectiveness of a combination of crop rotation and rotation of herbicide classes in controlling Johnsongrass and delaying the evolution of herbicide resistance. However, they included resistance against only one of the herbicides, resulting from gene flow between resistant cultivated *Sorghum* and its wild relative. Our model can be extended to study the effect of crop rotations in terms of competition with the crop (BARGUÉS-RIBERA et al., 2020). This extension can be achieved by adding the corresponding competition terms in the equations for density-dependant mortality (Eq. (2.9)) and reproduction (Eqs. (2.11) and (2.12)).

Besides standing genetic variation and de novo mutations, resistance can also arise from pollen-mediated gene flow from neighbouring resistant populations or closely related resistant crops and the introduction of herbicide-resistant rhizomes and seeds via contaminated equipment (BECKIE et al., 2019; VRBNÍČANIN et al., 2017). MAXWELL et al. (1990) included seed immigration and gene flow from pollen in their population model of annual weeds, while HOLMES et al. (2022) modelled resistance evolution in Johnsongrass with gene flow from resistant *Sorghum*. Extending our model to a landscape scale with intra- and inter-specific gene flow will comprehensively assess resistance

evolution in herbicide-treated perennial weeds.

Overall, we have presented a theoretical framework capable of capturing the complex life cycle of perennial plants. The framework can be modified to model specific cases, as we have shown for Johnsongrass, and extended to answer pertinent questions from the points of view of different stakeholders in sustainable agriculture. For example, weed control measures always come with economic and socioecological facets. Thus, combining weed control and the associated socioeconomics in a single theoretical framework would be a fully functional tool built on our current model's foundation.

Methods

Routed in the biology of Johnsongrass or model describes the dynamics of a weed population growing in one field. We capture the perennial life cycle by considering five life-history stages: seeds, seedlings, rhizomes, shoots and adult plants. One time step of the model corresponds to one year. We implement stochastic simulations of this population-based model capturing the stochastic nature of mutations, genetic drift and extinctions. In this section, we describe the equations reflecting the plant reproduction and management interventions, as well as the stochastic simulations. We provide the derivation of the model parameters in the Supplementary Information with [Table B.1](#) showing all parameter values.

Life cycle

Johnsongrass plants are dormant throughout the winter, overwintering as seeds or rhizomes in the ground (WARWICK et al., 1983). At the beginning of a growing season, seeds germinate to produce seedlings, and shoots emerge from nodes on the rhizomes (PEERZADA et al., 2023). Mature plants produce new rhizomes and, after flowering, viable seeds (PEERZADA et al., 2023; WARWICK et al., 1983). [Figure 2.1](#) illustrates the life cycle of Johnsongrass and its implementation in the model with both reproduction pathways, sexual via seeds and asexual through rhizomes.

Initially, we neglect herbicide resistance and its genetics and concentrate solely on the plant life cycle. Rhizomes in the ground at the beginning of season t are denoted by $Z(t)$. Not all axillary buds formed on the nodes of a rhizome develop as shoots; some develop into secondary rhizomes or stay dormant (BEASLEY, 1970; PATERSON et al., 2020). 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 ($T(t)$) at time t is given by,

$$T(t) = g_Z b Z(t). \quad (2.1)$$

To begin with, 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 ($L(t)$) (LEGUIZAMÓN, 1986),

$$L(t) = g B. \quad (2.2)$$

Seedlings and shoots grow up to adult plants (P). We do not distinguish adult plants by their origin from seeds or rhizomes as they show similar growth characteristics (HOROWITZ, 1972; McWHORTER, 1961). Therefore,

$$P(t) = L(t) + T(t), \quad (2.3)$$

gives the number of adult plants ($P(t)$) present in season t . Plants form new rhizomes of which only a certain amount $(1 - d_Z)$ survives the winter to become the rhizomes of the next season $t + 1$,

$$Z(t + 1) = (1 - d_Z) P(t). \quad (2.4)$$

Control and resistance

Our model includes herbicide application and soil tillage as weed management techniques. We consider target-site resistance endowed by a single resistance allele R in a diploid genome. We include spontaneous mutations to the resistance allele R in sexual reproduction but no back mutations. We assume the homozygous resistant genotype confers complete resistance against the herbicide. The factor $0 \leq k_h \leq 1$ captures the dominance of resistance such that the herbicide controls WW plants with efficacy h and RW plants with efficacy $(1 - k_h)h$. Due to incomplete control of the rhizomes by herbicides and resprouting from dormant rhizome buds, the herbicide efficacy on rhizome shoots h_T is reduced compared to the herbicide efficacy on seedlings h_L (VIDRINE, 1989). Soil tillage controls Johnsongrass rhizomes by exposing the fragments to low temperatures during winter, increasing mortality d_Z^* (WARWICK et al., 1983), and enhances herbicide control of rhizome shoots h_T^* (HULL, 1970; TUESCA et al., 1999).

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

$$\mathbf{T}(t) = g_Z b \mathbf{Z}(t), \quad (2.5)$$

$$\mathbf{L}(t) = g \mathbf{B}, \quad (2.6)$$

$$\mathbf{P}(t) = \mathbf{L}(t) \begin{pmatrix} 1 - h_L & 0 & 0 \\ 0 & 1 - (1 - k_h)h_L & 0 \\ 0 & 0 & 1 \end{pmatrix} + \mathbf{T}(t) \begin{pmatrix} 1 - h_T & 0 & 0 \\ 0 & 1 - (1 - k_h)h_T & 0 \\ 0 & 0 & 1 \end{pmatrix}, \quad (2.7)$$

$$\mathbf{Z}(t+1) = (1 - d_Z) \mathbf{P}(t). \quad (2.8)$$

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, the herbicide efficacy on rhizome shoots h_T^* is additionally increased.

Intraspecific competition

Intraspecific competition affects the survival and the formation of reproductive structures in Johnsongrass (SCHWINNING et al., 2017; WILLIAMS et al., 1977). We describe the density-dependent mortality of Johnsongrass, resulting from intraspecific competition, by the hyperbolic self-thinning function

$$\tilde{\mathbf{P}}(t) = \mathbf{P}(t) \left(1 + m \frac{n_P(t)}{A} \right)^{-1}, \quad (2.9)$$

where $\tilde{\mathbf{P}}(t)$ is the vector of plants surviving till the end of season t , A the field size, $n_P(t) = \sum_{i \in \{WW, RW, RR\}} P_i(t)$ the total number of plants in the field without self-thinning (might be interpreted as young plants about to experience competition) and m^{-1} gives the highest possible density of Johnsongrass after self-thinning (YODA et al., 1963). Therefore,

$$\mathbf{Z}(t+1) = (1 - d_Z) \tilde{\mathbf{P}}(t), \quad (2.10)$$

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

Comprehensive competition experiments in Johnsongrass are missing. Therefore, we base the fertility functions on the assumption that the total seed and rhizome yield per square meter approaches a constant value at moderate to high densities (FIRBANK et al., 1985). Then the mean seed production ($f(t)$) and mean rhizome bud production ($b(t)$) per plant in season t can be

described by

$$f(t) = f \left(1 + a \frac{n_{\tilde{P}}(t)}{A} \right)^{-1}, \quad (2.11)$$

$$b(t) = b \left(1 + a \frac{n_{\tilde{P}}(t)}{A} \right)^{-1}, \quad (2.12)$$

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 $n_{\tilde{P}}(t) = \sum_{i \in \{WW, RW, RR\}} \tilde{P}_i(t)$ 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 determines the number of shoots that can emerge from this rhizome in the next growing season,

$$\mathbf{T}(t) = g_Z b(t-1) \mathbf{Z}(t), \quad (2.13)$$

gives the shoots emerging in season t .

Pollination and seed bank dynamics

We consider a fitness cost c on seed production associated with herbicide resistance (PANOZZO et al., 2021). A dominance factor $0 \leq k_c \leq 1$ controls the dominance of this fitness cost, such that the fecundity of RR and RS plants is reduced by c and $k_c c$, respectively, compared to the fecundity f of susceptible plants. Johnsongrass is primarily self-pollinated (WARWICK et al., 1983). Let p_{self} denote the proportion of self-pollination. Then the number of seeds with genotype i ($S_i^{self}(t)$) produced during season t by self-pollinated plants can be calculated as,

$$S_i^{self}(t) = p_{self} f(t) \tilde{\mathbf{P}}(t) \mathbf{m}^i \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 - k_c c & 0 \\ 0 & 0 & 1 - c \end{pmatrix}, \quad (2.14)$$

with

$$\mathbf{m}^i = (M_{WW \ WW}^i, M_{RW \ RW}^i, M_{RR \ RR}^i)^T, \quad (2.15)$$

where M_{jk}^i gives the proportion of type i seeds produced by a plant of genotype j pollinated by type k pollen. The seed production follows Mendelian inheritance and includes spontaneous mutations to the resistance allele R occurring with probability μ (see Supplementary Information). However, up to 5 % of cross-pollination has been observed in fields with Johnsongrass plants growing at sufficiently small distances (WARWICK et al., 1983). Over the t^{th} season a number of

$$S_i^{cross}(t) = \frac{(1 - p_{self}) f(t)}{n_{\tilde{P}}(t)} \tilde{\mathbf{P}}(t) \mathbf{M}^i \tilde{\mathbf{P}}(t)^T \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 - k_c c & 0 \\ 0 & 0 & 1 - c \end{pmatrix} \quad (2.16)$$

type i seeds ($S_i^{cross}(t)$) are produced by cross-pollinated plants, where

$$\mathbf{M}^i = \begin{pmatrix} M_{WW \ WW}^i & M_{WW \ RW}^i & M_{WW \ RR}^i \\ M_{RW \ WW}^i & M_{RW \ RW}^i & M_{RW \ RR}^i \\ M_{RR \ WW}^i & M_{RR \ RW}^i & M_{RR \ RR}^i \end{pmatrix}, \quad (2.17)$$

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

i seeds ($S_i(t)$) produced in season t ,

$$\begin{aligned} S_i(t) &= S_i^{self}(t) + S_i^{cross}(t) \\ &= f(t) \widetilde{\mathbf{P}}(t) \begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 - k_c c & 0 \\ 0 & 0 & 1 - c \end{pmatrix} \left[\frac{1 - p_{self}}{n_{\widetilde{\mathbf{P}}}(t)} \mathbf{M}^i \widetilde{\mathbf{P}}(t)^T + p_{self} \mathbf{m}^i \right]. \end{aligned} \quad (2.18)$$

Johnsongrass seeds are dormant and form a seed bank in the ground (PEERZADA et al., 2023; WARWICK et al., 1983). Before new seeds enter the seed bank B , they are subject to post-dispersal predation and might lose viability or decay till the beginning of the next season with probability d_S (BAGAVATHIANNAN et al., 2013). Within a season t , only a certain proportion g of seeds in the seed bank ($B(t)$) germinates to produce seedlings ($L(t)$),

$$\mathbf{L}(t) = g \mathbf{B}(t), \quad (2.19)$$

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 et al., 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 ,

$$\mathbf{B}(t + 1) = (1 - d_B) (1 - g) \mathbf{B}(t) + (1 - d_S) \mathbf{S}(t). \quad (2.20)$$

Standing genetic variation

The natural frequency of target-site resistance exceeds the rate at which these mutations spontaneously occur, suggesting that herbicide resistance adaptation primarily proceeds from standing genetic variants (KREINER et al., 2017). We derive a matrix model approximation of the entire life cycle dynamics. We use this matrix model to calculate the expected standing genetic variation for target-site resistance depending on the fitness cost associated with resistance and its dominance.

Let $\mathbf{P}(t) = (P_{WW}(t), P_{RW}(t), P_{RR}(t))$ and $\mathbf{B}(t) = (B_{WW}(t), B_{RW}(t), B_{RR}(t))$ 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. We assume an infinite density limit (no self-thinning) and maximum reproduction ($f(t) = f$, $b(t) = b$, $\forall t \geq 0$). Further, we neglect cross-pollination ($p_{self} = 1$) here. Under these assumptions, the population dynamics of Johnsongrass can be approximated by

$$\begin{aligned} &(\mathbf{P}(t + 1), \mathbf{B}(t + 1)) \\ &= (\mathbf{P}(t), \mathbf{B}(t)) \underbrace{\begin{pmatrix} g_Z (1 - d_Z) b \mathbf{I} + g (1 - d_S) f \mathbf{D} \widetilde{\mathbf{M}} & (1 - d_S) f \mathbf{D} \widetilde{\mathbf{M}} \\ g (1 - d_B) (1 - g) \mathbf{I} & (1 - d_B) (1 - g) \mathbf{I} \end{pmatrix}}_{=: \mathbf{E}}, \end{aligned} \quad (2.21)$$

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 the resistance 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 Supplementary Information) to derive the long-term population dynamics

$$\lim_{t \rightarrow \infty} \frac{(\mathbf{P}(t+1), \mathbf{B}(t+1))}{\rho^t} = (\mathbf{P}(1), \mathbf{B}(1)) \mathbf{u}^T \mathbf{v}, \quad (2.22)$$

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} , regardless of the initial population. Therefore \mathbf{v} gives us a rough estimate of the expected standing genetic variation for target-site resistance in a Johnsongrass population never exposed to the herbicide (see Figure 2.2).

Stochastic simulations

As in the underlying deterministic model, we model the population dynamics 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 germination probability, death probabilities, and herbicide efficacy, 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 $B_{\text{WW}}(t)$ and g . We use multinomial random numbers 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 self-pollination by heterozygous plants and the inheritance vector $(\frac{1}{4}, \frac{1}{2}, \frac{1}{4})$ as parameters. For further details, we refer to the code available on [GitHub](#).

Data Availability. All data and plots are available on [GitHub](#).

Code Availability. All code is available on [GitHub](#).

Acknowledgements. We thank Demetris Taliadoros for helpful discussions and acknowledge the generous funding from the Max Planck Society.

Author Contributions Statement. Both authors conceived the study, developed the model, analysed the results and wrote the manuscript. D.L. formulated the mathematical model, the algorithm and ran the simulations.

Competing Interests Statement. The authors declare no competing interests.

CHAPTER 3

Assessing the probability and time of weed control failure due to herbicide resistance evolution

This chapter is currently under review and published as a preprint. Lauenroth, D., and Gokhale, C.S. *Assessing the probability and time of weed control failure due to herbicide resistance evolution*. bioRxiv (2024). DOI: [10.1101/2024.04.26.591266](https://doi.org/10.1101/2024.04.26.591266).

Abstract

The recurrent exposure to herbicides in agricultural landscapes forces weeds to adapt in a race against extinction. What role newly arising mutations and pre-existing variation play in this evolution of herbicide resistance is critical for developing management strategies. Here, we present a model of rapid adaptation in response to strong selection, capturing complex life cycles of sexual and asexual reproduction and dormancy in a perennial weed. Using a multitype Galton-Watson process, we derive the probability of herbicide resistance evolution and the waiting time distribution until resistant plants appear in the field. We analyse the effect of seed bank dynamics and details of the reproductive system in defining the probability and timing of resistance adaptation in *Sorghum halepense*. Further, we investigate key factors determining the primary source of adaptive variation. We find that even small fitness costs associated with resistance reduce adaptation from standing genetic variation. For herbicide resistance inherited in a (incompletely) dominant fashion, self-pollination also diminishes standing variation for herbicide resistance by increasing the homozygosity. Our study highlights the importance of seed banks for weeds' adaptive potential, preserving genetic information of forgone selection and prolonging the period in which the population can adapt.

Introduction

'The assurance of global food security via agricultural yields is [...] in need of a highly integrative management approach, in which population geneticists, evolutionary biologists, and weed scientists investigate the nature of resistance adaptation and the factors that affect it to both predict and prevent the evolution of herbicide-resistant weeds.' KREINER et al., 2017, p. 612 The commercialisation of herbicides during and after the Second World War has vastly improved weed management compared to labour- and cost-intensive mechanical weed control, enabling the intense farming we see nowadays (OERKE, 2006; TUDI et al., 2021). However, analogous to the antibiotic crisis, we ran into a situation where the global presence of resistance towards most known herbicide modes of action threatens the efficacy of chemical weed management (PEROTTI et al., 2020; POWLES et al., 2010). The evolution of herbicide resistance is an example of repeated rapid adaptation under human-induced strong selection (BAUCOM, 2016). It is possible via mutations in genes encoding the targeted protein (target-site resistance) or through nontarget-site alterations like changes in metabolic pathways, reducing the exposure of target proteins to herbicides (BAUCOM, 2016). Without gene flow from other populations, adaptation can result from standing genetic variation and *de novo* mutations, with existing variation leading to more rapid adaptation in novel environments (BARRETT et al., 2008). Identifying the source of adaptive variation is critical

to developing strategies to manage herbicide resistance (KREINER et al., 2017). In weeds with substantial standing variation for herbicide resistance, managing the seed bank, for example with stale seedbed preparation, might be vital (BENVENUTI et al., 2021). Contrastingly, if *de novo* mutations serve as the primary source of adaptation, preventing weed seed set by panicle removal or selective herbicide crop topping applications can be a useful (TIDEMANN et al., 2021).

Standing variation for herbicide resistance was present in populations of weeds such as annual ryegrass (*Lolium rigidum*) and blackgrass (*Alopecurus myosuroides*) that were never exposed to herbicides, even predating their introduction in blackgrass (DÉLYE et al., 2013a; PRESTON et al., 2002). Recent genomic analysis and simulations by KERSTEN et al. (2023) highlight the prevalent role of standing genetic variation in target-site resistance adaptation of the annual weed blackgrass. Conversely, in species like common waterhemp (*Amaranthus tuberculatus*) and foxtail millet (*Setaria italica*), high resistance costs might diminish standing variation, making novel mutations a more likely source of adaptation (VIGUEIRA et al., 2013). Genomic data of several waterhemp populations supports a prevailing role of *de novo* mutations in target-site resistance of this species (KREINER et al., 2022). Yet, the precise determinants of the primary source of adaptive variation in herbicide resistance remain unclear.

Branching processes are well-established in biological studies for modelling population evolution (ATHREYA et al., 1972; HARRIS, 1963; KIMMEL et al., 2002; SANTER et al., 2020). They have been used to model populations at risk of extinction due to low reproduction rates after an environmental change, investigating how the emergence of an adapted mutant could prevent population extinction via evolutionary rescue (AZEVEDO et al., 2021; ORR et al., 2008; UECKER et al., 2014). Multitype branching processes can yield further information about the waiting time until such a mutant appears (ALEXANDER, 2016; SERRA et al., 2007). Despite recognition of the value of concepts from evolutionary rescue for understanding population genetics of herbicide resistance evolution (KREINER et al., 2017; NEVE et al., 2014), this approach is lacking in weed science literature. Our work addresses this gap.

Using multitype Galton-Watson processes, we analyse the probability and timing of herbicide resistance evolution in perennial weeds causing treatment failure. In this context, our study focuses on the influence of specific weed traits – asexual propagation, self-pollination and seed dormancy. We emphasise the role of the seed bank in the evolution of herbicide resistance, preserving genetic information of forgone selection and prolonging the time for adaptation. Further, we study critical ecological and evolutionary factors that modify the role of *de novo* mutations and standing genetic variation in the evolution of herbicide resistance. The seed bank size determines the absolute frequency of pre-existing variants for target-site resistance, and the seed production capacity of the weed defines the number of newly arising mutations, affecting their relative contribution to resistance adaptation. Self-pollination and fitness costs associated with resistance both reduce adaptation from the standing genetic variation. Our theoretical results explain the variation in primary sources of adaptive variation observed in different weed species, determining effective strategies for herbicide resistance management.

Methods

We explore the dynamics of rare beneficial alleles within large populations facing sudden environmental shifts, like herbicide application. In particular, we model the appearance and spread of target-site mutations, conferring resistance to the herbicide, in a primarily sensitive weed population declining in response to the treatment.

We illustrate our modelling framework on the example of a perennial weed, *Sorghum halepense* (Johnsongrass), which reproduces sexually via seeds and asexually through rhizomes (PEERZADA et al., 2023; WARWICK et al., 1983). The weed enters dormancy during winter, surviving as seeds and rhizomes underground until new young plants emerge in spring and mature over the season to produce new rhizomes and seeds (PEERZADA et al., 2023; WARWICK et al., 1983). These seeds,

known for their high dormancy, contribute to forming a seed bank, ensuring the species' viability in the soil (PEERZADA et al., 2023; WARWICK et al., 1983). We illustrate this cyclical life pattern of Johnsongrass along with the further plant stages and herbicide application in Figure 3.1 a. The mathematical representation of the model is shown in Figure 3.1 b.

In LAUENROTH et al. (2023), we presented a deterministic model of Johnsongrass' life cycle capturing all life-history stages and intraspecific competition between the plants, allowing the prediction of long-term population dynamics (Figure 3.1 a). In the present study, we are interested in the probability and timing of herbicide resistance evolution. With herbicide exposure, the common sensitive allele, carried homozygously by the majority of the population, becomes maladaptive. Target-site mutations, in particular, can endow a high level of resistance (SCARABEL et al., 2014), warranting that the first resistant plant surviving till reproduction almost certainly ensures population survival. We, therefore, concentrate on adult plants and seeds within the seed bank, counted at the end of each season before the newly produced seeds enter the seed bank. Focussing on the invasion dynamics, our branching process model captures the early phase of adaptation in which the resistance allele is rare.

Branching process model

We consider a total of m genotypes, which may correspond to different mutations at one locus or several loci or take different levels of ploidy into account, denoted by $\{1, 2, \dots, m\}$, where genotype 1 is the prevailing sensitive type, and the rest are rare. This setup leads to tracking $2m$ types $\{1, 2, \dots, 2m\}$, with the first m being plants and the latter half representing seed genotypes. We define $C \subset \{1, 2, \dots, m\}$ as the set of plant types expected to grow positively, termed supercritical types, while the remainder, $\{1, 2, \dots, m\} \setminus C$, are subcritical, facing extinction unless a successful mutant arises. Producing at most one offspring, seed types $\{m + 1, m + 2, \dots, 2m\}$ are as well subcritical.

We formally assume all individuals – seeds and plants – perish at the end of the season and produce offspring independently, constituting the following season's population. A seed that stays dormant and part of the seed bank thus has one identical offspring – itself – in the next season. While seeds have at most one offspring in the next season, plants can generate multiple offspring, either as new plants or seeds (illustrated in Figure 3.1 c). The population in season n is represented by the random vector $\mathbf{Z}_n = (Z_{n,1}, Z_{n,2}, \dots, Z_{n,2m})$, where the first m entries count the different plant types and the second half contains absolute genotype frequencies in the seed bank. The offspring of type i individuals are described by independent and identical distributed random vectors $\mathbf{X}_i^{(l)}$, taking values in \mathbb{N}_0^{2m} , where the entries $X_{ij}^{(l)}$ give the number of type j offspring produced by the l -th type i individual. The population vector \mathbf{Z}_n is then for $n \in \mathbb{N}_0$ recursively defined by a given initial population $\mathbf{Z}_0 \in \mathbb{N}_0^{2m}$ and

$$\mathbf{Z}_{n+1} = \sum_{i=1}^{2m} \sum_{l=1}^{Z_{n,i}} \mathbf{X}_i^{(l)}. \quad (3.1)$$

The time discrete homogeneous Markov chain $(\mathbf{Z}_n)_{n \in \mathbb{N}_0}$ with state space \mathbb{N}_0^{2m} is a multitype Galton-Watson process modelling the weed population dynamics over time. We show a realisation of a simplified process in Figure 3.1 c.

Offspring generating function. We assume plants produce a Poisson-distributed number of seeds and rhizome buds, which might die over winter before they potentially develop into plants. A herbicide application can kill sensitive plants, while dormant seeds are unaffected. The processes, including survival and development, are considered independent among units, leading to offspring numbers that follow independent Poisson distributions (ACHARYA et al., 2014). We assume vegetative propagation produces genetically identical offspring, neglecting somatic mutations. Seeds can differ

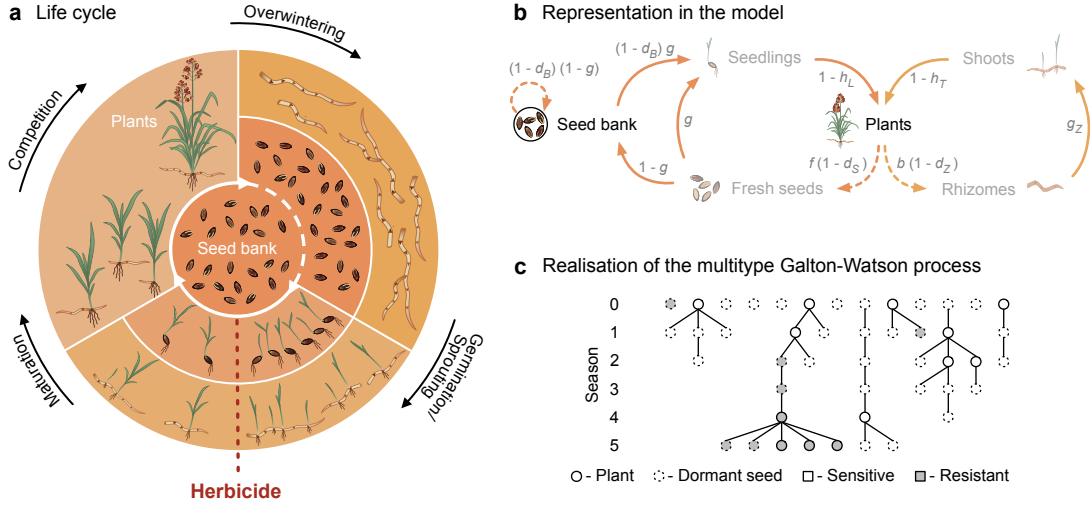


Figure 3.1 | Schematic illustration of Johnsongrass' life cycle and its representation in our model. **a**, Diagram of Johnsongrass' life cycle. Johnsongrass reproduces sexually via seeds (inner ring) and asexually through rhizomes (outer ring). Seeds can stay dormant in the ground for several years, forming a seed bank (central circle). New seeds and seeds from the seed bank might germinate in spring or stay dormant as part of the seed bank (expressed by the dashed line). Rhizomes give rise to shoots in the first spring after their production. Herbicide application (red dashed line) can kill susceptible seedlings and shoots. Surviving plants compete for resources as they mature. The aboveground plant material dies in winter, and Johnsongrass overwinters as seeds and rhizomes in the ground (adapted from LAUENROTH et al. (2023)). **b**, Representation of the life cycle in our model. The left part of (b) corresponds to the sexual reproduction of Johnsongrass, and the right part represents the asexual propagation. Our model tracks the seed bank and reproducing plants; the other life history stages depicted transparently are not explicitly modelled. Solid arrows depict within-season dynamics, and dashed arrows show dynamics between seasons. Survival probabilities and fecundity are shown in grey next to the corresponding arrows: number of seeds f and buds on the rhizome b ; winter survival of fresh seeds $1 - d_S$, seeds in the seed bank $1 - d_B$ and rhizome buds $1 - d_Z$; probability of seed germination g and rhizome bud sprouting g_Z ; herbicide survival of seedlings $1 - h_L$ and shoots $1 - h_T$. **c**, Illustration of a realisation of our multitype Galton-Watson process. Shown is the phylogeny of a branching process resembling a herbicide-treated weed population over six seasons. Open circles depict plants, and dashed circles represent dormant seeds in the seed bank; sensitive individuals are white, and grey are individuals resistant to the herbicide.

genetically from the parent plants due to Mendelian inheritance and mutations – mutations and back mutations at a given rate μ . Johnsongrass primarily self-pollinates, but since cross-pollination is possible at sufficiently high densities (WARWICK et al., 1983), our model captures a continuum, presuming abundant pollen results in complete ovule fertilisation. Our focus is on the proliferation of rare alleles, positing that a prevalent homozygous genotype, labelled genotype 1, predominates while alternative allele carriers are rare. We simplify by assuming these rare types do not engage in cross-pollination with each other, but type 1 plants can cross-pollinate with all genotypes. Let χ_S and χ_R denote the absolute frequency of the sensitive type 1 and rare resistant plants, respectively. The fraction of sensitive plants pollinated by resistant plants' pollen is $\chi_S \frac{\chi_R}{\chi_S + \chi_R} \approx \chi_R$ and the fraction cross-pollinated by pollen from sensitive plants is $\chi_S \frac{\chi_S}{\chi_S + \chi_R} \approx \chi_S$. It is, hence, natural to count all offspring produced by matings between type 1 and rare type plants as the rare type's offspring. Since the inheritance does not depend on which mating partner contributes

the male or female gametes, this altered offspring assignment does not constitute a deviation from Mendelian inheritance. These approximations of the cross-pollination allow us to capture sexual reproduction in the asexual Galton-Watson process. Taken together, the genotype j plants and dormant seeds produced by a plant of genotype i are independent and Poisson distributed, with means λ_{ij} and λ_{im+j} respectively, with Johnsongrass-specific parameters detailed in the Supplementary Information.

Seeds in the seed bank face three possible outcomes: germination into a plant of the same genotype i with probability α_i , remaining dormant with probability β , or disappearing without offspring with probability γ_i .

This defines the offspring distributions p_1, \dots, p_{2m} of the branching process, where $p_j(\mathbf{r})$ specifies the probability of a type j individual leaving offspring vector \mathbf{r} in the following season. For plants of genotype i , the generating function of their offspring is then given by

$$\begin{aligned} f^i(\mathbf{s}) &= \sum_{\mathbf{r} \in \mathbb{N}_0^{2m}} p_i(\mathbf{r}) \prod_{l=1}^{2m} s_l^{r_l} = \sum_{\mathbf{r} \in \mathbb{N}_0^{2m}} \prod_{l=1}^{2m} p_i(r_l) s_l^{r_l} \\ &= \prod_{l=1}^{2m} \sum_{r_l=0}^{\infty} \frac{\lambda_{il}^{r_l}}{r_l!} e^{-\lambda_{il}} s_l^{r_l} = \prod_{l=1}^{2m} e^{\lambda_{il}(s_l-1)} = \exp\left(\sum_{l=1}^{2m} \lambda_{il}(s_l-1)\right), \end{aligned} \quad (3.2)$$

where the second equality holds because we have independent Poisson distributions resulting from Poisson sampling (ACHARYA et al., 2014). The order of summation can be changed to reach the third equality due to the absolute convergence of the series on $[-1, 1]$. For seeds of genotype i ,

$$f^{m+i}(\mathbf{s}) = \alpha_i s_i + \beta s_{m+i} + \gamma_i, \quad (3.3)$$

is the offspring-generating function. The offspring generating functions for the different genotypes of plants and seeds will be combined in the vector $\mathbf{f}(\mathbf{s}) := (f^1(\mathbf{s}), f^2(\mathbf{s}), \dots, f^{2m}(\mathbf{s}))$.

Extinction probabilities. A branching process ultimately either becomes extinct or grows without bounds. The interest lies in the extinction and survival probabilities, which reflect the chance of treatment success (weed eradication) or failure (weed proliferation). We denote the probability that a process started by one type i individual becomes extinct with q_i . We summarise the type-specific extinction probabilities in a vector $\mathbf{q} = (q_1, q_2, \dots, q_{2m})$. The extinction probability vector \mathbf{q} is the solution of

$$\mathbf{q} = \mathbf{f}(\mathbf{q}), \quad (3.4)$$

in the cube $[0, 1]^{2m}$ closest to $\mathbf{0}$ (SEWASTJANOV, 1974, p.115). The vector $\mathbf{1}$ invariably satisfies Equation (3.4), indicating that in the absence of any other solutions within $[0, 1]^{2m}$, the associated branching process is sure to become extinct, with a probability of 1, regardless of the initial population.

The first m equations in (3.4) stem from the offspring-distributions of the plants, yielding extinction probabilities of processes initialised by a plant; the second half corresponds to processes starting with a single seed. Rearranging the bottom half of the equations in (3.4),

$$q_{m+i} = f^{m+i}(\mathbf{q}) = \alpha_i q_i + \beta q_{m+i} + \gamma_i, \quad (3.5)$$

we can express the extinction probabilities of processes starting with a single seed in terms of the

extinction probabilities when starting with a plant of the same genotype,

$$q_{m+i} = \frac{\alpha_i}{1-\beta} q_i + \frac{\gamma_i}{1-\beta}. \quad (3.6)$$

Substituting this relation into the top half of the equations in (3.4),

$$\begin{aligned} q_i = f^i(\mathbf{q}) &= \exp \left(\sum_{j=1}^m \left(\lambda_{ij} + \frac{\alpha_j}{1-\beta} \lambda_{i m+j} \right) q_j \right) \exp \left(\sum_{j=1}^m \lambda_{i m+j} \left(\frac{\gamma_j}{1-\beta} - 1 \right) - \lambda_{ij} \right) \\ &= \tilde{\gamma}_i \exp \left(\sum_{j=1}^m \tilde{\lambda}_{ij} q_j \right), \end{aligned} \quad (3.7)$$

where $\tilde{\lambda}_{ij} = \lambda_{ij} + \frac{\alpha_j}{1-\beta} \lambda_{i m+j}$, $\tilde{\gamma}_i = \exp \left(\sum_{j=1}^m \lambda_{i m+j} \left(\frac{\gamma_j}{1-\beta} - 1 \right) - \lambda_{ij} \right)$, $i, j \in \{1, 2, \dots, m\}$, we can effectively halve the dimension of the equation system.

Probability of treatment failure. The extinction probabilities, \mathbf{q} , for processes starting with one individual enable us to determine the probability of extinction or survival for a process initiated with any initial population \mathbf{z}_0 , corresponding to weed treatment success or failure.

$$\begin{aligned} \mathbb{P}(\text{'treatment failure'} \mid \mathbf{Z}_0 = \mathbf{z}_0) &= 1 - \mathbb{P}(\text{'treatment success'} \mid \mathbf{Z}_0 = \mathbf{z}_0) \\ &= 1 - \prod_{i=1}^{2m} q_i^{z_{0,i}}. \end{aligned} \quad (3.8)$$

The initial population distribution influences the extinction probability. Assuming Poisson distributed plant and seed numbers, with means n_P and n_B , the initial counts of each type are independent and Poisson distributed, with means $n_P v_i$ and $n_B v_i$, for seeds and plants respectively, where v_i denotes the frequency of genotype i in the pre-existing variation (ACHARYA et al., 2014). The probability of treatment failure is then analogue to (3.2) given by,

$$\begin{aligned} \mathbb{P}(\text{'treatment failure'}) &= 1 - \sum_{\mathbf{z}_0 \in \mathbb{N}_0^{2m}} \mathbb{P}(\mathbf{Z}_0 = \mathbf{z}_0) \prod_{i=1}^{2m} q_i^{z_{0,i}} \\ &= 1 - \exp \left(n_P \sum_{i=1}^m v_i (q_i - 1) + n_B \sum_{i=1}^m v_i (q_{m+i} - 1) \right). \end{aligned} \quad (3.9)$$

We can use Equation (3.9) to analyse the role standing genetic variation and *de novo* mutations play in herbicide resistance adaptation. By setting $v_1 = 1$ and $v_i = 0$ for $i > 1$, corresponding to the start population consisting of only sensitive homozygotes, we get the probability of treatment failure through *de novo* mutations

$$\begin{aligned} &\mathbb{P}(\text{'treatment failure through de novo mutation'}) \\ &= 1 - \exp(n_P(q_1 - 1) + n_B(q_{m+1} - 1)). \end{aligned} \quad (3.10)$$

Excluding mutations, we get the offspring generating function without mutation $\tilde{\mathbf{f}}$ and corresponding mutation free extinction probabilities as $\tilde{\mathbf{q}}$. To calculate the probability of treatment failure through standing genetic variation, we insert $\tilde{\mathbf{q}}$ in Equation (3.9),

$$\begin{aligned} &\mathbb{P}(\text{'treatment failure through standing variation'}) \\ &= 1 - \exp \left(n_P \sum_{i=1}^m v_i (\tilde{q}_i - 1) + n_B \sum_{i=1}^m v_i (\tilde{q}_{m+i} - 1) \right). \end{aligned} \quad (3.11)$$

Adaptation through *de novo* mutations and pre-existing genetic variation are intertwined, potentially contributing to population survival. Unlike the approach of HERMISSON et al. (2005) for adaptation from pre-existing variation and new mutations, our model does not separate the total adaptation probability into contributions from standing variation and *de novo* mutations as we have also included back mutations. Thus, Eqs. (3.10) and (3.11) represent the adaptation probabilities in populations without pre-existing target-site resistance or those that cannot mutate, relying solely on existing variation, respectively.

Waiting times. The time until resistant plants establish, allowing for the survival of the weed population against herbicide control, is critical. We apply findings from ALEXANDER (2016) to calculate the probability that resistant plant types have not appeared and to determine the distribution of waiting time for their emergence.

C denotes the set of supercritical types corresponding to resistant plants. We calculate the probability of C not appearing by a specific time. A particular type has yet to emerge by generation n if it did not appear by generation $n-1$ and no generation $n-1$ individual produced such offspring. Therefore, the probability $\Pi_i(n)$ that resistant plants have not appeared in a type i individual's lineage by generation n is given by

$$\mathbf{\Pi}(n) := (\Pi_1(n), \Pi_2(n), \dots, \Pi_{2m}(n)) = \mathbf{f}_*^{(n)}(\mathbf{1}_*), \quad (3.12)$$

where $\mathbf{f}_*^{(n)}$ denotes the n -th iteration of the offspring generating function \mathbf{f} with the components corresponding to the resistant plants C fixed at zero, i.e. $f_*^j = f^j$ unless $j \in C$ when it is 0. $\mathbf{1}_*$ is the vector of length $2m$ where all entries are 1 except for the entries of the resistant plants C , which are again 0.

The random variable N denotes the time until the first appearance of a resistant plant (first resistant plant establishes and sets seed). The probability of a resistant plant appearing until a given generation n is the complementary probability of no such plant appearing until generation n . Hence, we can derive the cumulative distribution of N from the probabilities $\Pi_i(n)$ in (3.12) that no resistant plant has appeared in the lineage of a type i individual by generation n . If the process starts with just one individual of type i ,

$$\mathbb{P}(N \leq n \mid \mathbf{Z}_0 = \mathbf{e}_i) = 1 - \Pi_i(n). \quad (3.13)$$

No resistant plants arise in the branching process until some time, if and only if no such individuals appear in any of the independent lines. Thus, we can express the cumulative function of N analogue to the treatment failure probability in (3.9) as

$$\begin{aligned} \mathbb{P}(N \leq n) &= 1 - \sum_{\mathbf{z}_0 \in \mathbb{N}_0^{2m}} \mathbb{P}(\mathbf{Z}_0 = \mathbf{z}_0) \prod_{i=1}^{2m} \Pi_i(n)^{z_{0,i}} \\ &= 1 - \exp \left(\sum_{i=1}^m v_i (n_P (\Pi_i(n) - 1) + n_B (\Pi_{m+i}(n) - 1)) \right). \end{aligned} \quad (3.14)$$

Since the process is time discrete, the probability mass function of the waiting time N until the

appearance of a resistant plant is given by

$$\begin{aligned}
 \mathbb{P}(N = n) &= \mathbb{P}(N \leq n) - \mathbb{P}(N \leq n - 1) \\
 &= \exp\left(-n_P \sum_{j \in C} v_j\right) \\
 &\quad \cdot \left(\exp\left(n_P \sum_{i \in \{1, \dots, m\} \setminus C} v_i (\Pi_i(n-1) - 1) + n_B \sum_{i=1}^m v_i (\Pi_{m+i}(n-1) - 1)\right) \right. \\
 &\quad \left. - \exp\left(n_P \sum_{i \in \{1, \dots, m\} \setminus C} v_i (\Pi_i(n) - 1) + n_B \sum_{i=1}^m v_i (\Pi_{m+i}(n) - 1)\right) \right), \tag{3.15}
 \end{aligned}$$

for $n \in \mathbb{N}$ and $\mathbb{P}(N = 0) = 1 - \exp\left(-n_P \sum_{j \in C} v_j\right)$. Since the population might go extinct without any resistant plant appearing, $\mathbb{P}(N = \infty) > 0$. Even if a resistant plant exists at some time in the population, it might still face extinction, $0 < \mathbb{P}(N = \infty) < \mathbb{P}(\text{'treatment success'})$.

In the following, we assume a diploid genetic architecture with one allele conferring herbicide resistance at a single biallelic locus (LAUENROTH et al., 2023). We consider the case of incomplete dominance of resistance, with homozygous resistant plants not affected by the herbicide and heterozygous plants being killed with an efficacy half as high as for sensitive homozygotes. We implement an incompletely dominant fitness cost c on seed production associated with resistance, such that heterozygotes suffer half the reduction in seed production capacity as plants carrying the resistance allele homozygously.

Computer simulations

We simulated our branching process model to observe the dynamics of reproducing plants and the seed bank in a Johnsongrass population (implemented in MATLAB version 9.14.0 (R2023a)). Initial populations of plants and seeds were based on independent Poisson distributions, using the product of expected gene frequencies in untreated populations v_i and the number of plants n_P or seeds n_B as means. We estimated the standing variation for target-site resistance $\mathbf{v} = (v_1, v_2, \dots, v_m)$ using a deterministic density-dependent model (LAUENROTH et al., 2023), simulating over 1000 years without management to achieve mutation-selection balance (Figure B.1 a). The plant and seed offspring of type i plants followed independent Poisson distributions with means $\hat{\lambda}_{ij}$ and $\hat{\lambda}_{i m+j}$, respectively, allowing for cross-pollination between all possible genotypes. Simulations included density-dependent reproduction effects, modifying fecundity based on high population density as per Eqs. (11) and (12) in LAUENROTH et al. (2023). The genotype i seeds had multinomially distributed plant and seed offspring of the same genotype – seeds germinating and remaining dormant. The process was deemed extinct if no plants or seeds remained, and treatment was considered failed once resistant plants were present (yielding a weed extinction probability of less than 10^{-65} under the standard parameter set in Table B.1). We recorded the proportions of extinctions and treatment failures, extinction times and time of appearance of the first resistant plant. The code is available on [GitHub](#). We provide separate folders for generating all figures in the manuscript with subfolders *Simulations* containing the Matlab files `Dynamics.m` that generates the population dynamics where relevant.

Results

The survival of a weed population under herbicide treatment is tied to the establishment of resistant plants on the field. For our standard parameter set given in Table B.1, lineages of sensitive plants and seeds face extinction with a probability close to one. With at most one successor in the next season, heterozygous and homozygous resistant seeds also have high extinction probabilities of around 0.88 and 0.75. Plants tracked by our model are the successful plants that reproduce. The high level of

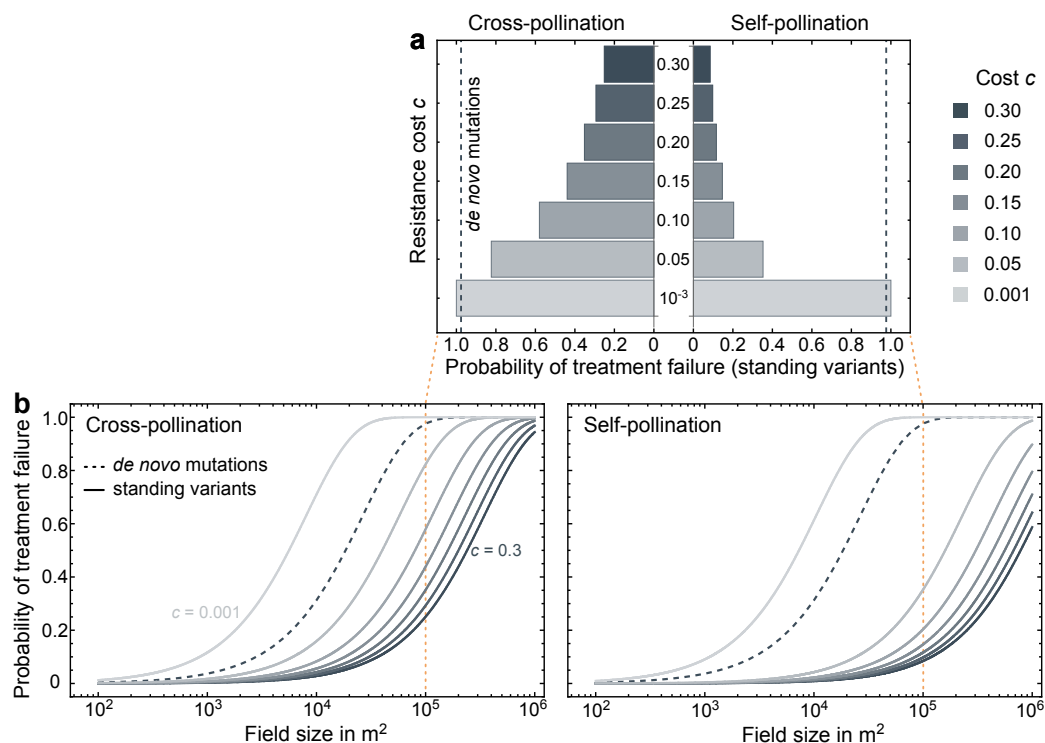


Figure 3.2 | Importance of *de novo* mutations and standing genetic variation for herbicide resistance adaptation depending on the associated resistance cost and self-pollination. We show the probabilities of treatment failure through standing genetic variants derived from Equation (3.11) under fitness costs on seed production between 0.1 and 30 %. Darker shades indicate higher costs of resistance. The dashed lines show the probability of treatment failure due to *de novo* mutations obtained from Equation (3.10). The left side reflects a fully cross-pollinating population, and the right represents a self-pollinated population. **a**, Probabilities of treatment failure in a field of 10⁵ square metres. **b**, Treatment failure probabilities as a function of field size. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

resistance endowed by the target-site mutation and the high fecundity of Johnsongrass cause the extinction probabilities of lineages started by a resistant plant to be close to zero, with $8.6 \cdot 10^{-66}$ for heterozygotes and $1.3 \cdot 10^{-109}$ for homozygotes. Hence, once a resistant plant appears on the field and survives until reproduction, its lineage is extremely unlikely to go extinct (Figure B.3 a).

Source of adaptive variation

Considering self-pollination. Herbicide resistance in weeds can arise from standing genetic variation and *de novo* mutations, determining the probability and rate of adaptation. The source of adaptive variation is thus relevant for the success of management strategies. We define the establishment of resistant plants under herbicide leading to population regrowth as treatment failure, corresponding to the evolutionary rescue of a population through successful mutants (BELL, 2017; GOMULKIEWICZ et al., 1995; KREINER et al., 2017; NEVE et al., 2014). By calculating probabilities of treatment failure through standing variants (Eq. (3.11)) or *de novo* mutations (Eq. (3.10)), we assess their impact on adaptation. A fitness cost c associated with resistance considerably lowers the probability of adaptation from standing variants by decreasing resistant plants' relative fitness in the absence of herbicides, lowering their abundance (Figure 3.2 a) (see LAUENROTH et al. (2023) for more details).

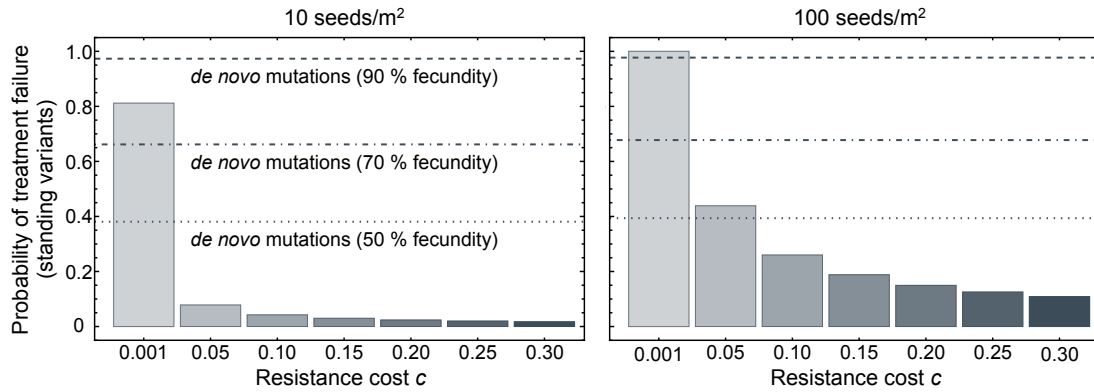


Figure 3.3 | Role of seed bank density and fecundity for the relevance of *de novo* mutations and standing genetic variation in herbicide resistance evolution. We show the probabilities of treatment failure through standing genetic variants derived from Equation (3.11) under fitness costs on seed production between 0.1 and 30 %. Darker shades indicate higher costs of resistance. The lines show the probability of treatment failure due to *de novo* mutations obtained from Equation (3.10) for 90 % of maximum fecundity (dashed), 70 % of maximum fecundity (dashed-dotted) and 50 % of maximum fecundity (dotted). The left panel corresponds to an initial seed bank density of 10 seeds per square metre, and the right panel reflects 100 seeds per square metre. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

The probability of treatment failure, influenced by field size, follows a sigmoidal curve, with larger fields or higher initial densities (effectively larger population sizes) increasing treatment failure probabilities (Figure 3.2 b). Self-pollination reduces adaptation from standing genetic variation caused by the increased homozygosity, decreasing the frequency of resistant individuals. However, the higher number of resistant homozygotes in the standing variation of self-pollinated weeds is advantageous if the mutation is highly recessive. In contrast, we expect the same number of *de novo* mutations regardless of the type of pollination (see Appendix B and Figure B.4 for more detail), causing treatment failure with similar probabilities if heterozygotes have high fitness. If heterozygous plants can not restore positive population growth, selfing again facilitates adaptation by efficiently generating resistant homozygotes. For self-pollinating species like Johnsongrass, considerable resistance costs make *de novo* mutations a likely source of target-site resistance.

Considering seed bank density and fecundity. The seed bank acts as a reservoir for genetic variation, with a larger seed bank increasing the chances of resistant plant emergence and treatment failure (Figure 3.3). The probability of new resistant mutations is tied to plant fecundity; thus, any decrease in seed production substantially lowers the probability of treatment failure through *de novo* mutations (lines in Figure 3.3). Besides affecting the overall probability of control failure, the initial seed bank size and fecundity considerably impact the relative importance of standing variation and *de novo* mutations as a source of adaptation, highlighting the necessity of considering the weeds ecology and infestation history in the design of resistance management.

Probability and time of control failure

Considering asexual propagation. The control of rhizomes is only partial as the herbicide active ingredients are not fully translocated into the rhizomes (BEASLEY, 1970; TUESCA et al., 1999). Potential resprouting from surviving rhizomes reduces the herbicide efficacy on rhizome shoots compared to seedlings (VIDRINE, 1989). We varied the weed's investment in sexual and asexual reproduction to analyse vegetative proliferation as a strategy to overcome control. Specifically, we fixed the total expected number of direct plant offspring one plant has in the following seasons

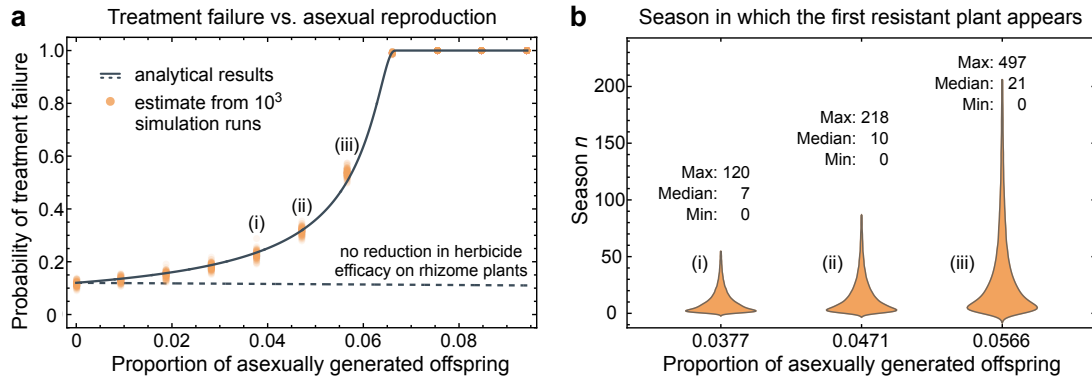


Figure 3.4 | Impact of asexual propagation on herbicide resistance adaptation. We show analytical results and simulation estimates for varying proportions of asexually generated offspring under a fixed expectation of plant offspring one plant generates without control measures.

a, Variation of the treatment failure probability with the proportion of asexually generated offspring. The grey lines show the probability of treatment failure derived analytically from Equation (3.9); as a solid line under reduced herbicide efficacy on rhizome shoots (see Table B.1) and as a dashed line for the herbicide efficacy on rhizome shoots equal to the efficacy on seedlings ($h_T = h_L$). The closed orange circles are estimates from 10³ simulation runs with 10² replicates each. **b**, Distribution of time until the appearance of the first resistant plant for different proportions of asexually generated offspring. The violin plots show smoothed probability density estimates of the season where the first resistant plant is established on the field. The density estimates were generated from 10⁵ simulation runs for the proportions of asexually generated offspring marked in (a). The maximum, median and minimum are displayed on the violin plots. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

without control measures and varied the proportion of asexually generated offspring, implicitly assuming that surviving sexual and vegetative offspring are equally costly. Though the mutational influx is less, since we do not consider mutations in vegetative growth, plants that invest more in asexual reproduction are more likely to escape control (Figure 3.4 a). Vegetative proliferation potentially improves population survival under herbicide treatment if standing variation for target-site resistance exists since the offspring of herbicide-resistant individuals are themselves resistant. Not all sexually generated offspring will be resistant, especially for heterozygous mother plants or due to cross-pollination in predominantly susceptible populations. However, clonal reproduction per se is not beneficial here (dashed line in Figure 3.4 a), likely due to the omission of somatic mutations and the high fitness of heterozygotes. Nevertheless, rhizomes as an organ of vegetative reproduction increase the persistence as a result of reduced control by the herbicide, buying the weed population time to generate a successful mutant (Figure 3.4 b). Thus, we expect control failure by herbicides to be likelier and to occur later in weeds investing more in asexual propagation (Figure 3.4).

Considering the seed bank. A seed bank stores genetic information from several past generations, preserving adaptation to previous herbicide applications. Further, seed banks buffer changes in population size induced by the herbicide. How long seeds stay in the seed bank and how many of them ultimately germinate is determined by the germination probability g and the seed mortality d_Z . High germination leads to a fast turnover – weak seed bank – and low germination causes a slow turnover – strong seed bank. To analyse the effect of seed bank strength, we varied the germination probability of weed seeds and the seed bank mortality while maintaining the total number of sexually generated offspring per plant constant. We simulated an earlier treatment with

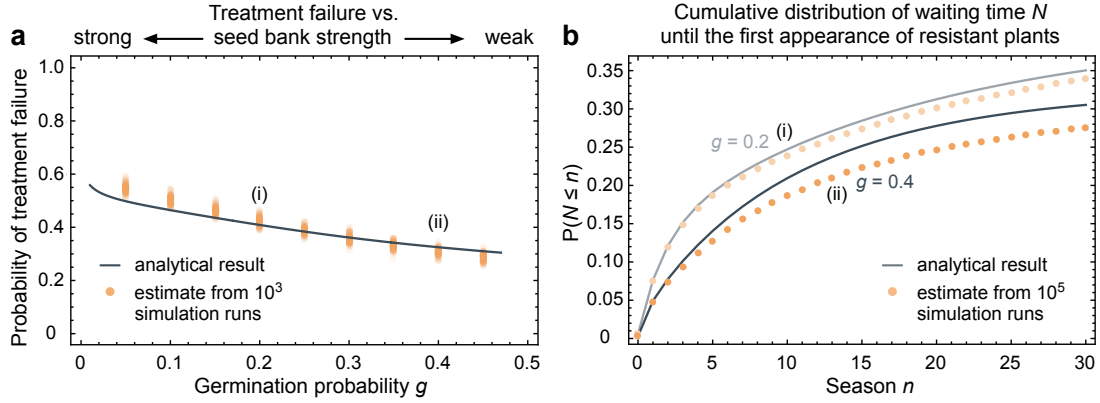


Figure 3.5 | Impact of the seed bank on herbicide resistance adaptation. We show the germination probability's effect, determining the seed bank's strength, on control failure. The seed bank mortality was adapted to maintain the total expected number of sexually generated offspring of a plant constant. We simulated a previous herbicide treatment followed by ten years without any control measures and used the result as the start population composition. **a**, Variation of the treatment failure probability with the germination probability. The grey line shows the probability of treatment failure derived analytically from Equation (3.9). The closed orange circles are estimates from 10^3 simulation runs with 10^2 replicates each. **b**, Distribution of waiting time until the first resistant plant appears in weed populations with varyingly strong seed banks. The cumulative waiting time distribution was derived analytically from (3.15), shown as grey lines. The closed orange circles are estimates from 10^5 simulation runs. The dark grey line and closed orange circles show the cumulative distribution of waiting time under a weaker seed bank with a germination probability of $g = 0.4$ (ii), and the light grey line and closed light orange circles correspond to the waiting in populations that have a stronger seed bank with a germination probability of $g = 0.2$ (i). The corresponding treatment failure probabilities in (a) are marked accordingly. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

the herbicide followed by ten seasons without control measures, yielding the composition of the start population (Figure B.1 b). Populations with a stronger seed bank have a higher probability of escaping from following herbicide applications (Figure 3.5 a) with higher probabilities of resistant plants establishing early on (Figure 3.5 b) since the change in allele frequencies induced by the previous application is preserved in the seed bank. Weaker seed banks with higher germination, in turn, slow the population decline after treatment onset, enabling the later appearance of herbicide-resistant plants due to *de novo* mutations (Figure 3.5 b). After several seasons of herbicide application, the weaker seed bank is depleted, and resistant plants are again slightly more likely to emerge in populations with seeds still preserved in a strong seed bank.

Considering self-pollination. Self-pollination increases the level of homozygosity in a population and reduces target-site resistance adaptation from standing variation (as in Subsection Source of adaptive variation). In the early seasons of treatment, resistant plants are more likely to appear in cross-pollinating than in self-pollinating populations (Figure 3.6 a and Figure B.2 a). This is caused by the reduced frequency of resistant individuals in the standing variation of self-pollinating weed populations. Resistance alleles cluster in homozygotes, decreasing the number of individuals resistant towards the herbicide unless the mutation is highly recessive. A recessive resistance cost further decreases the number of offspring a homozygous resistant plant generates compared to a heterozygote, diminishing standing variation for target site resistance. In later seasons, where adaptation likely results from *de novo* mutations, no notable effect of the mating system exists on the waiting time distributions for resistant plants (Figure 3.6 a and Figure B.2 a). Under

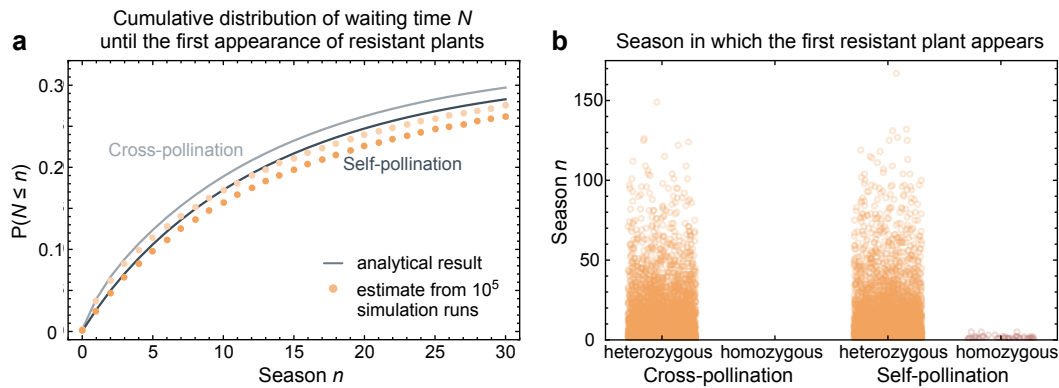


Figure 3.6 | Impact of self-pollination on the timing of resistance evolution. We show the time distribution until the establishment of resistant plants on the field under cross- and self-pollination. **a**, Distribution of waiting time until the first resistant plant appears. The cumulative waiting time distribution was derived analytically from (3.15), shown as grey lines. The closed orange circles are estimates from 10^5 simulation runs. The light grey line and closed light orange circles depict the cumulative waiting time distribution in a cross-pollinated population, and the line in dark grey and the closed orange circles correspond to the waiting time in a purely self-pollinated population. **b**, Time of the first appearance of a resistant plant. On the left, we show simulation results for purely cross-pollinating populations and on the right for weed populations that self-pollinate, with 10^4 replicates each. The open orange circles represent heterozygous resistant plants, and the open red circles show when the first resistant plant surviving on the field is homozygous. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

cross-pollination, it is unlikely that the first mutant plant to establish is homozygous, while for self-pollinating populations, there might be resistant homozygotes in the standing variation able to rescue the population (Figure 3.6 b). However, mutations arising *de novo* usually occur heterozygously. Resistant homozygotes generally only appear as the first resistant plant on a field in the first seasons of treatment; otherwise, they emerge as offspring of established heterozygotes.

Discussion

Various factors influence the primary source of adaptive variation. Using a haploid model focused on adaptation via a single beneficial allele, ORR et al. (2014) demonstrated that the probability of adaptation from *de novo* mutations versus standing variation is influenced by the frequency of the beneficial allele and mutation rate and also by the rate of population decline. Faster population declines enhance the role of standing variants, underscoring the potential significance of standing genetic variation for rapid adaptation following abrupt environmental shifts. HAWKINS et al. (2019) emphasise the role of the selection coefficient for the source of adaptation, arguing that, in the context of herbicide resistance, mechanisms conferring high levels of resistance, like target-site resistance, can emerge *de novo*. Genomic studies in blackgrass and waterhemp reveal different primary sources of target-site resistance in these two annual weeds (KERSTEN et al., 2023; KREINER et al., 2022). We find that fitness costs associated with resistance, the type of pollination, and the weed's fecundity and seed bank size are other key factors determining the relevance of *de novo* mutations and standing genetic variation for the evolution of herbicide resistance. Thus, we stress the necessity of ecological and evolutionary details in the assessment of strategies for resistance management. The high fecundity of Johnsongrass and the high level of resistance conferred by target-site mutation seem to fuel adaptation from *de novo* mutations. Simultaneously, resistance-related fitness costs and the prevalence of self-pollination reduce the frequency of pre-existing resistant variants.

The probability of a weed population successfully adapting to herbicide treatment is determined by the absolute number of the resistance allele, not its relative frequency. A larger seed bank, potentially containing more resistant seeds, boosts the probability of adaptation from pre-existing variation. The mutational influx increases with increasing fecundity of a weed, amplifying resistance evolution through *de novo* mutations. This accounts for the strong positive effect of seed production on the risk of resistance described by NEVE (2008). Resistance costs depleting the standing variation for target-site resistance reduce adaptation from pre-existing variation substantially (see LAUENROTH et al. (2023) for a detailed analysis of the standing variation for target-site resistance concerning the associated fitness cost). This result aligns with the theoretical predictions of NEVE (2008) for the total herbicide resistance probability. However, not all mutations endowing target-site resistance are known to involve a fitness cost (MENCHARI et al., 2008). Our finding that self-pollination diminishes the adaptation of target-site resistance from standing genetic variation in a perennial weed aligns with predictions from a fundamental single-locus model by GLÉMIN et al. (2013). However, the substantial contribution of new mutations might lead to similar probabilities of treatment failure for different pollination types. NEVE (2008) likewise predict little difference in the probability of herbicide resistance in cross- and self-pollinated populations when resistance is incompletely dominant, as in our study. Despite this, self-pollinating populations, especially where heterozygotes have low fitness, may have a higher chance of mutant allele establishment (UECKER, 2017). This suggests that under certain conditions, the effective generation of mutant homozygotes in selfing populations, increasing the efficacy of selection, could enhance the likelihood of successful adaptation. Apart from the pollination type, the reproduction type is another detail of a weed's reproductive system that influences adaptation.

Considering a quantitative trait with gametic and somatic mutations, ORIVE et al. (2017) demonstrated that clonal reproduction increases population survival after a sudden environmental shift if the standing variation allows for persistence. However, for a slightly beneficial allele with unfit heterozygotes, UECKER (2017) showed that whether asexual reproduction is advantageous depends on its dominance. For recessive target-site mutations, sexual reproduction generating fit homozygotes is vital. Our model captures mutations occurring during sexual reproduction but not somatic mutations. Genome-wide mutation rates (per nucleotide per generation) observed in a vegetatively propagated grass species are similar to those in germ cells (CAETANO-ANOLLÉS, 1999). Thus, our model underestimates the number of *de novo* resistance mutations that arise and, with that, the probability of control failure, especially in populations with high proportions of asexual reproduction. Together with the incomplete dominance of resistance, this might be why we find no direct positive effect of clonal reproduction based on the resistance heredity. Rhizomatous Johnsongrass is, however, less susceptible towards herbicides (BEASLEY, 1970; TUESCA et al., 1999; VIDRINE, 1989), increasing persistence and buying, similar to a seed bank, time for the weed population to adapt. The improved persistence under herbicide treatment is, besides the resistance heredity, a second mechanism by which vegetative propagation functions as a strategy to overcome control in weeds.

Seed banks preserve genetic information of forgone selection for resistance and prolong the period in which a weed can adapt towards a herbicide, emphasising the complications seed banks impose on managing herbicide resistance. A previous study showed that a seed bank substantially delays the extinction of a herbicide-treated weed population and increases the risk of resistance (LAUENROTH et al., 2023). Further, a seed bank buffers the genetic effects of selection, slowing resistance evolution if it occurs. Under a constant seed bank mortality, an increase in the germination probability of seeds increases the risk of resistant types in the seed bank establishing as resistant plants on the field (NEVE, 2008). We compensated the increase in germination by the seed bank mortality so that it did not result in a rise in germinated seeds and simulated a forgone treatment with the herbicide. Under these conditions, lower germination helps preserve resistant types in the seed bank, allowing the weed population to adapt readily to subsequent herbicide applications and increasing the risk

of treatment failure.

Our model ignores density dependence or resource competition between the plants. This simplifying assumption is reasonable for low initial plant densities but might systematically overestimate treatment failure probabilities if start densities are high (Figure B.3). Agronomically, manage a weed infestation before densities become high and a substantial seed bank builds up in the soil. In general, population density affects the growth of a mutant lineage, determining its establishment probability (WILSON et al., 2017). However, the herbicide drastically reduces the wild-type weed density from the first application onwards, and competition from the surviving susceptible plants against resistant mutants is neglectable. Precisely, the majority of weeds sensitive towards the herbicide are killed by its application when still in an early growth stage (DOGAN et al., 2005; JOHNSON et al., 2014), such that the population gets reduced before competition from maturing susceptible plants becomes substantial (SCHWINNING et al., 2017; WILLIAMS et al., 1977). Further, branching processes that survive grow to infinity. Though biologically unrealistic, this is no limitation to the results since resistant subpopulations that reached a sufficient size ensure population survival almost certainly (HARRIS, 1963, pp. 10-11). Therefore, competition between plants of one or more resistant lineages does not influence the results. Density effects in seed production reduce the generation of mutants in the first seasons of herbicide treatment, lowering the probability of resistant plants first appearing in these seasons slightly compared to analytical results from Equation (3.14) (Figure B.2). Estimates from simulations implementing density-dependent reproduction match well with the probability of treatment failure derived analytically from Equation (3.9) for an initial weed density of one plant per square metre (Figure 3.4 a and Figure 3.5 a). However, for higher weed densities in season 0, the analytical results systematically deviate from the density-dependent simulations, overestimating the probability of treatment failure (Figure B.3 b). Since, in our model, the start population is untreated, population density affects the number of offspring generated by these initial plants, determining the rescue probability.

Most mathematical models of resistance evolution implement a single biallelic locus in a haploid or diploid population (but see SANTER et al. (2020) HOLMES et al. (2022) and GAROÑA et al. (2023)). The results presented in this manuscript are for a single locus in a diploid genome. Though we did not show it here, our flexible model can explicitly capture several loci with epistatic interactions and any ploidy level. The higher number of genotypes results in a larger type space of the branching process, increasing the computational complexity. A broad mutational target size increases the probability of treatment failure. ORR et al. (2008) observed an effect similar to an increase in population size when there are no epistatic interactions. Furthermore, our model can describe quantitative genetic scenarios often observed for non-target-site resistance, though the type space will become rather large. It would be interesting to study the differences between monogenic and polygenic resistance mechanisms and the factors shaping their role in the evolution of herbicide resistance.

Mathematical models of evolutionary rescue, e.g. ORR et al. (2008), ORR et al. (2014), and TOMASINI et al. (2020), commonly use HALDANE's (1927) classic approximation of the probability that a unique beneficial mutation escapes stochastic loss in order to obtain analytical results. It assumes the mutant's absolute fitness to be greater than but close to one. The evolution of resistances in herbicide-treated weed populations is an example of rapid adaptation under strong selection (KREINER et al., 2017). Target-site resistance typically endows a high fitness, not captured by HALDANE's (1927) approximation. Our approach allows for mutants with absolute fitness considerably larger than one, with the drawback that we do not obtain analytical solutions for all considered probabilities. The probabilities calculated in Paragraph [Waiting times](#) are analytically exact results, while the extinction and rescue probabilities presented in Paragraph [Probability of treatment failure](#) are numerical results. The m -dimensional equation system (3.7) is solved numerically, using Newton methods, to derive the type-specific extinction probabilities \mathbf{q} ([FindRoot](#) in Mathematica version 13.1).

We demonstrated how concepts of rescue theory help to understand the factors determining the probability and timing of treatment failure resulting from the establishment of herbicide-resistant weeds. The methods can be employed to identify weedy traits that favour adaptation from *de novo* mutations or standing genetic variation. Sustainable weed management strategies comprise diverse control measures, including crop and herbicide rotations. Studying the evolution of resistance and risk of treatment failure in these fluctuating environments will be of added practical relevance. In addition to new mutations or pre-existing variation, herbicide resistance might also result from gene flow from resistant neighbouring fields. As herbicide susceptibility is a shared resource among farmers, a metapopulation model allowing for pollen, seed and rhizome migration between fields could be an exciting extension of our approach.

Data Availability. All data and plots are available on [GitHub](#).

Code Availability. All code is available on [GitHub](#).

Acknowledgements. We thank Hildegard Uecker, Gaurav Athreya and Demetris Taliadoros, and the research group Theoretical Models in Eco-evolutionary Dynamics for their valuable comments on the manuscript and the research group Theoretical Models in Eco-evolutionary Dynamics for helpful discussions. This work is dedicated to the memory of Karsten Keller, an exceptional teacher and mentor known for his kindness and wholehearted support for his students. Although Karsten could no longer contribute to this work directly, his lectures and guidance over the years built its foundation.

Author Contributions Statement. Both authors conceived the study, developed the model, analysed the results and wrote the manuscript. D.L. formulated the mathematical model, the algorithm and ran the simulations.

Competing Interests Statement. The authors declare no competing interests.

CHAPTER 4

Summary and Outlook

Summary

First predicted in the 1950s, herbicide resistance now threatens the sustainability of global food production (BAUCOM, 2019; HEAP, 2024). The development of management strategies mitigating resistance evolution must be informed by a comprehensive understanding of the eco-evolutionary processes driving herbicide resistance adaptation in weeds (NEVE et al., 2014). At the same time, herbicide-resistant weeds pose uniquely suited models for studying rapid evolution in the face of human-mediated sudden environmental change (BAUCOM, 2019; NEVE et al., 2014). I emphasise that mathematical modelling, integrating knowledge, concepts, and hypotheses from different disciplines, can facilitate the conjunction of ecology, evolution, and weed science. In the following, I highlight how the approaches developed in this thesis contribute valuable insights into the ecological and evolutionary drivers of herbicide resistance adaptation and address fundamental questions concerning adaptation to anthropogenic regimes of strong selection. I further discuss limitations and possible extensions of this work and end with some concluding remarks.

Life cycle model. Weed population dynamic models commonly implement the weed life cycle at varying levels of detail and temporal resolution (EVANS et al., 2018; HOLMES et al., 2022; HOLST et al., 2007; LIU et al., 2019). These models have been used to study weed shifts in response to weed management and farming regimes and are an integral component of models of herbicide resistance evolution. However, the vast majority of studies deal with annual species (HOLST et al., 2007, but see LIU et al., 2019; HOLMES et al., 2022). The life cycle model presented in [Chapter 2](#) captures the demography of seed- and rhizome-propagated perennials with a persistent seed bank, inheritance of target-site resistance, and weed management strategies. A set of discrete-time equations, each corresponding to the included life stages, describes the dynamics deterministically, accounting for consequences of intraspecific competition on two levels – survival and reproduction. Manipulation of genetic details and life history traits unveils their impact on the rate of herbicide resistance evolution, stressing the importance of these characteristics in the assessment. A matrix approximation yields the mutation-selection balance in untreated populations, indicating that the associated fitness cost modulates the frequency of standing variants for target-site resistance. The model further allows in silico comparison of weed and resistance control provided by different herbicide regimes, revealing that mixtures are superior to rotations as they induce higher heterogeneity in selection pressure over timescales critical for herbicide resistance evolution.

Mathematical formulations of population dynamic models generally do not account for the stochastic nature of herbicide resistance evolution and weed eradication. The initial decline of the sensitive weed population in response to herbicide treatment is well reflected by the deterministic time-recursive equations. However, small populations are prone to stochastic extinction, yet the model populations decline to abundances below one but never actually reach zero. The model weed populations do not go extinct and can effectively regrow from numbers close to zero. New mutants appear by chance, and while at low frequencies, even target-site mutations providing a high fitness advantage in the presence of herbicide are subject to stochastic loss. The life cycle models implement mutations deterministically, causing the model populations to evolve resistance towards

the herbicide with certainty. The population dynamical model in [Chapter 2](#) is complemented by stochastic simulations yielding estimates of the probability for evolved herbicide resistance and weed population regrowth. Simulation models can reflect the stochastic nature of mutations and also demographic stochasticity and environmental fluctuations but lack analysability (BAGAVATHIANNAN et al., 2020; EVANS et al., 2018; HOLMES et al., 2022; LIU et al., 2019).

Stochastic processes like branching processes account for the stochastic nature of herbicide resistance evolution and weed population extinction and provide a rich body of mathematical theory for their analysis (ATHREYA et al., 1972; HARRIS, 1963). Branching processes have been used to model populations at risk of extinction due to low reproduction rates after an environmental change, investigating how the emergence of an adapted mutant could prevent population extinction via evolutionary rescue (AZEVEDO et al., 2021; ORR et al., 2008; UECKER et al., 2014). While processes with a single type are used to derive the probability of this phenomenon, multitype branching processes can yield further information about the waiting time until such a mutant appears (ALEXANDER, 2016; AZEVEDO et al., 2021; SERRA et al., 2007). The value of concepts from evolutionary rescue theory for understanding population genetics of herbicide resistance evolution has been recognised (KREINER et al., 2017; NEVE et al., 2014); nevertheless, to the best of my knowledge, no study has developed a relevant model before.

Branching process model. [Chapter 2](#) focussed on weed population dynamics, the outcomes of management regimes and how specific weed characteristics impact these outcomes. [Chapter 3](#) is concerned with the relative importance of *de novo* mutations and standing variation in herbicide resistance evolution and its timing. The presented multitype Galton-Watson process model captures the appearance and spread of target-site mutations, conferring resistance to the herbicide, in a primarily sensitive weed population declining in response to the treatment. Concepts from rescue and branching process theory help to understand how seed bank dynamics and details of the reproductive system modulate the probability and timing of treatment failure resulting from the establishment of herbicide-resistant weeds. The efficacy of management strategies for herbicide resistance is tied to the primary source of adaptive variation. Analysis of the evolutionary pathways in isolation identifies weedy traits and genetic factors that favour adaptation from *de novo* mutations or standing genetic variation, explaining the variation observed in different weed species.

The branching process model accounts for the stochastic nature of resistance evolution at the cost of less realism concerning weed ecology. It neglects density effects and matings between resistant individuals and is thus inadequate for evaluating population and evolutionary dynamics beyond the establishment of resistant mutants and weed population rescue. Simulations show that the branching process model accurately captures the early phase of herbicide resistance adaptation, in which the resistance allele is still rare and weed density low, owing to the application of herbicides. A drawback of the mathematical framework developed in [Chapter 3](#) is that heterogeneous selection regimes posed by herbicide and crop rotations cannot be included straightforwardly; however, they can be readily investigated through corresponding simulations.

The two approaches introduced in this thesis are each uniquely designed for studying different aspects of herbicide resistance adaptation and management of a perennial weed species. These models do not aim to make realistic predictions but rather provide a mechanistic understanding of the processes that drive herbicide resistance evolution and the factors determining its rate, probability and primary source. Their simple design, with a good representation of the weed life cycle and the resistance mechanism, assures interpretability and improves communication. While presented in the context of Johnsongrass, the models can capture the population and evolutionary dynamics of herbicide resistance adaptation in other perennial weed species. More generally, the

approaches may be adapted to any species comprising sexual reproduction in the continuum of selfing and outcrossing, asexual propagation, and potentially a seed bank component. Especially the branching process model in [Chapter 3](#), describing the dynamics of rare beneficial alleles within large populations facing extinction due to a sudden environmental change, is also flexible to account for different genetic architectures.

Outlook

Spatial structure. The herbicide resistance models introduced in this thesis capture the dynamics of a spatially homogeneous and panmictic weed population infesting a single field. However, spatial structure affects the rates of resistance evolution and population growth. Resistance mutations might arise *de novo* in a population or be part of the standing genetic variation. Gene flow from resistant weed populations in neighbouring fields – via pollen, seed or vegetative propagule dispersal – facilitates the spread of resistance traits in agricultural landscapes, suggesting herbicide susceptibility is a shared resource among farmers (BECKIE et al., 2019; MAITY et al., 2022). A spatial simulation study concluded that best herbicide management practices reduce herbicide resistance evolution most effectively when aggregated at a landscape scale (EVANS et al., 2018). Field data from farms in Argentina showed a positive relation between field size and abundance of herbicide-resistant weeds; smaller fields had a lower density of resistant weeds (GARIBALDI et al., 2023). These observations align with the theoretical results for panmictic populations in [Chapter 3](#). Spatial structure within local weed populations and dispersal limitations influence evolutionary and population dynamics within individual fields. Weed distributions in agricultural fields are not uniform but exhibit local variations in density and resistance gene frequency. Limited pollen dispersal causes cross-pollination to occur primarily between neighbouring plants (EVANS et al., 2018). Seeds mainly end up close to the mother plant, although agricultural practice might disperse some seeds and rhizome pieces further (EVANS et al., 2018). In a herbicide-managed field, this localised dispersal results in spatial aggregation of herbicide-resistant weeds and, ultimately, a reduction in effective outcrossing and localised competition within the weed patches limiting reproduction (NEVE, 2008; SOMERVILLE et al., 2017). The patchy growth pattern of weeds slows population growth and resistance evolution (SOMERVILLE et al., 2017). An extension of the presented approaches to a metapopulation model that describes spatial structure at field and landscape scale – accounting for spatial aggregation and gene flow within and between weed populations – would provide a well-suited framework for studying the impact of different management strategies and their aggregation at the landscape scale more realistically.

Integrated weed management. Weed management systems relying solely on herbicides are not sustainable and will ultimately fail due to resistance development (PEROTTI et al., 2020). The sustainability of cropping systems and herbicides can only be achieved by the diverse integration of non-herbicide practices for weed management, such as crop rotations including competitive cultivars or cover crops, higher seeding rates of the crop intensifying competition and mechanical weed control measures (PEROTTI et al., 2020; SHANER et al., 2014). Diversified weed management strategies aim at minimising the survival and reproduction of herbicide-resistant weeds, mitigating the evolution of resistance and limiting its spread. Ideally, future weed and herbicide resistance modelling approaches should consider integrated weed management practices as they have proven to slow the evolution of herbicide resistance.

Human behaviour and cooperation. Herbicide sensitivity is a common resource shared among farmers and societies. The number of known herbicide modes of action is limited, and the reported cases of resistance are ever-increasing, while new herbicides are missing (HEAP, 2024; QU et al., 2021; SELBY et al., 2023). Agricultural crop production relies almost exclusively on herbicides as

an easy and cost-effective solution to weed management; if rendered ineffective owing to herbicide resistance, global food production will suffer severe cascading consequences (PEROTTI et al., 2020; POWLES et al., 2010). Despite clear evidence for its capability to slow herbicide resistance adaptation – from theoretical models and field data – and awareness among farmers, true integrated weed management has been poorly adopted (MOSS, 2019; SHANER et al., 2014). The architecture of agricultural landscapes controls the spread of resistance traits, with smaller crop fields with more edges and an increased number of natural habitats functioning as a barrier to the spread of herbicide-resistant weeds (GARIBALDI et al., 2023). Nevertheless, in practice, we often perceive an opposing development (RAMANKUTTY et al., 2018). Further, herbicide management strategies are less effective in mitigating resistance evolution when implemented at the farm scale than best practices aggregated at a landscape scale, necessitating cooperation and coordination between farms (EVANS et al., 2018). Overall, an improved understanding of the human component in weed management would benefit the resolution of this multifaceted issue of preserving herbicide efficacy and achieving sustainability of weed management. Game theoretical approaches might be used to model farmers' behaviour and identify factors promoting cooperation, similar to approaches aiming at limiting antibiotic resistance evolution in medicine (PORCO et al., 2012; COLMAN et al., 2019; and see MORISON et al., 2024 for a comprehensive review on evolutionary game theory in disease evolution and spread).

Economics of weed management. An assessment of the economic consequences needs to forego the implementation of novel weed control measures or management systems. Where field studies are limited in time, models integrating weed population dynamics with economic evaluation can yield long-term predictions (FRECKLETON et al., 2009). Linking the weed demographic model from Chapter 2 with predictions of yield loss based on weed density and the costs of applying control measures will provide a holistic framework for evaluating the economic costs and benefits of different weed and resistance management options. Maximising the present value of a farmer's profit, discounted at a rate relevant to agricultural time scales, will help identify the best practice over long timescales. This research endeavour has started in collaboration with Martin Quaas and Hanna Schenk from the iDiv in Leipzig.

Conclusions

The ultimate aim of weed population dynamical modelling is to inform the development and selection of sustainable weed management regimes. Models promote a comprehensive understanding of the eco-evolutionary processes underlying the dynamics of herbicide resistance adaptation in weeds (BAGAVATHIANNAN et al., 2020; RENTON et al., 2014). Where field experiments face limitations, modelling approaches allow the comparison of weed management outcomes at large time and spatial scales (FRECKLETON et al., 2009; HOLST et al., 2007). Models can never capture these agricultural systems in all their complexity and should not attempt to, but rather be understood as tools yielding qualitative, not quantitative, predictions (BAGAVATHIANNAN et al., 2020). We designed the models in this thesis to capture all necessary detail yet strive for simplicity and accessibility. In this way, our approaches allow the identification of pivotal factors influencing the evolution of herbicide resistance and general conclusions about the potential of management strategies for mitigating resistance. The inherent simplicity and flexibility further facilitate communication and adaptation across disciplines.

While pesticides were once perceived as a “silver bullet”, their efficacy is nowadays threatened by widespread resistance (HAWKINS et al., 2019; LANDAU et al., 2023). We know that, by means of evolution, nature will sooner or later overcome any strategy we implement for managing harmful organisms (e.g. crop mimicry in weeds (BARRETT, 1983; COLEMAN et al., 2023)). The ubiquity of resistance in agriculture and medicine and the ever-increasing threat posed, increased the awareness

of human-mediated selection regimes rapidly altering the environment (PALUMBI, 2001). However, a comprehensive understanding of the ecological, evolutionary and anthropogenic drivers of resistance evolution can inform the development of mitigation strategies. Modelling studies integrating our current knowledge and hypothesis provide a means to grasp the complex nature of eco-evolutionary dynamics of resistance adaptation of pests and to reveal directions for further research. Moreover, pesticide resistance is a highly repeated example of rapid evolution in action, posing a unique model for studying fundamental questions concerning adaptation to human-mediated regimes of strong selection, sources of adaptive variation and evolutionary rescue (HAWKINS et al., 2019). I hope to have demonstrated this mutual benefit provided by pesticide resistance modelling here, assessing herbicide resistance evolution in a perennial weed – *Sorghum halepense*.

Bibliography

- ACCIARESI, HORACIO A. and HUGO O. CHIDICHIMO (2005): ‘Ecophysiological response of Sorghum halepense populations to reduced rates of nicosulfuron’. *Pesquisa Agropecuária Brasileira*, vol. 40(6): pp. 541–547 (cit. on p. 62).
- ACHARYA, JAYADEV, ASHKAN JAFARPOUR, ALON ORLITSKY, and ANANDA THEERTHA SURESH (2014): ‘Poissonization and Universal Compression of Envelope Classes’. *IEEE International Symposium on Information Theory*: pp. 1872–1876 (cit. on pp. 31, 33, 34, 75).
- ALEXANDER, HELEN K. (2016): ‘Conditional Distributions and Waiting Times in Multitype Branching Processes’. *Advances in Applied Probability*, vol. 45(3): pp. 692–718 (cit. on pp. 7, 30, 35, 46).
- ALEXANDER, HELEN K., GUILLAUME MARTIN, OLIVER Y. MARTIN, and SEBASTIAN BONHOEFER (2014): ‘Evolutionary rescue: linking theory for conservation and medicine’. *Evolutionary Applications*, vol. 7(10): pp. 1161–1179 (cit. on p. 6).
- ANDERSON, L.E., A.P. APPLEBY, and J.W. WESELOH (1960): ‘Characteristics of Johnsongrass Rhizomes’. *Weeds*, vol. 8(3): p. 402 (cit. on p. 62).
- ATHREYA, KRISHNA B. and PETER E. NEY (1972): *Branching Processes*. Berlin: Springer (cit. on pp. 7, 30, 46).
- AZEVEDO, RICARDO B.R. and PETER OLOFSSON (2021): ‘A branching process model of evolutionary rescue’. *Mathematical Biosciences*: p. 108708 (cit. on pp. 7, 30, 46, 77).
- BAGAVATHIANNAN, MUTHUKUMAR V., HUGH J. BECKIE, GUILLERMO R. CHANTRE, JOSE L. GONZALEZ-ANDUJAR, RAMON G. LEON, PAUL NEVE, SANTIAGO L. POGGIO, BRIAN J. SCHUTTE, GAYLE J. SOMERVILLE, RODRIGO WERLE, and RENE VAN ACKER (2020): ‘Simulation Models on the Ecology and Management of Arable Weeds: Structure, Quantitative Insights, and Applications’. *Agronomy*, vol. 10(10): p. 1611 (cit. on pp. 3, 46, 48).
- BAGAVATHIANNAN, MUTHUKUMAR V. and JASON K. NORSWORTHY (2013): ‘Postdispersal Loss of Important Arable Weed Seeds in the Midsouthern United States’. *Weed Science*, vol. 61(4): pp. 570–579 (cit. on pp. 11, 27, 62, 64).
- BARGUÉS-RIBERA, MARIA and CHAITANYA S. GOKHALE (2020): ‘Eco-evolutionary agriculture: Host-pathogen dynamics in crop rotations’. *PLoS Computational Biology*, vol. 16(1): e1007546 (cit. on p. 23).
- BARRETT, ROWAN D.H. and DOLPH SCHLUTER (2008): ‘Adaptation from standing genetic variation’. *Trends in Ecology & Evolution*, vol. 23(1): pp. 38–44 (cit. on pp. 6, 29).
- BARRETT, SPENCER H. (1983): ‘Crop mimicry in weeds’. *Economic Botany*, vol. 37(3): pp. 255–282 (cit. on p. 48).
- BAUCOM, REGINA S. (2016): ‘The remarkable repeated evolution of herbicide resistance’. *American Journal of Botany*: pp. 181–3 (cit. on pp. 2, 3, 29).
- (2019): ‘Evolutionary and ecological insights from herbicide-resistant weeds: what have we learned about plant adaptation, and what is left to uncover?’ *New Phytologist*, vol. 223(1): pp. 68–82 (cit. on pp. 2, 3, 13, 45).
- BEASLEY, C.A. (1970): ‘Development of Axillary Buds from Johnsongrass Rhizomes’. *Weed Science*, vol. 18(2): pp. 218–222 (cit. on pp. 11, 13, 24, 38, 42, 63, 65).

- BECKIE, HUGH J., ROBERTO BUSI, MUTHUKUMAR V. BAGAVATHIANNAN, and SARA L. MARTIN (2019): 'Herbicide resistance gene flow in weeds: Under-estimated and under-appreciated'. *Agriculture, Ecosystems & Environment*: p. 106566 (cit. on pp. 2, 23, 47).
- BELL, GRAHAM (2017): 'Evolutionary Rescue'. *Annual Review of Ecology, Evolution and Systematics*: pp. 605–627 (cit. on pp. 6, 37).
- BENVENUTI, STEFANO, MASSIMO SELVI, SARA MERCATI, GIANLUCA CARDINALI, VALENTINO MERCATI, and MARCO MAZZONCINI (2021): 'Stale seedbed preparation for sustainable weed seed bank management in organic cropping systems'. *Scientia Horticulturae*: p. 110453 (cit. on p. 30).
- BESCANSA, PALOMA, MARÍA J. IMAZ, IÑIGO VIRTO, ALBERTO ENRIQUE, and WILLEM B. HOOGMOED (2006): 'Soil water retention as affected by tillage and residue management in semiarid Spain'. *Soil and Tillage Research*, vol. 87(1): pp. 19–27 (cit. on p. 22).
- BLISS, C.I. (1939): 'The Toxicity of Poisons Applied Jointly'. *Annals of Applied Biology*, vol. 26(3): pp. 585–615 (cit. on p. 13).
- BUSI, ROBERTO, STEPHEN B. POWLES, HUGH J. BECKIE, and MICHAEL RENTON (2020): 'Rotations and mixtures of soil-applied herbicides delay resistance'. *Pest Management Science*, vol. 76(2): pp. 487–496 (cit. on pp. 10, 23).
- CAETANO-ANOLLÉS, GUSTAVO (1999): 'High genome-wide mutation rates in vegetatively propagated bermudagrass'. *Molecular Ecology*, vol. 8(7): pp. 1211–1221 (cit. on p. 42).
- CARLSON, STEPHANIE M., CURRY J. CUNNINGHAM, and PETER A.H. WESTLEY (2014): 'Evolutionary rescue in a changing world'. *Trends in Ecology and Evolution*, vol. 29(9): pp. 521–530 (cit. on p. 6).
- CARVALHO, FERNANDO P. (2017): 'Pesticides, environment, and food safety'. *Food and Energy Security*, vol. 6(2): pp. 48–60 (cit. on p. 1).
- COLEMAN, GUY R.Y., ASHER BENDER, MICHAEL J. WALSH, and PAUL NEVE (2023): 'Image-based weed recognition and control: Can it select for crop mimicry?' *Weed Research*, vol. 63(2): pp. 77–82 (cit. on p. 48).
- COLMAN, ANDREW M., EVA M. KROCKOW, EDMUND CHATTOE-BROWN, and CAROLYN TARRANT (2019): 'Medical prescribing and antibiotic resistance: A game-theoretic analysis of a potentially catastrophic social dilemma'. *PLoS One*, vol. 14(4): pp. 1–13 (cit. on p. 48).
- DÉLYE, CHRISTOPHE (2013): 'Unravelling the genetic bases of non-target-site-based resistance (NTSR) to herbicides: a major challenge for weed science in the forthcoming decade'. *Pest Management Science*, vol. 69(2): pp. 176–187 (cit. on pp. 2, 5).
- DÉLYE, CHRISTOPHE, CHRYSTEL DEULVOT, and BRUNO CHAUVEL (2013a): 'DNA Analysis of Herbarium Specimens of the Grass Weed *Alopecurus myosuroides* Reveals Herbicide Resistance Pre-Dated Herbicides'. *PLoS ONE*, vol. 8(10): e75117 (cit. on pp. 13–15, 21, 30).
- DÉLYE, CHRISTOPHE, MARIE JASIENIUK, and VALÉRIE LE CORRE (2013b): 'Deciphering the evolution of herbicide resistance in weeds'. *Trends in Genetics*, vol. 29(11): pp. 649–658 (cit. on pp. 4–6).
- DIGGLE, ARTHUR J., PAUL NEVE, and F. PATRICK SMITH (2003): 'Herbicides used in combination can reduce the probability of herbicide resistance in finite weed populations'. *Weed Research*, vol. 43(5): pp. 371–382 (cit. on pp. 10, 21–23).
- DIJK, MICHIEL VAN, TOM MORLEY, MARIE LUISE RAU, and YASHAR SAGHAI (2021): 'A meta-analysis of projected global food demand and population at risk of hunger for the period 2010–2050'. *Nature Food*, vol. 2(7): pp. 494–501 (cit. on p. 1).
- DOGAN, MEHMET N. and ÖZGÜR O. BOZ (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*: pp. 71–79 (cit. on pp. 43, 62, 65).
- DUKE, STEPHEN O. (2012): 'Why have no new herbicide modes of action appeared in recent years?' *Pest Management Science*, vol. 68(4): pp. 505–512 (cit. on pp. 1, 2).

- EGLEY, GRANT H. and JAMES M. CHANDLER (1978): 'Germination and Viability of Weed Seeds After 2.5 Years in a 50-Year Buried Seed Study'. *Weed Science*, vol. 26(3): pp. 230–239 (cit. on pp. 61–64).
- EVANS, JEFFREY A., ALWYN WILLIAMS, AARON G. HAGER, STEVEN B. MIRSKY, PATRICK J. TRANEL, and ADAM S. DAVIS (2018): 'Confronting herbicide resistance with cooperative management'. *Pest Management Science*, vol. 74(11): pp. 2424–2431 (cit. on pp. 45–48).
- EVANS, MARGARET E.K., RÉGIS FERRIÈRE, MICHAEL J. KANE, and D. LAWRENCE VENABLE (2007): 'Bet Hedging via Seed Banking in Desert Evening Primroses (*Oenothera*, *Onagraceae*): Demographic Evidence from Natural Populations'. *The American Naturalist*, vol. 169(2): pp. 184–194 (cit. on p. 4).
- FIRBANK, LES G. and ANDREW R. WATKINSON (1985): 'On the Analysis of Competition Within Two-Species Mixtures of Plants'. *The Journal of Applied Ecology*, vol. 22(2): p. 503 (cit. on pp. 11, 25).
- FRECKLETON, ROBERT P. and PHILIP ANDREW STEPHENS (2009): 'Predictive models of weed population dynamics'. *Weed Research*, vol. 49(3): pp. 225–232 (cit. on pp. 3, 10, 48).
- FRITSCH, CORALIE, DENIS VILLEMONAIS, and NICOLÁS ZALDUENDO (2022): 'The Multi-type Bisexual Galton-Watson Branching Process'. *arXiv* (cit. on p. 76).
- GARIBALDI, LUCAS A., MATÍAS G. GOLDENBERG, ALFRED BURIAN, FERNANDA SANTIBAÑEZ, EMILIO H. SATORRE, GUSTAVO D. MARTINI, and RALF SEPPELT (2023): 'Smaller agricultural fields, more edges, and natural habitats reduce herbicide-resistant weeds'. *Agriculture, Ecosystems & Environment*: p. 108260 (cit. on pp. 47, 48).
- GAROÑA, ANA, MARIO SANTER, NILS F. HÜLTER, HILDEGARD UECKER, and TAL DAGAN (2023): 'Segregational drift hinders the evolution of antibiotic resistance on polyploid replicons'. *PLoS Genetics*, vol. 19(8): e1010829 (cit. on p. 43).
- GHERSA, CLAUDIO M., EMILIO H. SATORRE, and MIGUEL L. VAN ESSO (1985): 'Seasonal Patterns of Johnsongrass Seed Production in Different Agricultural Systems'. *Israel Journal of Plant Sciences*, vol. 34(1): pp. 24–30 (cit. on p. 62).
- GLÉMIN, SYLVAIN and JOËLLE RONFORT (2013): 'Adaptation and Maladaptation in Selfing and Outcrossing Species: New Mutations Versus Standing Variation'. *Evolution*, vol. 67(1): pp. 225–240 (cit. on pp. 6, 42).
- GOMULKIEWICZ, RICHARD and ROBERT D. HOLT (1995): 'When does Evolution by Natural Selection Prevent Extinction?'. *Evolution*, vol. 49(1): p. 201 (cit. on pp. 6, 37).
- HAIRSTON, NELSON G. and BART T. de STASIO (1988): 'Rate of evolution slowed by a dormant propagule pool'. *Nature*, vol. 336(6196): pp. 239–242 (cit. on p. 4).
- HALDANE, JOHN B.S. (1927): 'A mathematical theory of natural and artificial selection. Part V: Selection and mutation'. *Proceedings of the Cambridge Philosophical Society*: pp. 838–844 (cit. on p. 43).
- HAMILL, AL, JODIE HOLT, and CAROL MALLORY-SMITH (2004): 'Contributions of Weed Science to Weed Control and Management1'. *Weed Technology*: pp. 1563–1565 (cit. on p. 1).
- HARMS, CHRISTIAN T. and JOSEPH J. DiMAIO (1991): 'Primisulfuron Herbicide-resistant Tobacco Cell Lines. Application of Fluctuation Test Design to in vitro Mutant Selection with Plant Cells'. *Journal of Plant Physiology*, vol. 137(5): pp. 513–519 (cit. on pp. 14, 15, 61, 66).
- HARRIS, THEODORE E. (1963): *The Theory of Branching Processes*. Berlin: Springer (cit. on pp. 7, 28, 30, 43, 46, 66, 78).
- HARTZLER, ROBERT G., ART GOVER, and JOANNE STELLINGWERF (1991): 'Factors Affecting Winter Survival of Johnsongrass (*Sorghum halepense*) Rhizomes'. *Weed Technology*, vol. 5(1): pp. 108–110 (cit. on pp. 62, 64).
- HAUGHN, GEORGE W. and CHRISTOPHER R. SOMERVILLE (1987): 'Selection for Herbicide Resistance at the Whole-Plant Level'. *Biotechnology in Agricultural Chemistry*. American Chemical Society. Chap. 7: pp. 98–107 (cit. on pp. 14, 15, 61, 66).

- HAWKINS, NICHOLA J., CHRIS BASS, ANDREA DIXON, and PAUL NEVE (2019): 'The evolutionary origins of pesticide resistance'. *Biological Reviews*, vol. 94(1): pp. 135–155 (cit. on pp. 1, 6, 41, 48, 49).
- HEAP, IAN (2024): *The International Herbicide-Resistant Weed Database*. <https://www.weedscience.org> [Accessed: 24 May 2024] (cit. on pp. 1, 2, 45, 47).
- HERMISSON, JOACHIM and PLEUNI S. PENNING (2005): 'Soft Sweeps'. *Genetics*, vol. 169(4): pp. 2335–2352 (cit. on p. 35).
- HERNANDEZ, MARA J., ROCO LEN, ALBERT J. FISCHER, MARLENE GEBAUER, RAFAEL GALDAMES, and RODRIGO FIGUEROA (2015): 'Target-Site Resistance to Nicosulfuron in Johnsongrass (*Sorghum halepense*) from Chilean Corn Fields'. *Weed Science*, vol. 63(3): pp. 631–640 (cit. on p. 12).
- HOLM, LEROY G., DONALD L. PLUCKNETT, JUAN V. PANCHO, and JAMES P. HERBERGER (1977): *The World's Worst Weeds: Distribution and Biology*. East-West Center, University Press of Hawaii (cit. on pp. 4, 9).
- HOLMES, K. HARRISON, JOHN L. LINDQUIST, RICHARD REBARBER, RODRIGO WERLE, MELINDA YERKA, and BRIGITTE TENHUMBERG (2022): 'Modeling the evolution of herbicide resistance in weed species with a complex life cycle'. *Ecological Applications*, vol. 32(1): e02473 (cit. on pp. 4, 10, 12, 22, 23, 43, 45, 46).
- HOLST, NIELS, ILSE A. RASMUSSEN, and LAMMERT BASTIANS (2007): 'Field weed population dynamics: a review of model approaches and applications'. *Weed Research*, vol. 47(1): pp. 1–14 (cit. on pp. 2–4, 10, 45, 48).
- HOROWITZ, MENASHE (1972): 'Early Development of Johnsongrass'. *Weed Science*, vol. 20(3): pp. 271–273 (cit. on pp. 11, 24, 61–63).
- HULL, DAVID M. (1998): 'A Reconsideration of Galton's Problem (Using a Two-Sex Population)'. *Theoretical Population Biology*, vol. 54(2): pp. 105–116 (cit. on p. 76).
- HULL, RICHARD J. (1970): 'Germination Control of Johnsongrass Rhizome Buds'. *Weed Science*, vol. 18(1): pp. 118–121 (cit. on pp. 13, 25, 61, 63, 65).
- JOHNSON, DENNIS B. and JASON K. NORSWORTHY (2014): 'Johnsongrass (*Sorghum halepense*) Management as Influenced by Herbicide Selection and Application Timing'. *Weed Technology*, vol. 28(1): pp. 142–150 (cit. on pp. 2, 43, 62, 65).
- JOHNSON, WILLIAM G. and ROBERT E. FRANS (1991): 'Johnsongrass (*Sorghum halepense*) Control in Soybeans (*Glycine max*) with Postemergence Herbicides'. *Weed Technology*, vol. 5(1): pp. 87–91 (cit. on p. 62).
- JOHNSON, WILLIAM G., JIANMEI LI, and JIMMY D. WAIT (2003): 'Johnsongrass Control, Total Nonstructural Carbohydrates in Rhizomes, and Regrowth After Application of Herbicides Used in Herbicide-Resistant Corn (*Zea mays*)¹'. *Weed Technology*, vol. 17(1): pp. 36–41 (cit. on pp. 62, 65).
- KALISZ, SUSAN and MARK A. MCPEEK (1993): 'Extinction dynamics, population growth and seed banks'. *Oecologia*, vol. (3): pp. 314–320 (cit. on p. 4).
- KEELEY, PAUL E. and ROBERT J. THULLEN (1979): 'Influence of Planting Date on the Growth of Johnsongrass (*Sorghum halepense*) from Seed'. *Weed Science*, vol. 27(5): pp. 554–558 (cit. on pp. 61–63).
- KERSTEN, SONJA, JIYANG CHANG, CHRISTIAN D. HUBER, YOAV VOICHEK, CHRISTA LANZ, TIMO HAGMAIER, PATRICIA LANG, ULRICH LUTZ, INSA HIRSCHBERG, JENS LERCHL, AIMONE PORRI, YVES VAN DE PEER, KARL SCHMID, DETLEF WEIGEL, and FERNANDO A. RABANAL (2023): 'Standing genetic variation fuels rapid evolution of herbicide resistance in blackgrass'. *Proceedings of the National Academy of Sciences*, vol. 120(16): e2206808120 (cit. on pp. 6, 30, 41).
- KIMMEL, MAREK and DAVID E. AXELROD (2002): *Branching Processes in Biology*. Springer NY (cit. on pp. 7, 30).

- KLEIN, PETER and CHARLES MICHAEL SMITH (2021): 'Invasive Johnsongrass, a threat to native grasslands and agriculture'. *Biologia*, vol. 76(2): pp. 413–420 (cit. on pp. 4, 9).
- KREINER, JULIA M., GEORGE SANDLER, AARON J. STERN, PATRICK J. TRANEL, DETLEF WEIGEL, JOHN R. STINCHCOMBE, and STEPHEN I. WRIGHT (2022): 'Repeated origins, widespread gene flow, and allelic interactions of target-site herbicide resistance mutations'. *eLife*: e70242 (cit. on pp. 6, 30, 41).
- KREINER, JULIA M., JOHN R. STINCHCOMBE, and STEPHEN I. WRIGHT (2017): 'Population Genomics of Herbicide Resistance: Adaptation via Evolutionary Rescue'. *Annual Review of Plant Biology*, vol. 69(1): pp. 1–25 (cit. on pp. 2, 3, 6, 12, 13, 27, 29, 30, 37, 43, 46).
- KRENCHINSKI, FÁBIO H., ALFREDO J.P. ALBRECHT, LEANDRO P. ALBRECHT, H.L. VILLETTI, GABRIEL ORSO, ARTHUR A.M. BARROSO, and RICARDO VICTORIA FILHO (2015): 'Germination and Dormancy in Seeds of *Sorghum halepense* and *Sorghum arundinaceum*'. *Planta Daninha*, vol. 33(2): pp. 223–230 (cit. on pp. 61, 62).
- LANDAU, CHRISTOPHER et al. (2023): 'The silver bullet that wasn't: Rapid agronomic weed adaptations to glyphosate in North America'. *PNAS Nexus*, vol. 2(12): pgad338 (cit. on p. 48).
- LANDRY, RANDALL L., DANIEL O. STEPHENSON, and BRANDI C. WOOLAM (2016): 'Glufosinate Rate and Timing for Control of Glyphosate-Resistant Rhizomatous Johnsongrass (*Sorghum halepense*) in Glufosinate-Resistant Soybean'. *International Journal of Agronomy*: pp. 1–6 (cit. on pp. 61, 63).
- LAUENROTH, DANA and CHAITANYA S. GOKHALE (2023): 'Theoretical assessment of persistence and adaptation in weeds with complex life cycles'. *Nature Plants*: pp. 1267–1279 (cit. on pp. 31, 32, 36, 37, 42, 73, 75, 79, 81).
- LEGUIZAMÓN, EDUARDO S. (1986): 'Seed survival and patterns of seedling emergence in *Sorghum halepense* (L.) Pers.'. *Weed Research*, vol. 26(6): pp. 397–404 (cit. on pp. 11, 24, 61–64).
- LENNON, JAY T., FRANK DEN HOLLANDER, MAITE WILKE-BERENGUER, and JOCHEN BLATH (2021): 'Principles of seed banks and the emergence of complexity from dormancy'. *Nature Communications*, vol. 12(1): p. 4807 (cit. on p. 4).
- LIU, CHUN, JULIO A. SCURSONI, RAÚL MORENO, IAN A. ZELAYA, MARÍA SOL MUÑOZ, and SHIV S. KAUNDUN (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*, vol. 9(17): pp. 10017–10028 (cit. on pp. 4, 10, 18, 22, 45, 46, 62, 70).
- LODDO, DONATO, ROBERTA MASIN, STEFAN OTTO, and GIUSEPPE ZANIN (2012): 'Estimation of base temperature for *Sorghum halepense* rhizome sprouting'. *Weed Research*, vol. 52(1): pp. 42–49 (cit. on pp. 61, 63).
- LOLAS, PETROS C. and HAROLD D. COBLE (1980): 'Johnsongrass (*Sorghum halepense*) growth characteristics as related to rhizome length'. *Weed Research*, vol. 20(4): pp. 205–210 (cit. on p. 63).
- LORENTZ, LOTHAR (2014): 'Herbicide Resistance - Molecular and Physiological Characterization of the Glyphosate Resistant Weeds *Amaranthus* ssp. and *Sorghum* ssp.'. PhD thesis. Rheinische Friedrich-Wilhelms-Universität Bonn (cit. on pp. 4, 5, 10, 22).
- MAITY, ANIRUDDHA, BLAKE YOUNG, NITHYA SUBRAMANIAN, and MUTHUKUMAR BAGAVATHIAN-NAN (2022): 'Pollen-mediated transfer of herbicide resistance between johnsongrass (*Sorghum halepense*) biotypes'. *Scientific Reports*, vol. 12(1): p. 7663 (cit. on pp. 2, 47).
- MAQSOOD, QAISER, RANA N. ABBAS, MUHAMMAD A. IQBAL, KIZIL A. SERAP, ASIF IQBAL, and AYMAN EL SABAGH (2020): 'Overviewing of weed management practices to reduce weed seed bank and to increase maize yield'. *Planta daninha*: e020199716 (cit. on p. 3).
- MAXWELL, BRUCE D., MARY LYNN ROUSH, and STEVEN R. RADOSEVICH (1990): 'Predicting the Evolution and Dynamics of Herbicide Resistance in Weed Populations'. *Weed Technology*, vol. 4(1): pp. 2–13 (cit. on p. 23).

- MCWHORTER, CHESTER G. (1961): 'Morphology and Development of Johnsongrass Plants from Seeds and Rhizomes'. *Weeds*, vol. 9(4): p. 558 (cit. on pp. 11, 24, 65).
- (1971): 'Growth and Development of Johnsongrass Ecotypes'. *Weed Science*, vol. 19(2): pp. 141–147 (cit. on pp. 61–63).
- MCWHORTER, CHESTER G. and EDGAR E. HARTWIG (1965): 'Effectiveness of Preplanting Tillage in Relation to Herbicides in Controlling Johnsongrass for Soybean Production'. *Agronomy Journal*, vol. 57(4): pp. 385–389 (cit. on pp. 13, 22).
- MENCHARI, YOSRA, BRUNO CHAUVEL, HENRI DARMENCY, and CHRISTOPHE DÉLYE (2008): 'Fitness costs associated with three mutant acetyl-coenzyme A carboxylase alleles endowing herbicide resistance in black-grass *Alopecurus myosuroides*'. *Journal of Applied Ecology*, vol. 45(3): pp. 939–947 (cit. on pp. 15, 21, 23, 42, 61).
- MORISON, CHRISTO, MAŁGORZATA FIC, THOMAS MARCOU, JAVAD MOHAMADICHAMGAVI, JAVIER REDONDO ANTÓN, GOLSA SAYYAR, ALEXANDER STEIN, FRANK BASTIAN, HANA KRAKOVSKÁ, NANDAKISHOR KRISHNAN, DIOGO PIRES, MOHAMMAD REZA SATOURI, FREDERIK JASPER THOMSEN, KAUSUTUA TJIKUNDI, and WAJID ALI (2024): 'Public Goods Games in Disease Evolution and Spread'. *arXiv* (cit. on p. 48).
- MOSS, STEPHEN (2019): 'Integrated weed management (IWM): why are farmers reluctant to adopt non-chemical alternatives to herbicides?' *Pest Management Science*, vol. 75(5): pp. 1205–1211 (cit. on p. 48).
- NEVE, PAUL (2008): 'Simulation modelling to understand the evolution and management of glyphosate resistance in weeds'. *Pest Management Science*, vol. 64(4): pp. 392–401 (cit. on pp. 6, 42, 47).
- NEVE, PAUL, ROBERTO BUSI, MICHAEL RENTON, and MARTIN M. VILA-AIUB (2014): 'Expanding the eco-evolutionary context of herbicide resistance research'. *Pest Management Science*, vol. 70(9): pp. 1385–1393 (cit. on pp. 5, 6, 30, 37, 45, 46).
- OERKE, ERICH-CHRISTIAN (2006): 'Crop losses to pests'. *Journal of Agricultural Science*: pp. 31–43 (cit. on pp. 1, 29).
- OHADI, SARA, GEORGE HODNETT, WILLIAM ROONEY, and MUTHUKUMAR BAGAVATHIANNAN (2018): 'Gene Flow and its Consequences in Sorghum spp.'. *Critical Reviews in Plant Sciences*, vol. 36(5-6): pp. 1–19 (cit. on p. 61).
- ORIVE, MARIA E., MICHAEL BARFIELD, CARLOS FERNANDEZ, and ROBERT D. HOLT (2017): 'Effects of Clonal Reproduction on Evolutionary Lag and Evolutionary Rescue'. *The American Naturalist*, vol. 190(4): pp. 469–490 (cit. on pp. 4, 6, 42).
- ORR, H. ALLEN (2005): 'The genetic theory of adaptation: a brief history'. *Nature Reviews Genetics*, vol. 6(2): pp. 119–127 (cit. on p. 1).
- ORR, H. ALLEN and ROBERT L. UNCKLESS (2008): 'Population extinction and the genetics of adaptation'. *The American Naturalist*: pp. 160–169 (cit. on pp. 7, 30, 43, 46).
- (2014): 'The Population Genetics of Evolutionary Rescue'. *PLoS Genetics*, vol. 10(8): e1004551 (cit. on pp. 41, 43, 79).
- PALUMBI, STEPHEN R. (2001): 'Humans as the World's Greatest Evolutionary Force'. *Science*: pp. 1786–1790 (cit. on pp. 1, 49).
- PANOZZO, SILVIA and MAURIZIO SATTIN (2021): 'Fitness Costs Associated to an Ile2041Asn Mutation in the Geophyte Sorghum halepense Resistant to ACCase-Inhibiting Herbicides'. *Frontiers in Agronomy*: p. 711840 (cit. on pp. 6, 13, 15, 21, 26, 61).
- PATERSON, ANDREW H. et al. (2020): 'The Evolution of an Invasive Plant, Sorghum halepense L. ('Johnsongrass')'. *Frontiers in Genetics*: p. 317 (cit. on pp. 11, 24).
- PEERZADA, ARSLAN MASOOD, HAFIZ HAIDER ALI, ZARKA HANIF, ALI AHSAN BAJWA, LYNDIA KEBASO, DAVID FRIMPONG, NADEEM IQBAL, HALIMA NAMUBIRU, SAIMA HASHIM, GHULAM RASOOL, SUDHEESH MANALIL, ANNEMIEKE van der MEULEN, and BHAGIRATH SINGH CHAUHAN

- (2023): ‘Eco-biology, impact, and management of *Sorghum halepense* (L.) Pers.’ *Biological Invasions*, vol. 25(4): pp. 955–973 (cit. on pp. 4, 5, 9–11, 13, 21, 24, 27, 30, 31, 75).
- PEROTTI, VALERIA E., ALVARO S. LARRAN, VALERIA E. PALMIERI, ANDREA K. MARTINATTO, and HUGO R. PERMINGEAT (2020): ‘Herbicide resistant weeds: A call to integrate conventional agricultural practices, molecular biology knowledge and new technologies’. *Plant Science*: p. 110255 (cit. on pp. 1, 2, 4, 29, 47, 48).
- PORCO, TRAVIS C., DAOZHOU GAO, JAMES C. SCOTT, EUNHA SHIM, WAYNE T. ENANORIA, ALISON P. GALVANI, and THOMAS M. LIETMAN (2012): ‘When Does Overuse of Antibiotics Become a Tragedy of the Commons?’ *PLoS One*, vol. 7(12): pp. 1–12 (cit. on p. 48).
- POWLES, STEPHEN B. and QIN YU (2010): ‘Evolution in Action: Plants Resistant to Herbicides’. *Annual Review of Plant Biology*, vol. 61(1): pp. 317–347 (cit. on pp. 1, 4–6, 29, 48).
- PRESTON, CHRISTOPHER and STEPHEN B. POWLES (2002): ‘Evolution of herbicide resistance in weeds: initial frequency of target site-based resistance to acetolactate synthase-inhibiting herbicides in *Lolium rigidum*’. *Heredity*, vol. 88(1): pp. 8–13 (cit. on pp. 14, 15, 21, 30).
- PURRINGTON, COLIN B. (2000): ‘Costs of resistance’. *Current Opinion in Plant Biology*, vol. 3(4): pp. 305–308 (cit. on p. 5).
- QU, REN-YU, BO HE, JING-FANG YANG, HONG-YAN LIN, WEN-CHAO YANG, QIONG-YOU WU, QING X LI, and GUANG-FU YANG (2021): ‘Where are the new herbicides?’ *Pest Management Science*, vol. 77(6): pp. 2620–2625 (cit. on pp. 1, 2, 5, 47).
- RAMANKUTTY, NAVIN, ZIA MEHRABI, KATHARINA WAHA, LARISSA JARVIS, CLAIRE KREMEN, MARIO HERRERO, and LOREN H. RIESEBERG (2018): ‘Trends in Global Agricultural Land Use: Implications for Environmental Health and Food Security’. *Annual Review of Plant Biology*: pp. 789–815 (cit. on p. 48).
- REDDY, KRISHNA N. and VIJAY K. NANDULA (2012): ‘Herbicide resistant crops: History, development and current technologies’. *Indian Journal of Agronomy*, vol. 57(1): pp. 1–7 (cit. on p. 2).
- RENTON, MICHAEL, ROBERTO BUSI, PAUL NEVE, DAVID THORNBY, and MARTIN M. VILA-AIUB (2014): ‘Herbicide resistance modelling: past, present and future’. *Pest Management Science*, vol. 70(9): pp. 1394–1404 (cit. on pp. 3, 48).
- SAGER, G. R. and A. M. MORTIMER (1976): ‘An approach to the study of the population dynamics of plants with special reference to weeds’. *Applied Biology*: pp. 1–47 (cit. on pp. 2, 10).
- SANTER, MARIO and HILDEGARD UECKER (2020): ‘Evolutionary Rescue and Drug Resistance on Multicopy Plasmids’. *Genetics*, vol. 215(3): pp. 847–868 (cit. on pp. 7, 30, 43).
- SCARABEL, LAURA, SILVIA PANOZZO, WALTER SAVOIA, and MAURIZIO SATTIN (2014): ‘Target-Site ACCase-Resistant Johnsongrass (*Sorghum halepense*) Selected in Summer Dicot Crops’. *Weed Technology*, vol. 28(2): pp. 307–315 (cit. on pp. 12, 31).
- SCHWINNING, SUSANNE, HEATHER MECKEL, LARA G. REICHMANN, H. WAYNE POLLEY, and PHILIP A. FAY (2017): ‘Accelerated development in Johnsongrass seedlings (*Sorghum halepense*) suppresses the growth of native grasses through size-asymmetric competition’. *PLoS ONE*, vol. 12(5): e0176042 (cit. on pp. 4, 9, 11, 25, 43, 75).
- SCOPEL, ANA L., CARLOS L. BALLARE, and CLAUDIO M. GHERSA (1988): ‘Role of Seed Reproduction in the Population Ecology of *Sorghum halepense* in Maize Crops’. *The Journal of Applied Ecology*, vol. 25(3): p. 951 (cit. on p. 23).
- SELBY, THOMAS P., ANDREW D. SATTERFIELD, ATUL PURI, THOMAS M. STEVENSON, D. ANDREW TRAVIS, MATTHEW J. CAMPBELL, ANDREW E. TAGGI, KENNETH A. HUGHES, and JAMES BEREZNAK (2023): ‘Bioisosteric Tactics in the Discovery of Tetflupyrolimet: A New Mode-of-Action Herbicide’. *Journal of Agricultural and Food Chemistry*, vol. 71(47): pp. 18197–18204 (cit. on pp. 2, 47).
- SERRA, MARIA CONCEIÇÃO and PATSY HACCOU (2007): ‘Dynamics of escape mutants’. *Theoretical Population Biology*, vol. 72(1): pp. 167–178 (cit. on pp. 7, 30, 46).

- SEWASTJANOV, BORIS A. (1974): *Verzweigungsprozesse*. Trans. by WARMUTH, WALTER. Berlin: Akademie-Verlag (cit. on p. 33).
- SHANER, DALE L. and HUGH J. BECKIE (2014): 'The future for weed control and technology'. *Pest Management Science*, vol. 70(9): pp. 1329–1339 (cit. on pp. 47, 48).
- SKØIEN, SVEIN ERIK, TROND BØRRESEN, and MARIANNE BECHMANN (2012): 'Effect of tillage methods on soil erosion in Norway'. *Acta Agriculturae Scandinavica, Section B — Soil & Plant Science*, vol. 62(sup2): pp. 191–198 (cit. on p. 22).
- SOMERVILLE, GAYLE J., STEPHEN B. POWLES, MICHAEL J. WALSH, and MICHAEL RENTON (2017): 'How do spatial heterogeneity and dispersal in weed population models affect predictions of herbicide resistance evolution?' *Ecological Modelling*: pp. 37–53 (cit. on p. 47).
- STOLLER, EDWARD W. (1977): 'Differential Cold Tolerance of Quackgrass and Johnsongrass Rhizomes'. *Weed Science*, vol. 25(4): pp. 348–351 (cit. on pp. 62, 64).
- STORKEY, JONATHAN, ANTOINE GARDARIN, NATHALIE COLBACH, HELEN METCALFE, and ALICE MILNE (2022): 'Modelling the Persistence of Weed Populations'. *Persistence Strategies of Weeds*. John Wiley & Sons, Ltd. Chap. 9: pp. 165–183 (cit. on p. 3).
- THE MATHWORKS INC. (2022): *MATLAB version: 9.14.0 (R2023a)*. Natick, Massachusetts, United States (cit. on p. 36).
- TIDEMANN, BREANNE D., K. NEIL HARKER, STEVE J. SHIRTLIFFE, CHRISTIAN J. WILLENBORG, ERIC N. JOHNSON, ELIZABETH SROKA, JENNIFER ZUIDHOF, and HEMA DUDDU (2021): 'Evaluation of panicle removal methods and crop topping applications as supplemental tools for wild oat (*Avena fatua*) management'. *Weed Technology*, vol. 35(6): pp. 932–944 (cit. on p. 30).
- TOMASINI, MATTEO and STEPHAN PEISCHL (2020): 'When does gene flow facilitate evolutionary rescue?' *Evolution*, vol. 74(8): pp. 1640–1653 (cit. on p. 43).
- TORRA, JOEL, JOSÉ MARÍA MONTULL, ANDREU TABERNER, NAWAPORN ONKOKESUNG, NEIL BOONHAM, and ROBERT EDWARDS (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*: p. 625138 (cit. on p. 13).
- TÓTH, VIKTOR and ÉVA LEHOCZKY (2006): 'Investigations on the germination depth of Johnson grass (*Sorghum halepense* [L.] pers)'. *Communications in Agricultural and Applied Biological Sciences*, vol. 71(3): pp. 803–808 (cit. on pp. 61, 62).
- TUDI, MUYESAIE, HUADA DANIEL RUAN, LI WANG, JIA LYU, ROSS SADLER, DES CONNELL, CORDIA CHU, and DUNG TRI PHUNG (2021): 'Agriculture Development, Pesticide Application and Its Impact on the Environment'. *International Journal of Environmental Research and Public Health*, vol. 18(3): p. 1112 (cit. on pp. 1, 29).
- TUESCA, DANIEL, EDUARDO PURICELLI, LUISA NISENSOHN, DELMA FACCINI, and JUAN PAPA (1999): 'Decision criteria for optimizing postemergence johnsongrass control in soybean crops in Argentina'. *Pesquisa Agropecuária Brasileira*, vol. 34(5): pp. 749–753 (cit. on pp. 13, 25, 38, 42, 62, 65).
- UECKER, HILDEGARD (2017): 'Evolutionary rescue in randomly mating, selfing, and clonal populations'. *Evolution*, vol. 71(4): pp. 845–858 (cit. on pp. 6, 42).
- UECKER, HILDEGARD and JOACHIM HERMISSON (2016): 'The Role of Recombination in Evolutionary Rescue'. *Genetics*, vol. 202(2): pp. 721–732 (cit. on p. 7).
- UECKER, HILDEGARD, SARAH P. OTTO, and JOACHIM HERMISSON (2014): 'Evolutionary Rescue in Structured Populations'. *The American Naturalist*, vol. 183(1): E17–E35 (cit. on pp. 7, 30, 46).
- VIDRINE, PAUL R. (1989): 'Johnsongrass (*Sorghum halepense*) Control in Soybeans (*Glycine max*) with Postemergence Herbicides'. *Weed Technology*, vol. 3(3): pp. 455–458 (cit. on pp. 13, 25, 38, 42, 62, 65).
- VIGUEIRA, CINDY C., KENNETH M. OLSEN, and ANA L. CAICEDO (2013): 'The red queen in the corn: agricultural weeds as models of rapid adaptive evolution'. *Heredity*, vol. 110(4): pp. 303–311 (cit. on pp. 5, 30).

- VILA-AIUB, MARTIN M., MARIA C. BALBI, PEDRO E. GUNDEL, CLAUDIO M. GHERSA, and STEPHEN B. POWLES (2007): 'Evolution of Glyphosate-Resistant Johnsongrass (*Sorghum halepense*) in Glyphosate-Resistant Soybean'. *Weed Science*, vol. 55(6): pp. 566–571 (cit. on pp. 4, 5, 10).
- VILA-AIUB, MARTIN M., PAUL NEVE, and STEPHEN B. POWLES (2009): 'Fitness costs associated with evolved herbicide resistance alleles in plants'. *New Phytologist*, vol. 184(4): pp. 751–767 (cit. on p. 6).
- VRBNIČANIN, SAVA, DRAGANA BOŽIĆ, and DANIJELA PAVLOVIĆ (2017): 'Gene Flow from Herbicide-Resistant Crops to Wild Relatives'. *Herbicide Resistance in Weeds and Crops*. Ed. by PACANOSKI, ZVONKO. Rijeka: IntechOpen. Chap. 3 (cit. on p. 23).
- WARWICK, SUZANNE I. and LYALL D. BLACK (1983): 'The biology of Canadian weeds. 61. *Sorghum halepense* (L.) Pers.'. *Canadian Journal of Plant Science*: pp. 997–1014 (cit. on pp. 4, 9–13, 24–27, 30–32, 61, 62, 75).
- WARWICK, SUZANNE I., DARREN PHILLIPS, and CHRISTOPHER ANDREWS (1986): 'Rhizome depth: the critical factor in winter survival of *Sorghum halepense* (L.) Pers. (Johnson grass)'. *Weed Research*, vol. 26(6): pp. 381–388 (cit. on pp. 4, 62, 64, 65).
- WATKINSON, ANDREW R. (1980): 'Density-dependence in single-species populations of plants'. *Journal of Theoretical Biology*, vol. 83(2): pp. 345–357 (cit. on p. 26).
- WEISBERGER, DAVID, VIRGINIA NICHOLS, and MATT LIEBMAN (2019): 'Does diversifying crop rotations suppress weeds? A meta-analysis'. *PLoS ONE*, vol. 14(7): e0219847 (cit. on p. 23).
- WILLIAMS, R.D. and B.F. INGBER (1977): 'The Effect of Intraspecific Competition on the Growth and Development of Johnsongrass under Greenhouse Conditions'. *Weed Science*, vol. 25(4): pp. 293–297 (cit. on pp. 4, 11, 25, 43, 61, 63).
- WILSON, BENJAMIN A., PLEUNI S. PENNINGS, and DMITRI A. PETROV (2017): 'Soft Selective Sweeps in Evolutionary Rescue'. *Genetics*, vol. 205(4): pp. 1573–1586 (cit. on p. 43).
- WINTON-DANIELS, KIM, ROBERT FRANS, and MARILYN MCCLELLAND (1990): 'Herbicide Systems for Johnsongrass (*Sorghum halepense*) Control in Soybeans (*Glycine max*)'. *Weed Technology*, vol. 4(1): pp. 115–122 (cit. on p. 65).
- WOLFRAM RESEARCH, INC. (2022): *Mathematica, Version 13.1*. Wolfram Research, Inc. Champaign, IL, 2022 (cit. on p. 43).
- YAMAMICHI, MASATO, NELSON G. HAIRSTON, MARK REES, and STEPHEN P. ELLNER (2019): 'Rapid evolution with generation overlap: the double-edged effect of dormancy'. *Theoretical Ecology*, vol. 12(2): pp. 179–195 (cit. on p. 6).
- YODA, K., T. KIRA, H. OGAWA, and K. HOZUMI (1963): 'Self-Thinning in Overcrowded Pure Stands under Cultivated and Natural Conditions'. *Journal of Biology*: pp. 107–129 (cit. on pp. 11, 25).
- ZELLER, ALEXANDER K., YASMIN I. ZELLER, and ROLAND GERHARDS (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*: p. 105613 (cit. on pp. 10, 22, 23).

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

This chapter is the appendix of [Chapter 2](#) and published as such in a peer-reviewed journal. Lauenroth, D., and Gokhale, C.S. *Theoretical assessment of persistence and adaptation in weeds with complex life cycles*. Nature Plants 9, 1267–1279 (2023). DOI: [10.1038/s41477-023-01482-1](#).

Model parameters

Table A.1 | Model parameters, values and references.

Model parameters	Value	Comments	References
Sprouting probability without tillage after tillage	$g_Z = 0.2$ $g_Z^* = 0.3$	influenced by apical dominance and temperature; wide variation in reported values (HOROWITZ, 1972; HULL, 1970; KEELEY et al., 1979)	LODDO et al. (2012)
Germination probability	$g = 0.3$	influenced by environmental factors not considered in the model, e.g. temperature, light and burial depth (HOROWITZ, 1972; KRENCHINSKI et al., 2015; TÓTH et al., 2006)	EGLEY et al. (1978), LEGUIZAMÓN (1986), and WARWICK et al. (1983)
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 et al. (2021)
Proportion of self-pollination	$p_{self} = 0.95$	there are arguments for higher levels of cross-pollination (OHADI et al., 2018)	WARWICK et al. (1983)
Mutation rate	$\mu = 10^{-8}$	estimates of spontaneous mutation rates of ALS resistance alleles in thale cress (<i>Arabidopsis thaliana</i>) and cultivated tobacco (<i>Nicotiana tabacum</i>)	HARMS et al. (1991) and HAUGHN et al. (1987)
Possible highest limit of plant density (m^{-2})	$m^{-1} = 220$	a lower ceiling density is reached for populations growing from low densities	LANDRY et al. (2016), MCWHORTER (1971), and WILLIAMS et al. (1977)

(continues)

Table A.1 | (continued)

Model parameters	Value	Comments	References
Herbicide efficacy			
ACCcase-inhibitor (high efficacy ALS-inhibitor)			
on seedlings	$h_L = 0.998$	influenced by environmental factors	VIDRINE (1989)
on shoots	$h_T = 0.985$	and application timing (DOGAN	
on shoots after tillage	$h_T^* = 0.992$	et al., 2005; JOHNSON et al., 2014;	
ALS-inhibitor (low efficacy)		TUESCA et al., 1999); wide variation	
on seedlings	$h_L = 0.92$	in reported regrowth (JOHNSON	JOHNSON et al. (2003)
on shoots	$h_T = 0.90$	et al., 1991; VIDRINE, 1989)	
on shoots after tillage	$h_T^* = 0.91$		
Number of rhizome buds produced per isolated plant	$b = 140$	wide variation in reported numbers (ACCIARESI et al., 2005; ANDERSON et al., 1960; HARTZLER et al., 1991)	KEELEY et al. (1979)
Rhizome loss		influenced by climatic conditions and	
winter mortality		burial depth not considered in the	
without tillage	$d_Z = 0.35$	model (HARTZLER et al., 1991;	HARTZLER et al. (1991)
after tillage	$d_Z^* = 0.6$	STOLLER, 1977; WARWICK et al., 1986)	
Number of viable seeds produced per isolated plant	$f = 13,000$	wide variation between ecotypes and in reported numbers (ACCIARESI et al., 2005; GHERSA et al., 1985; MCWHORTER, 1971)	KEELEY et al. (1979)
Seed loss		conflicting information on the impact	
fresh seeds	$d_S = 0.94$	of burial depth and wide variation in	BAGAVATHIANNAN et al. (2013)
old seeds in seed bank	$d_B = 0.48$	reported values (BAGAVATHIANNAN et al., 2013; EGLEY et al., 1978; LEGUIZAMÓN, 1986)	
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 et al. (1979)
Initial density (m ⁻²)			
seeds	10		LIU et al. (2019)
rhizomes	1		
Field size (m ²)	$A = 10,000$		LIU et al. (2019)

Seed germination. Germination of Johnsongrass seeds is significantly influenced by temperature, light and burial depth (HOROWITZ, 1972; KRENCHINSKI et al., 2015; TÓTH et al., 2006). KEELEY et al. (1979) observed germination frequencies in the range of 24 to 91.5 % within three 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, lower optimal germination temperature and reduced germination in the absence of light. They documented maximum germination at 30°C of 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 et al., 2006).

However, the population-level model does not account for these environmental factors. WARWICK

et al. (1983) found a mean seed germination frequency of 38 % in overwintering populations of Johnsongrass. This observation agrees with a study by LEGUIZAMÓN (1986). Therein, 37 % of seeds left on the soil surface germinated within the first year and 25 % of seeds transferred from the surface to 15 cm depth in autumn, simulating tillage practice. Our germination probability $g = 0.3$ is 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 et al. (1978).

Rhizome bud sprouting. In Johnsongrass, the development of rhizome buds is markedly controlled by apical dominance (BEASLEY, 1970; HULL, 1970). Rhizome apices 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 Johnsongrass are reported to be highly temperature dependant (HOROWITZ, 1972; HULL, 1970), with a threshold temperature of 10°C, 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 et al. (1979) detected no relevant influence of temperature on rhizome bud sprouting (56 % at 18 – 35°C) in their experiments, but a significant impact on shoot development. Shoots emerged from 13 and 88 % of the sprouted buds at 18 and 24 – 35°C, respectively.

Our sprouting probability for buds on fragmented rhizomes in a tillage regime 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. Johnsongrass produces tufts as a result of tillering, i.e. the development of secondary shoots from the crown area of existing plants (HOROWITZ, 1972; MCWHORTER, 1971). We do not explicitly model the production of tillers. However, the process of tillering is reflected in density-dependent reproduction. LOLAS et al. (1980) reported a considerable variation between different growing seasons in the number of tillers produced by a plant, 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 et al. (1979) on average counted 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, on average, counted 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 et al., 1977). We assume that intraspecific competition becomes appreciable at a density of 10 plants per m^2 , i.e. 200 culms m^{-2} under maximum tillering. In the study of KEELEY et al. (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 et al. (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 animal predation. 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 (LEGUIZAMÓN, 1986). After three 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 six years. Moreover, LEGUIZAMÓN (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 et al. (2013) reported 64 to 75 % of seed loss due to predation, from dispersal in autumn till next spring, when seeds were left on the soil surface (no-tillage). Most recovered seeds (78 – 91 %) were buried in the soil. In the same 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 et al., 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 then buried in the soil and therefore subsequently inaccessible for predators, at a proportion of 75 % (BAGAVATHIANNAN et al., 2013). Our model includes 17 % seed decay and 23 % loss in viability from the 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 et al. (2013), but higher than found in the undisturbed experiments of EGLEY et al. (1978) and LEGUIZAMÓN (1986). However, our parameters are in good agreement with the simulated cultivation experiments conducted by LEGUIZAMÓN (1986). BAGAVATHIANNAN et al. (2013) calculated the proportion of predation, correcting for the proportion of decayed seeds. Following this, 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 (HARTZLER et al., 1991; STOLLER, 1977; WARWICK et al., 1986). 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 a 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, 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 are 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 $d_Z = 0.35$, used in our model as the winter mortality in no-tillage regimes. 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 efficacy. The herbicide efficacy on Johnsongrass aboveground material depends on the developmental stage of the plants, with small young plants killed with higher efficacy than larger older plants (DOGAN et al., 2005; JOHNSON et al., 2014; TUESCA et al., 1999). The efficacy also varies between years due to environmental factors and application timing (TUESCA et al., 1999; VIDRINE, 1989).

JOHNSON et al. (2003) and JOHNSON et al. (2014) report the investigated ACCase-inhibitors to have a higher efficacy on Johnsongrass than ALS-inhibitors included in their studies. A single application of the ACCase-inhibitor Quizalofop led to a 97 % reduction in Johnsongrass biomass three 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 % two weeks after treatment while Nicosulfuron provided 90 % control of 15 cm Johnsongrass and only 74 % control of 30 cm plants (JOHNSON et al., 2014). Even though they noticed Johnsongrass regrowth after two weeks, stand reduction four 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 efficacy 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 % three weeks after treatment and 95 % for rhizome shoots. Sethoxydim failed to control Johnsongrass shoots effectively in one year of the experiment. Excluding sethoxydim, 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 be unable to produce new rhizomes and viable seeds until 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 efficacy of ACCase-inhibiting herbicides to be 99.8 % on seedlings and to be reduced on shoots with 98.5 %. In some parts of our manuscript, this efficacy is also assumed for the ALS-inhibitor to secure effective weed reduction, for example, in herbicide rotations, and is denoted by 'high efficacy'. As ALS-inhibiting herbicides are reported to control Johnsongrass with a lower efficacy (JOHNSON et al., 2014; JOHNSON et al., 2003), we also investigate a low ALS-inhibitor efficacy of 92 and 90 % on seedlings and rhizome shoots, respectively, denoted by 'low efficacy'. 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 efficacy 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 (HARMS et al., 1991; HAUGHN et al., 1987). Therefore the inheritance matrices (2.17) used to calculate the number of seeds arising from cross-pollination (2.16) are given by,

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

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

$$\mathbf{M}^{\text{RR}} = \mu^2 \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix} + \mu \begin{pmatrix} 0 & 0.5 & 1 \\ 0.5 & 0.5 & 0.5 \\ 1 & 0.5 & 0 \end{pmatrix} + \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0.25 & 0.5 \\ 0 & 0.5 & 1 \end{pmatrix}. \quad (\text{A.3})$$

Matrix theory

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 $k \times k$ 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*

$$\mathbf{A}^n = \rho^n \mathbf{u}^T \mathbf{v} + \mathbf{R}^n, \quad (\text{A.4})$$

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

$$\lim_{n \rightarrow \infty} \frac{\mathbf{A}^n}{\rho^n} = \mathbf{u}^T \mathbf{v}, \quad (\text{A.5})$$

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

Supplementary figures

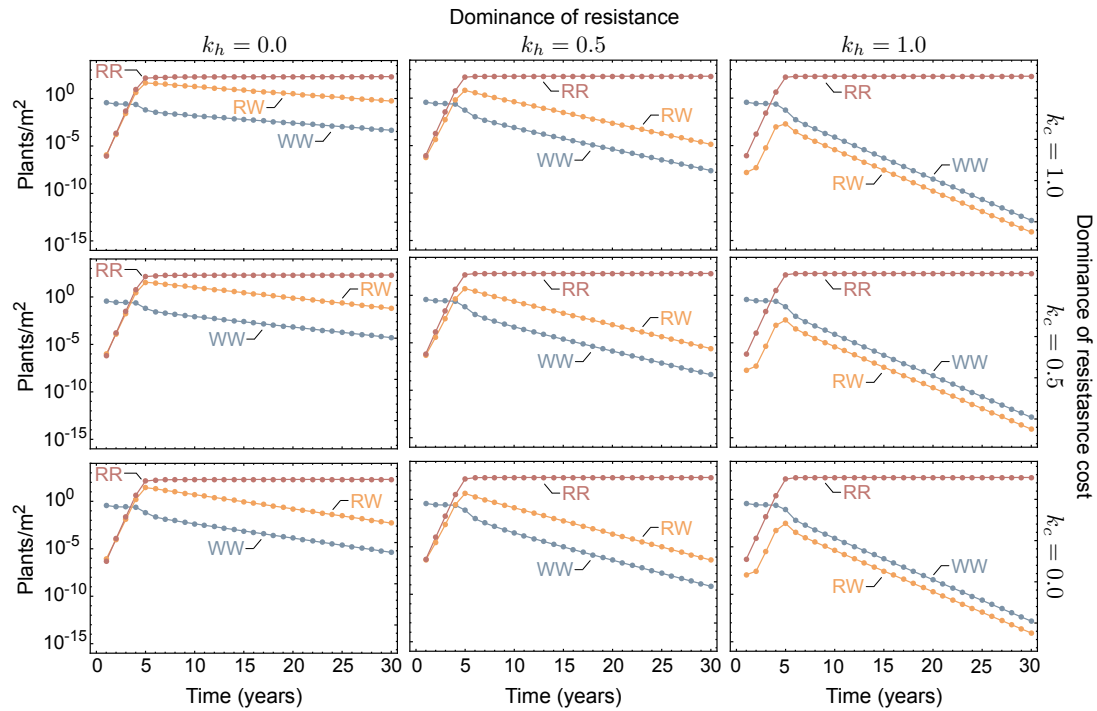


Figure A.1 | Predicted target-site resistance evolution in herbicide-treated Johnsongrass depending on the resistance and cost dominance. Shown are predictions of our deterministic model for the genotype composition of Johnsongrass plants (\tilde{P}) over 30 years of herbicide application for different degrees of dominance regarding resistance (k_h) and the fitness cost associated with resistance (k_c). ($k = 0$) corresponds to recessivity, ($k = 0.5$) indicates partial dominance and ($k = 1$) complete dominance. The frequency of sensitive plants (WW) is shown in blue, resistant heterozygotes (RW) in yellow and resistant homozygotes (RR) are represented in red. The initial genotype composition differs with the dominance of resistance and fitness cost.

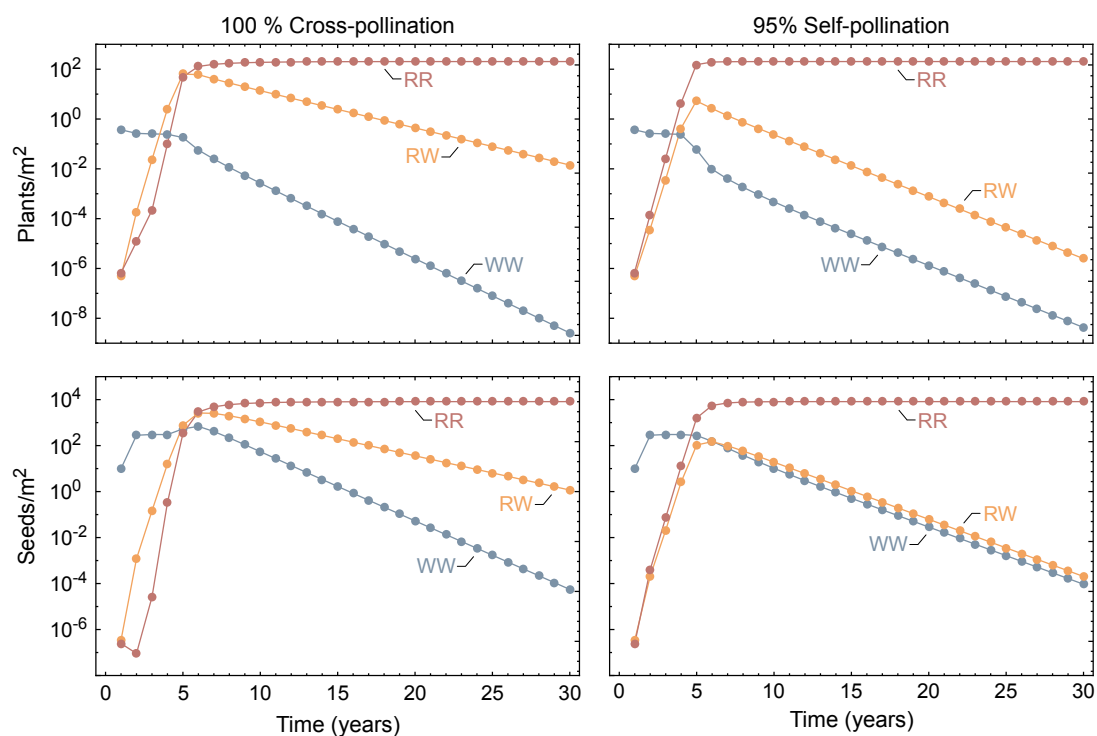


Figure A.2 | Predicted target-site resistance evolution in herbicide-treated Johnsongrass depending on self-pollination. The results are obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). Shown are predictions of our deterministic model for the genotype composition of Johnsongrass plants (\tilde{P}) and seeds (B) over 30 years of herbicide application for pure cross-pollination and 95 % self-pollination. The frequency of sensitive plants (WW) is shown in blue, resistant heterozygotes (RW) in yellow and resistant homozygotes (RR) are represented in red.

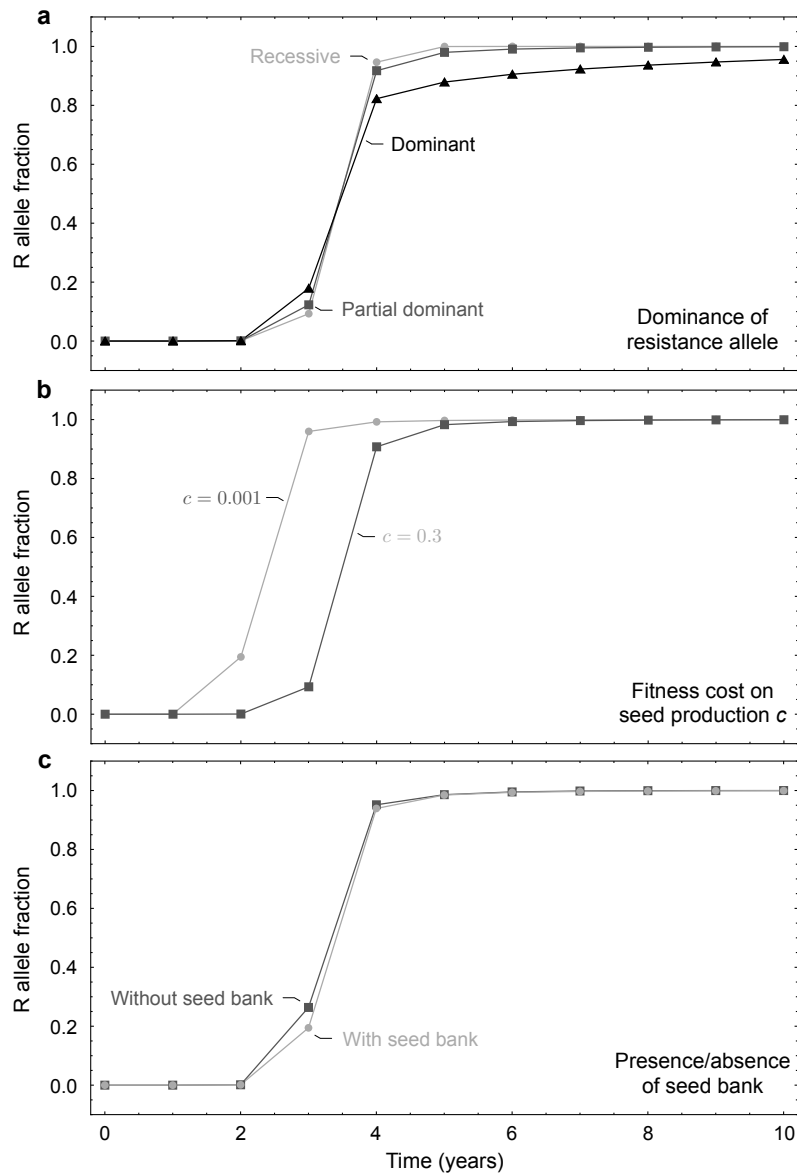


Figure A.3 | Predicted target-site resistance evolution in herbicide-treated Johnsongrass for different degrees of resistance dominance, low and high resistance cost and depending on seed bank formation. Shown are predictions of our deterministic model obtained for a partially dominant resistance allele ($k_h = 0.5$) (varies in **a**) and fitness cost ($k_c = 0.5$). **a**, Changes in frequency of the resistance allele R in Johnsongrass plants (\tilde{P}) under herbicide application for different degrees of resistance dominance (k_h). Where recessive corresponds to $k_h = 0$ (light grey line with closed circles), partially dominant to $k_h = 0.5$ (dark grey line with closed squares) and dominant to $k_h = 1$ (black line with closed triangles). **b**, Changes in frequency of the resistance allele R in Johnsongrass plants (\tilde{P}) under herbicide application for low ($c = 0.001$) and high ($c = 0.3$) resistance cost. The initial genotype composition differs between the low (light grey line with closed circles) and high (dark grey line with closed squares) fitness cost (compare Fig. 2.2 b). **c**, Changes in frequency of the resistance allele R in Johnsongrass plants (\tilde{P}) under herbicide application depending on the formation of a seed bank. The light grey line with closed circles corresponds to a population with a seed bank and the dark grey line with closed squares to a population without a seed bank.

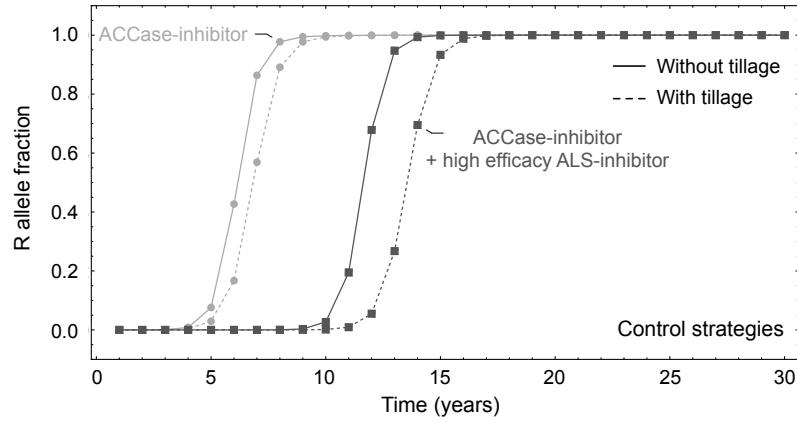


Figure A.4 | Predicted target-site resistance evolution in herbicide-treated Johnsongrass for different control regimes. Shown are deterministic predictions for the changes in the frequency of the resistance allele R in Johnsongrass plants (\tilde{P}) under different control strategies. The distinct control strategies are: ACCase-inhibitor (light grey line with closed circles) or ACCase-inhibitor and ALS-inhibitor with equal efficacy (dark grey line with closed squares) applied solely (solid line) or combined with tillage (dashed line). These dynamics were obtained with a different version of our population-based model. Compared to the model presented in this manuscript, this model does not include density dependence in 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 %. The implementation with all parameter values is available on [GitHub](#).

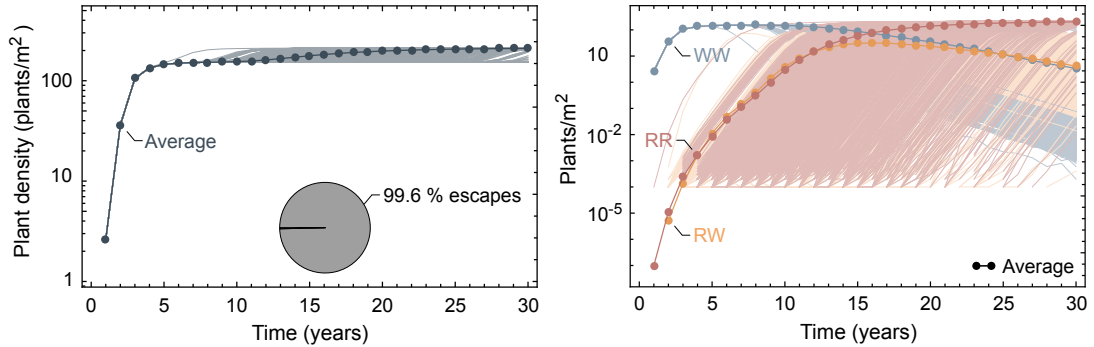


Figure A.5 | Simulated population dynamics and target-site resistance evolution in Johnsongrass treated with low efficacy ALS-inhibitor. Shown are changes in Johnsongrass density (\tilde{P}/A) and genotype composition of plants (\tilde{P}) over 30 years of ALS-inhibitor application with low efficacy of 1000 simulation runs obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). The thick lines with closed circles correspond to the average of all simulation runs, and the thin lines represent the individual realisations. The frequency of sensitive seeds (WW) is shown in blue, resistant heterozygotes (RW) in yellow and resistant homozygotes (RR) are represented in red. The pie charts display the proportion of simulation runs where the weed population escapes from control and regrows due to herbicide resistance evolution.

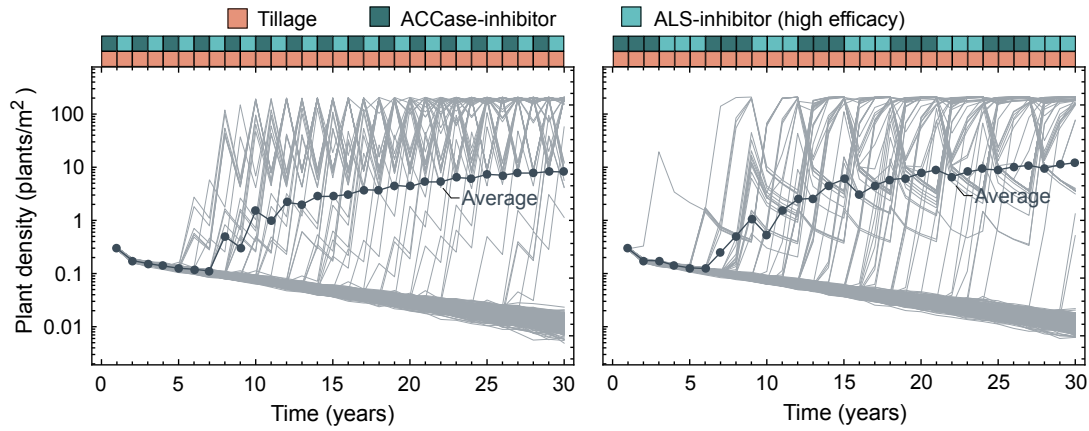


Figure A.6 | Simulated population dynamics in Johnsongrass for annual and three-annual herbicide rotations. The simulation results are obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). The left panel shows changes in Johnsongrass density (\tilde{P}/A) over 30 years for a yearly rotation of ACCase- and ALS-inhibitor combined with soil tillage. Population dynamics under a rotation of the same herbicides with a cycle length of three years combined with tillage are displayed in the right panel.

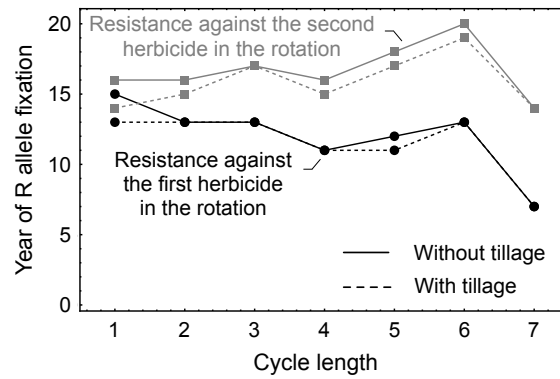


Figure A.7 | Predicted fixation time of target-site resistance in Johnsongrass under binary herbicide rotations. The results are obtained for a partially dominant resistance allele ($k_h = 0.5$) and fitness cost ($k_c = 0.5$). Considered are rotations of two herbicides with distinct target sites, ACCase- and ALS-inhibitors, Johnsongrass is known to develop target-site resistance towards. We assume equal efficacy here. The cycle length refers to the number of years one herbicide is recurrently applied before the treatment switches to the other herbicide. The year of resistance allele fixation is when the resistance allele frequency in plants (\tilde{P}), obtained from the deterministic model, reaches 99.5 %. The black lines with closed circles show the fixation of target-site resistance against the first herbicide in the rotation, and the grey line with closed rectangles resistance fixation against the second herbicide applied, where the herbicides are applied without mechanical control (solid line) or combined with tillage (dashed line).

B Appendix—Assessing the probability and time of weed control failure due to herbicide resistance evolution

This chapter is the appendix of [Chapter 3](#) and published as such in the preprint. Lauenroth, D., and Gokhale, C.S. *Assessing the probability and time of weed control failure due to herbicide resistance evolution*. bioRxiv (2024). DOI: [10.1101/2024.04.26.591266](https://doi.org/10.1101/2024.04.26.591266).

Supplementary figures

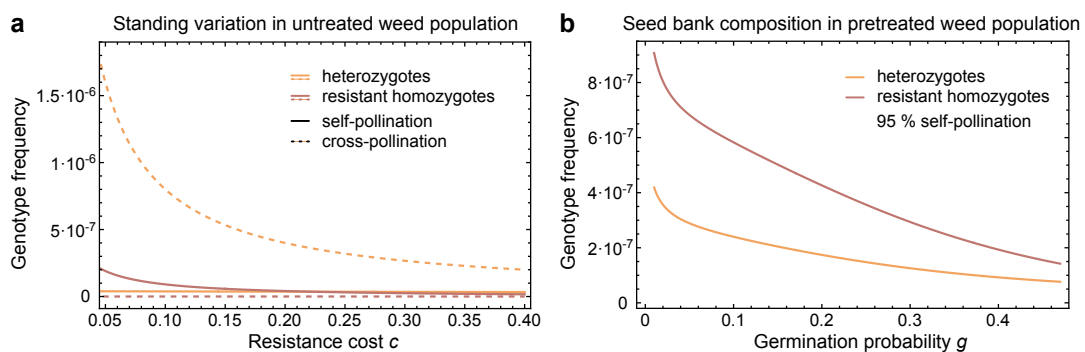


Figure B.1 | Standing genetic variation for target-site resistance. We used a deterministic density-dependent model (LAUENROTH et al., 2023) to estimate the standing variation for target-site resistance. The relative frequency of heterozygotes is displayed in orange and the relative frequency of resistant homozygotes is shown in red. **a**, Standing variation in weed populations never exposed to the herbicide before. We simulated over 1000 years without management interventions to achieve mutation-selection balance. The equilibrium frequencies are shown for fully self-pollinating populations as solid lines and for cross-pollinating populations as dashed lines. **b**, Seed bank composition of a weed population previously treated with the herbicide. We simulated one year with herbicide application followed by ten years without any control measures, reporting the final seed bank composition. The figures use the default parameter set as defined in [Table B.1](#) except when specified otherwise.

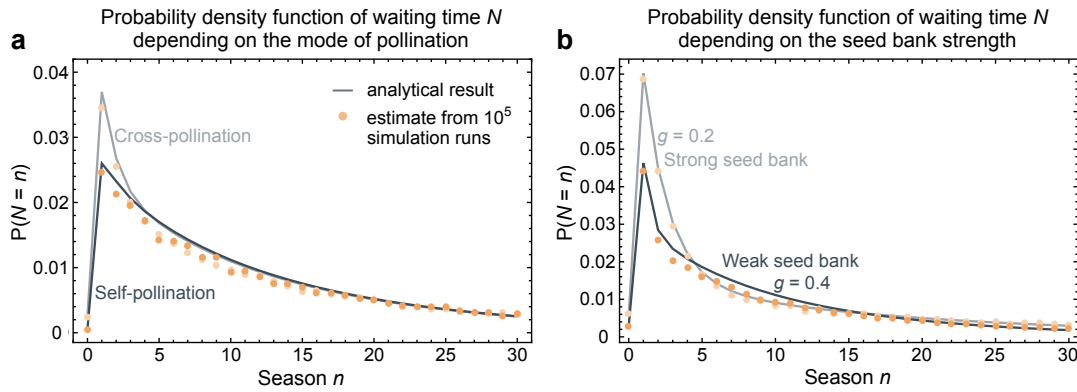


Figure B.2 | Waiting time until the first resistant plant appears. The probability mass functions of the time N until the first appearance of a resistant plant were derived analytically from Equation (3.15), shown as grey lines. The closed orange circles are estimates from 10^5 simulation runs. **a**, Waiting times in cross- and self-pollinated weed populations. The waiting time distribution is shown for cross-pollinated populations as a light grey line with closed light orange circles and for self-pollinated populations as a dark grey line with closed orange circles. **b**, Waiting times in weed populations with varyingly strong seed banks. We simulated a previous herbicide treatment followed by ten years without any control measures and used the results as the start population compositions. The dark grey line with closed orange circles shows the probability mass function of waiting time under a weaker seed bank with a germination probability of $g = 0.4$, and the light grey line with closed light orange circles corresponds to the waiting in populations that have a stronger seed bank with a germination probability of $g = 0.2$. The seed bank mortality was adapted to maintain the total expected number of sexually generated offspring, which one plant has, constant. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

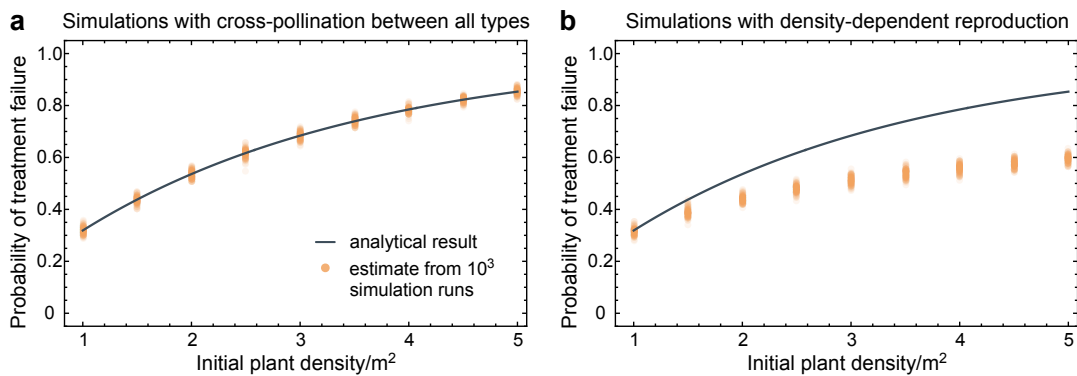


Figure B.3 | Impact of density-dependence and mating between resistant plants on the probability of treatment failure. We display the variation of the rescue probability with the initial population density. The grey lines show the probability of treatment failure derived analytically from Equation (3.9), not considering any density effects on reproduction and matings between resistant plants. The closed orange circles are estimates from 10^3 simulation runs with 10^2 replicates each. The initial seed density was 80 times the initial plant density. **a**, Impact of cross-pollination between resistant plant types on the probability of treatment failure. We show estimates from simulations comprising cross-pollination between all genotypes. **b**, Impact of density-dependent reproduction on the probability of treatment failure. We show estimates from simulations comprising density-dependent reproduction. The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

Supplementary methods

Branching process model

Offspring generating function for Sorghum halepense. We illustrate our modelling framework here on the example of *Sorghum halepense* (Johnsongrass). The perennial weed Johnsongrass is a highly competitive invasive species, able to reproduce sexually via seeds and asexually through rhizomes (PEERZADA et al., 2023; SCHWINNING et al., 2017; WARWICK et al., 1983). The plants are dormant throughout the winter, overwintering as seeds and rhizomes in the ground (WARWICK et al., 1983). At the beginning of a growing season, seeds germinate to produce seedlings, and buds on the rhizomes sprout to generate shoots (PEERZADA et al., 2023). Mature plants produce new rhizomes and fresh seeds (PEERZADA et al., 2023; WARWICK et al., 1983). We illustrate the life cycle of Johnsongrass in Figure 3.1 a.

The numbers of rhizome buds and seeds produced by a plant of genotype i are assumed to be Poisson distributed with mean b and $(1 - c_i) f$, respectively. Seeds might die over the winter with a given probability d_S . In contrast to LAUENROTH et al. (2023), here we assume that buds on a rhizome survive the winter independently of each other with a probability $(1 - d_Z)$. Therefore, the numbers of surviving rhizome buds and seeds produced by a plant of genotype i follow again Poisson distributions with means $(1 - d_Z) b$ and $(1 - d_S) (1 - c_i) f$ (ACHARYA et al., 2014). The seeds germinate in the subsequent spring independently of each other to produce seedlings with probability g and similarly shoots sprout from the buds on a rhizome with probability g_Z . Non-germinated seeds stay in the seed bank, unaffected by potential herbicide application. Seedlings and shoots can die due to herbicide application before reaching reproductive age at given efficiencies h_L and h_T , respectively. We illustrate this representation of Johnsongrass' life cycle in our model in Figure 3.1 b. The numbers of plants reaching the reproductive stage that originated from a single plant of genotype i through asexual propagation and sexual reproduction are consequently Poisson distributed with means $b(1 - d_Z) g_Z (1 - h_{Tj})$ and $(1 - c_i) f(1 - d_S) g(1 - h_{Lj})$ and independent of each other (ACHARYA et al., 2014). As the herbicide efficiency might differ between different genotypes, the type-specific efficiencies on seedlings and shoots are denoted by h_{Lj} and h_{Tj} .

Likewise, the number of seeds in the next season's seed bank generated by a type i plant is independent of its plant offspring and follows a Poisson distribution with mean $(1 - c_i) f(1 - d_S) (1 - g)$. While we assume plants only produce rhizome buds of their genotype, seeds might have a different genotype following simple Mendelian inheritance and could include mutations. Let $\mathbf{M}(j)$, $j \in \{1, 2, \dots, m\}$, be the set of inheritance matrices, where the entries $M_{ik}(j)$ give the proportions of type j seeds produced by a plant of genotype i pollinated by type k pollen. Plants self-pollinate at a proportion p_{self} , corresponding to the pollen-producing plant being of the same genotype as the mother plant ($k = i$). We assume that pollen is abundant enough that pollination results in complete ovule fertilisation, neglecting a selective advantage of self-pollination here.

With interest in the establishment of rare alleles, we assume that the sensitive homozygous type, referred to as genotype 1, is abundant, while the types carrying the resistance allele are rare. Therefore, we assume resistant plants do not cross-pollinate with other plants of these types. For type 1 plants, we consider cross-pollination by plants of all possible genotypes. The fraction of sensitive plants pollinated by pollen from a resistant plant type $i \neq 1$ in a season n is $Z_{n,1} \frac{Z_{n,i}}{Z_{n,1} + \sum_{l=2}^m Z_{n,l}} \approx Z_{n,i}$ and the fraction cross-pollinated by pollen from sensitive plants is $Z_{n,1} \frac{Z_{n,1}}{Z_{n,1} + \sum_{l=2}^m Z_{n,l}} \approx Z_{n,1}$. It is, hence, natural to count all offspring from matings between type 1 and rare type plants as the rare type's offspring. Since the inheritance does not depend on which mating partner contributes the male and which the female gametes, this altered offspring assignment does not constitute a deviation from Mendelian inheritance. Hence, the numbers of genotype j plants and dormant seeds produced through sexual reproduction by a resistant plant of genotype $i \neq 1$ are mutually independent and Poisson distributed with mean $f((1 - c_i) (M_{ii}(j) p_{\text{self}} + M_{i1}(j) (1 - p_{\text{self}})) + (1 - c_1) M_{1i}(j) (1 -$

$p_{\text{self}})) (1-d_S) g (1-h_{Lj})$ and $f((1-c_i) (M_{ii}(j) p_{\text{self}} + M_{i1}(j) (1-p_{\text{self}})) + (1-c_1) M_{1i}(j) (1-p_{\text{self}})) (1-d_S) (1-g)$, respectively. Since we consider simple Mendelian inheritance, $\mathbf{M}(j)$ is symmetric and the respective means reduce to $f(M_{ii}(j) (1-c_i) p_{\text{self}} + M_{i1}(j) (2-c_1-c_i) (1-p_{\text{self}})) (1-d_S) g (1-h_{Lj})$ and $f(M_{ii}(j) (1-c_i) p_{\text{self}} + M_{i1}(j) (2-c_1-c_i) (1-p_{\text{self}})) (1-d_S) (1-g)$. The numbers of type j plants and dormant seeds a type 1 plant has as sexually generated offspring in the next generation are independent and follow Poisson distributions with means $M_{11}(j) f(1-d_S) g (1-h_{Lj})$ and $M_{11}(j) f(1-d_S) (1-g)$.

These approximations of the cross-pollination allow us to capture sexual reproduction in the asexual Galton-Watson process and to obtain analytical and numerical results. Since the extinction probabilities of processes starting with a resistant plant type are very close to zero, the survival of a weed population under herbicide control depends almost exclusively on the establishment of resistant plants (see Section **Results**). The weed survival probability is not notably affected by cross-pollination between resistant types (Figure B.3 a). For the sexual multitype Galton-Watson process, all theoretical results were derived exclusively for superadditive mating functions (FRITSCH et al., 2022; HULL, 1998). However, random mating in a population of several types results in a non-superadditive mating function, not allowing any analysis of the process but solely simulations.

Seeds that did not readily germinate in the first spring after their production die before the following spring with probability d_B . Those seeds that stayed in the seed bank germinate in spring with probability g . Accordingly, a seed in the seed bank that is of genotype i produces one type i plant in the next season with the probability $(1-d_B) g (1-h_{Li})$ and by staying dormant one seed of genotype i – itself – with probability $(1-d_B) (1-g)$. $(d_B + (1-d_B) g h_{Li})$ gives the probability of a seed disappearing from the seed bank without having any offspring in the next generation.

Knowing the offspring distributions, we can now specify the parameters of the offspring-generating functions for Johnsongrass. For

$$f^i(\mathbf{s}) = \exp \left(\sum_{l=1}^{2m} \lambda_{il} (s_l - 1) \right), \quad i \in \{1, 2, \dots, m\},$$

the generating functions of the plant's offspring,

$$\begin{aligned} \lambda_{ij} &= (M_{ii}(j) p_{\text{self}} S_i + M_{i1}(j) (1-p_{\text{self}}) (S_i + (1-\delta_{1i}) S_1)) g (1-h_{Lj}) + \delta_{ij} T (1-h_{Tj}), \\ \lambda_{im+j} &= (M_{ii}(j) p_{\text{self}} S_i + M_{i1}(j) (1-p_{\text{self}}) (S_i + (1-\delta_{1i}) S_1)) (1-g), \quad i, j \in \{1, 2, \dots, m\}, \end{aligned}$$

are the parameters of the respective Poisson distributions with $T = b(1-d_Z) g_Z$ and $S_i = (1-c_i) f(1-d_S)$. For the offspring-generating functions of seeds,

$$f^{m+i}(\mathbf{s}) = \alpha_i s_i + \beta s_{m+i} + \gamma_i, \quad i \in \{1, 2, \dots, m\},$$

the parameters are given by $\alpha_i = (1-d_B) g (1-h_{Li})$, $\gamma_i = d_B + (1-d_B) g h_{Li}$, $i \in \{1, \dots, m\}$, and $\beta = (1-d_B) (1-g)$.

Expected population over time. The offspring generating function $\mathbf{f}(\mathbf{s}) = (f^1(\mathbf{s}), f^2(\mathbf{s}), \dots, f^{2m}(\mathbf{s}))$ can be used to derive the matrix of first-order moments \mathbf{A} with entries

$$\begin{aligned} a_{ij} &= \mathbb{E}(Z_{1,j} \mid \mathbf{Z}_0 = \mathbf{e}_i) = \mathbb{E}(X_{ij}) = \lim_{\mathbf{s} \uparrow \mathbf{1}} \frac{\partial f^i(\mathbf{s})}{\partial s_j} = \lambda_{ij}, \\ a_{m+i,j} &= \mathbb{E}(Z_{1,j} \mid \mathbf{Z}_0 = \mathbf{e}_{m+i}) = \mathbb{E}(X_{m+i,j}) = \lim_{\mathbf{s} \uparrow \mathbf{1}} \frac{\partial f^{m+i}(\mathbf{s})}{\partial s_j} = \begin{cases} \alpha_i & j = i, \\ \beta & j = m+i, \\ 0 & \text{otherwise,} \end{cases} \\ i &\in \{1, 2, \dots, m\}, j \in \{1, 2, \dots, 2m\}. \end{aligned}$$

For given genotypes $i, j \in \{1, 2, \dots, m\}$ the entries a_{ij} and $a_{i,m+j}$ correspond to the expected numbers of genotype j plants and seeds a single plant of genotype i leaves in the subsequent season. Likewise, $a_{m+i,j}$ and $a_{m+i,m+j}$ give the expected counts for genotype j plants and seeds originating from one genotype i seed in the preceding season, where $a_{ij}, a_{i,m+j} \leq 1$.

Obviously $\mathbb{E}(\mathbf{Z}_1 \mid \mathbf{Z}_0 = \mathbf{e}_i) = \mathbf{e}_i \mathbf{A}$ holds. Using (3.1) we can more generally express the expectation of \mathbf{Z}_{n+1} , the population in season $n+1$, conditioned on the population of the previous season \mathbf{Z}_n as

$$\begin{aligned} \mathbb{E}(\mathbf{Z}_{n+1} \mid \mathbf{Z}_n) &= \mathbb{E}\left(\sum_{i=1}^k \sum_{l=1}^{Z_{n,i}} \mathbf{X}_i^{(l)} \mid \mathbf{Z}_n\right) \\ &= \sum_{i=1}^k \sum_{l=1}^{Z_{n,i}} \mathbb{E}(\mathbf{X}_i^{(l)}) \\ &= \sum_{i=1}^k Z_{n,i} \mathbb{E}(\mathbf{X}_i) = \mathbf{Z}_n \mathbf{A}, \quad n \in \mathbb{N}_0. \end{aligned}$$

Taking the expectation with respect to \mathbf{Z}_n yields a recurrence formula for the expected population in season $n+1$ when the initial population is given by \mathbf{Z}_0 ,

$$\mathbb{E}(\mathbf{Z}_{n+1}) = \mathbb{E}(\mathbf{Z}_n) \mathbf{A} = \dots = \mathbf{Z}_0 \mathbf{A}^{n+1}, \quad n \in \mathbb{N}_0. \quad (\text{B.1})$$

Number of mutations. We can derive the number of new mutants in a similar way as AZEVEDO et al. (2021). Let the random variable $K \in \mathbb{N}_0$ describe the total number of distinct resistant plant lineages generated by *de novo* mutation in the lineage of a sensitive plant (type 1). Further, let ν denote the expectation of K . We first need to show that $\nu < \infty$. Consider a two-type Galton-Watson process $(\tilde{\mathbf{Z}}_n)_{n \in \mathbb{N}_0}$ of sensitive plants and seeds, starting with a single plant. The matrix of first-order moments is given by

$$\tilde{\mathbf{A}} = \begin{pmatrix} S_1 g(1 - h_{L1}) + T(1 - h_{T1}) & S_1(1 - g) \\ \alpha_1 & \beta \end{pmatrix},$$

with spectral radius $\rho < 1$ (for the parameter set given in Table B.1, $\rho \approx 0.935$). Let Y_{total} denote the total number of seeds produced in the lineage of a sensitive plant and Y_n the number of seeds produced in season n . Since we only consider mutations occurring in seed production, the total

number of new mutants arising is lower than the total number of produced seeds

$$\begin{aligned}\nu < \mathbb{E}(Y_{total}) &= \sum_{n=0}^{\infty} \mathbb{E}(Y_n) \\ &= \sum_{n=0}^{\infty} \mathbb{E}(\tilde{Z}_{n,1}) S_1 \\ &= S_1 \sum_{n=0}^{\infty} (\tilde{A}^n)_{11} < S_1 \sum_{n=0}^{\infty} 2 \rho^n = 2 S_1 \frac{1}{1-\rho},\end{aligned}$$

where the third equality follows from Equation (B.1) and the last inequality is a consequence of the Perron-Frobenius Theorem (HARRIS, 1963, pp. 37-38).

Let us return to our usual branching process with $2m$ types. Conditioning ν on the offspring generated by a sensitive plant, we get the relation

$$\begin{aligned}\nu &= \mathbb{E}(K) = \sum_{\mathbf{x} \in \mathbb{N}_0^{2m}} \mathbb{E}(K \mid \mathbf{X}_1 = \mathbf{x}) \mathbb{P}(\mathbf{X}_1 = \mathbf{x}) \\ &= \sum_{\mathbf{x} \in \mathbb{N}_0^{2m}} \left(\nu x_1 + \tilde{o}_1 \nu x_m + 1 + \sum_{i=2}^m x_i + \tilde{o}_i x_{m+i} \right) \prod_{j=1}^{2m} \mathbb{P}(X_{ij} = x_j) \\ &= \nu \mathbb{E}(X_{11}) + \tilde{o}_1 \nu \mathbb{E}(X_{1m+1}) + \sum_{i=2}^m \mathbb{E}(X_{1i}) + \tilde{o}_i \mathbb{E}(X_{1m+1}) \\ &= \nu \lambda_{11} + \tilde{o}_1 \nu \lambda_{1m+1} + \sum_{i=2}^m \lambda_{1i} + \tilde{o}_i \lambda_{1m+1},\end{aligned}$$

where $\tilde{o}_i = (1 - d_B) o_i$ with $o_i = g(1 - h_{Li}) \sum_{k=0}^{\infty} ((1 - d_B)(1 - g))^k = \frac{g(1-h_{Li})}{1-(1-d_B)(1-g)}$ being the proportion of genotype i seeds that successfully establish a plant on the field. The third equality follows from the independence of the Poisson distributed offspring, and the fourth equality holds because of the absolute convergence of the series ($\nu < \infty$). The expected number of new mutants generated in the lineage of a sensitive individual is then given by

$$\nu = \frac{S_1 \sum_{i=2}^m M_{11}(i) o_i}{1 - S_1 M_{11}(1) o_1 - T(1 - h_{T1})}.$$

The total number of resistant plants generated by *de novo* mutation in a process starting with Poisson distributed numbers of seeds and plants equals

$$\begin{aligned}\nu_{total} &= \mathbb{E}\left(Z_{0,1} \nu\right) + \mathbb{E}\left(Z_{0,m+1} \tilde{o}_1 \nu\right) \\ &= \nu v_1 (n_P + \tilde{o}_1 n_B).\end{aligned}\tag{B.2}$$

On the other hand, the total contribution of the standing genetic variation to resistant plant lineages appearing on the field is

$$\omega = \sum_{i=2}^m v_i (n_P + \tilde{o}_i n_B).\tag{B.3}$$

Since $\tilde{o}_i < \tilde{o}_{i+1}$ for $i \in \{1, 2, \dots, m-1\}$, we can derive an upper bound for the ratio of resistant

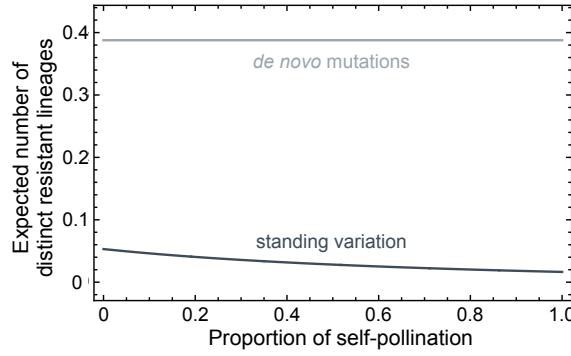


Figure B.4 | Origin of resistant lineages. We show the effect of self-pollination on the contributions of *de novo* mutations ν_{total} (light grey line) and standing genetic variation ω (dark grey line) to the resistant plant lineages appearing on the field. The expected numbers of distinct resistant lineages were derived analytically from Equations (B.2) and (B.3). The figures use the default parameter set as defined in Table B.1 except when specified otherwise.

plant lineages emerging in the field that results from *de novo* mutation vs. standing genetic variation

$$\frac{\nu_{total}}{\omega} = \frac{\nu v_1 (n_P + \tilde{o}_1 n_B)}{\sum_{i=2}^m v_i (n_P + \tilde{o}_i n_B)} < \frac{\nu v_1 (n_P + \tilde{o}_1 n_B)}{\sum_{i=2}^m v_i (n_P + \tilde{o}_1 n_B)} = \frac{\nu v_1}{\sum_{i=2}^m v_i}.$$

With the parameter set given in Table B.1, *de novo* mutations contribute approximately 22 times more resistant plant lineages than the standing genetic variation. However, mutants from the standing variation typically establish earlier, providing an advantage compared to resistant lineages resulting from mutations that appear later in the declining population. Generally, evolutionary rescue from standing variation is faster and requires a smaller population size than rescue from *de novo* mutations (ORR et al., 2014). While the expected number of resistant lineages resulting from *de novo* mutations ν_{total} is primarily independent of the level of self-pollination ($\nu_{total} \approx 0.388$), the contribution of the standing variation ω is determined by the pollination mode ($\omega \approx 0.016$ in a purely self-pollinating population and $\omega \approx 0.053$ in a fully cross-pollinated population) (Figure B.4). While new mutations arise unaffected by the source of pollen, self-pollination increases the level of homozygosity in a population, reducing the frequency of standing variants for target-site resistance as resistance alleles cluster in homozygotes. If the mutation is highly recessive, the increased homozygosity, however, yields more resistant individuals compared to outcrossing populations.

Computer simulations

We used simulations to analyse the impact of density effects and cross-pollination between resistant plants on the obtained probability and time of herbicide resistance evolution. We implemented our Galton-Watson model with modifications in the sexual reproduction. Our simulations allowed cross-pollination between all possible genotypes and included density-dependent fecundity (as per Eqs. (11) and (12) in LAUENROTH et al. (2023)). The plant and seed offspring of type i plants followed Poisson distributions with population-dependent means

$$\begin{aligned} \hat{\lambda}_{ij}(n) &= \left(M_{ii}(j) p_{\text{self}} + (1 - p_{\text{self}}) \sum_{l=1}^m \frac{Z_{n,l}}{\sum_{r=1}^m Z_{n,r}} M_{il}(j) \right) \hat{S}_i g (1 - h_{Lj}) + \delta_{ij} \hat{T} (1 - h_{Tj}), \\ \hat{\lambda}_{i m+j}(n) &= \left(M_{ii}(j) p_{\text{self}} + (1 - p_{\text{self}}) \sum_{l=1}^m \frac{Z_{n,l}}{\sum_{r=1}^m Z_{n,r}} M_{il}(j) \right) \hat{S}_i (1 - g), \quad i, j \in \{1, 2, \dots, m\}, \end{aligned}$$

with $\hat{T} = 0.93 b_{\max} (1 + \frac{0.1}{A} \sum_{r=1}^m Z_{n,r})^{-1} (1 - d_Z) g_Z$ and $\hat{S}_i = (1 - c_i) 0.93 f_{\max} (1 + \frac{0.1}{A} \sum_{r=1}^m Z_{n,r})^{-1} (1 - d_S)$.

Parameterisation

The results presented in this study are for a single locus in a diploid genome with two alleles – a sensitive allele and an allele conferring herbicide resistance. The sensitive genotype corresponds to genotype 1, the heterozygotes are referred to as genotype 2, and genotype 3 is the homozygous resistant genotype.

Standard parameter set

Table B.1 shows the parameter set of our model with the standard values used for this study. We fixed the parameters displayed in black throughout the whole study. The grey parameters varied in some figures, but take the values indicated here if we do not state them explicitly. We considered incomplete dominance of resistance and the associated fitness cost, such that the type-specific herbicide efficacy and fitness costs are $c_1 = 0$, $c_2 = 0.5c$, $c_3 = c$ and $h_{*1} = h_*$, $h_{*2} = 0.5h_*$, $h_{*3} = 0$.

Inheritance matrices. We suppose simple Mendelian inheritance with mutations in both directions occurring with probability μ . The inheritance matrices in the sexual reproduction of plants are then given by,

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

$$\begin{aligned} \mathbf{M}(2) = & 2\mu(1 - \mu) \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix} + ((1 - \mu)^2 + \mu^2) \begin{pmatrix} 0 & 0.5 & 1 \\ 0.5 & 0.5 & 0.5 \\ 1 & 0.5 & 0 \end{pmatrix} \\ & + 2\mu(1 - \mu) \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0.25 & 0.5 \\ 0 & 0.5 & 1 \end{pmatrix}, \end{aligned}$$

$$\mathbf{M}(3) = \mu^2 \begin{pmatrix} 1 & 0.5 & 0 \\ 0.5 & 0.25 & 0 \\ 0 & 0 & 0 \end{pmatrix} + \mu(1 - \mu) \begin{pmatrix} 0 & 0.5 & 1 \\ 0.5 & 0.5 & 0.5 \\ 1 & 0.5 & 0 \end{pmatrix} + (1 - \mu)^2 \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0.25 & 0.5 \\ 0 & 0.5 & 1 \end{pmatrix},$$

where the entries $M_{ik}(j)$ give the proportions of type j seeds produced by a plant of genotype i pollinated by type k pollen.

Table B.1 | Model parameters and their values. Parameters in black are fixed in all calculations and simulations. The parameters displayed in grey are varied in some parts and take the values given here if not stated differently. Detailed explanations and references are given in the Supplementary Information of LAUENROTH et al. (2023).

Model parameter	Symbol	Value
Sprouting probability	g_Z	0.2
Germination probability	g	0.3
Fitness cost on seed production	c	0.3
Proportion of self-pollination	p_{self}	0.95
Mutation rate	μ	10^{-8}
Herbicide efficacy		
on seedlings	h_L	0.998
on shoots	h_T	0.985
Maximum rhizome bud number produced per plant	b_{max}	140
rhizome bud number produced per plant ^a	b	$0.9 b_{\text{max}}$
Maximum viable seed number produced per plant	f_{max}	13,000
viable seed number produced per plant ^a	f	$0.9 f_{\text{max}}$
Rhizome winter mortality	d_Z	0.35
Seed loss		
fresh seeds	d_S	0.94
old seeds in seed bank	d_B	0.48
Initial density (m^{-2})		
seeds		80
plants		1
Field size (m^2)	A	10,000

^a In the simulations, the limits of seed and rhizome bud numbers produced per plant are $0.93 f_{\text{max}}$ and $0.93 b_{\text{max}}$ to compensate for the fecundity reduction by $(1 + \frac{0.1}{A} \sum_{i=1}^m Z_{n,i})^{-1}$ apparent even at extremely low densities (LAUENROTH et al., 2023).

Acknowledgements

I feel fortunate to have had only excellent and supportive supervisors on my scientific path.

First, I especially want to thank Chaitanya Gokhale for his guidance and constant support over the past few years. As a caring and encouraging group leader, he created an enjoyable work environment. I remember him once saying, ‘I guess I am not important enough to have enemies.’ As I told Chaitanya then, his kindness and open mind make it easy to like him. Seeing him finding inspiration in all kinds of topics and building new connections everywhere he goes is genuinely inspiring.

I also want to express my gratitude to Karsten Keller, who has encouraged me to do a doctorate and even pointed me to the position with Chaitanya. He was an exceptional teacher and caring mentor during my studies at Lübeck University. Although Karsten could no longer contribute directly to the work presented in this thesis, his lectures and guidance over the years built its foundation.

I want to thank all members of my Research Group *Theoretical Models of Eco-evolutionary Dynamics* – known as *the Unicorns* – for being the most amazing colleagues, office mates and friends. Thank you for being there, helping me through the lows of my doctoral studies, and making the highs feel even higher.

I had a wonderful time at the MPI in Plön and am incredibly grateful to everyone who contributed – to the friends I made on the way, to fantastic colleagues, and to the team behind the scenes that keeps the Institute running. Special thanks go to Arne Traulsen and all current and former members of the *Department for Theoretical Biology* for creating such a positive, welcoming and supportive work environment.

I can not thank my family and friends enough. None of this would have been possible without their unconditional love and support.

