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Seed banking strategies and host-parasite coevolution

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Vollstandiger Abdruck der von der Fakultät
Wissenschaftszentrum Weihenstephan fur Ernahrung, Landnutzung und Umwelt
der Technischen Universitat Munchen zur Erlangung des akademischen Grades eines

Doktors der Naturwissenschaften (Dr. rer. Nat.)

genehmigten Dissertation.

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Die Dissertation wurde am 08.05.2018 bei der Technischen Universität München eingereicht und durch die Fakultät Wissenschaftszentrum Weihenstephan für Ernährung, Landnutzung und Umwelt am 27.06.2018 angenommen.

To my brothers,
always.

Acknowledgements

It is with a lot of emotions that I finally write a few words to properly thank all of the people who helped me and gave me so much support throughout my PhD.

First and foremost thank you Aurélien for trusting me with this project. I cannot thank you enough for the amount of freedom you gave me. At times it has been slightly overwhelming, but that is exactly what I needed to become confident in my knowledges and make this project my own. Thank you so much for your kindness, which is unfortunately a quality largely underrated in academia, and your constant positivism. You always trusted me to get results and you gave me the time to achieve it, this saved me a lot of time doubting myself and kept me motivated. Also thank you for never pointing out my chaotic spatial organisation…

I wish to thank Hanno for agreeing to be the chair person of my examining committee and Prof. Dr. Hans-Peter Comes and Dr. Frédéric Hamelin for agreeing to review my work.

Thank you so much Etienne (Rajon), you gave me so many advices since I left for Germany. You responded to all my questions, and always found time to discuss my doubts about my future. I am also really happy that I participated to my first international conference with you, it was a big step for me. If I had not cross your path during my Master, I would not be where I am right now.

Also thank you Frederic (Menu) for introducing me to the world of “bet-hedging”. While attending your enthusiastic lectures and listening to your many stories about the chestnut weevil, I would have never imagined that one day, I would myself write about bet-hedging theory.

Looking back to my work, I realise that I truly shared my time between only 4 tasks: wondering why my system never works, why my system is never stable, why it can be stable or unstable, and finally how to make it even more unstable. It was not easy for me to communicate about my issues, but I found a lot of support in this group going through my stability drama, which at times tend to affect my mood.

Firstly Hanna, you have been a wonderful office mate, workshop and conference companion, “stability” counsellor, “how to paint a ceiling” advisor and “how to cycle to work” teacher. I really enjoyed all our discussions, definition debates, volley ball games and most of all, every silly talk we had around a coffee (or a cocktail). I will miss you.

Daniel, meeting you brought so much fun in my life. Thank you for providing me with an unlimited supply of chocolate and perhaps too many bubbles during our shared office times. You have helped me so much outside of work, kept track with my administrative records and

sometimes even managed to find a slight interest in my work. Thank you for lightning up my time here, it has been quite a cookie ride.

Remco, first of all you have an amazing sense of humour, but mostly thank you a lot for the motivation talks, especially this last year, and helping me to prepare letters and interviews for postdoc. Your help was invaluable.

I wish to thank all the members/past members of the lab Silke, Daniela, Gustavo, Di, Thibaut, Sona, Saurabh, Amarylis, Drazen and of course Jean Tristan, oh dear, I will never forget the fun we had in bars. We were also joined by many nice students, bringing a lot of fun during or coffee breaks, especially Michaela, Unai, Rebecca and her beautiful dog Alice. Petting a dog is definitely the best cure to an article rejection.

I will never forget that my arrival in Germany has been made very easy thanks to you Florence. You literally took care of everything when I arrived, but also introduced me to Hind and Guillaume. Speaking of, Hind thank you for your invaluable support.

I should also thank Ariane for sharing many skype sessions with me, and despite her young age actively participating in the meetings. I however, do not support you in pursuing a career in spoon percussion.

I am so grateful for Pauline, Agathe, Thérèse, Thibaut and Marine, my Masternelle group of friends that supported me every day of this journey. I am so proud of each of us. Also Maud (and Lexie) thank you so much for always finding time to meet me when I am back at home.

I want to address a special thank you to Ron for bearing with me while I was writing and arranging me a desk space wherever we were. You patiently listened to my never-ending to do lists and even made your first step in science helping me with my Zusammenfassung. For everything, thank you.

En dernier lieu je tiens à remercier ma famille. Je ne pense pas que beaucoup de frère irait jusqu'à apprendre une nouvelle langue juste parce que leur petite sœur déménage à l'étranger. Merci pour tout ton soutien Mikael, je pense que tu le sais mais notre entente parfaite est l'une des choses qui compte le plus pour moi. Un grand merci aussi à tes enfants Mathis et Mila, si drôles, gentils et intelligents, qui rendent la vie tellement belle. Je tiens aussi à remercier ma grand-mère pour ses nombreuses lettres qui m'apportent tant de joie et pour être si présente simplement. Pour finir je souhaite remercier mes parents, pour leur soutien constant et m'avoir permis de poursuivre toutes mes passions et envies sans avoir jamais douté de moi. Merci pour tout.

Summary

Seed banking, the storage of seeds (eggs or other dormant stages) in the soil, is a life history strategy common to many plants, insects and crustaceans playing a significant role in the survival of species in natural environments. For annual plants, this strategy consists in producing seeds expressing varying timing of emergence within and between years, so that seedlings encounter variable environmental conditions over time. Therefore, if environmental conditions happen to be unfavourable, such as severe drought in a given year preventing seedlings to establish, an amount of seeds remains available in the soil for the next generations. Seed banking is expected to evolve when the occurrence of unfavourable conditions is not predictable, and such life history strategies evolving in response to environmental stochasticity are defined as "bet-hedging" strategies. The aim of this thesis is to explore host-parasite coevolutionary dynamics as a novel cause for the evolution of seed banking as a temporal bet-hedging strategy.

Cycles of coevolution promote a gradually changing environment for hosts and parasites, which drives and is driven in return by changes in the seed banking strategy. Via a modelling approach, we study the evolution of host germination as a quantitative adaptive trait using different simulation methods. We test how the genetic interaction assumed between hosts and parasites, namely the Gene-For-Gene (GFG) and Matching-Allele (MA) models, influences the evolution of the seed banking strategy. We also investigate how the age specific seed recruitment and the and the persistence of the seed bank considered do influence the coevolutionary dynamics. We additionally test for the effect of linkage equilibrium/ disequilibrium between the GFG and MA locus and the germination loci on the evolution of bet-hedging strategies. Finally, the evolution of seed banking in investigated in more complex models including multiple sources

of variations such as density-dependent regulation of the host population and environmental stochasticity in addition to coevolution.

We demonstrate for the first time that coevolution between hosts and their parasites can promote the evolution of several optimal seed bank strategies. The optimal bet-hedging strategy depends on the speed and amplitude of coevolutionary cycles. We also demonstrate the complex interplay between coevolutionary dynamics and the evolution of seed banking as across the ecological and evolutionary time scale. We discuss as well the generality of our results, and how to test this new body of theoretical results in several plant-pathogens systems but also in populations of *Daphnia sp.* (a crustacean) with well documented ongoing coevolution with microparasites.

Zusammenfassung

Das Samenbanking, die Lagerung von Samen (Eiern oder anderen ruhenden Stadien) im Boden, ist eine lebensgeschichtliche Strategie, die vielen Pflanzen, Insekten und Krebstieren gemeinsam ist und eine bedeutende Rolle beim Überleben von Arten in natürlichen Umgebungen spielt. Für einjährige Pflanzen besteht diese Strategie darin, Samen zu produzieren, die unterschiedliche Zeitpunkte der Emergenz innerhalb und zwischen den Jahren zeigen, so dass die Sämlinge im Laufe der Zeit auf unterschiedliche Umweltbedingungen treffen. Wenn die Umweltbedingungen ungünstig sind, wie zum Beispiel eine schwere Trockenheit in einem bestimmten Jahr, die die Keimbildung verhindert, bleibt eine Menge an Saatgut für die nächsten Generationen im Boden verfügbar. Es wird erwartet, dass sich das Saatgutbanking entwickelt, wenn das Auftreten ungünstiger Bedingungen nicht vorhersehbar ist, und solche Lebensverlaufsstrategien, die sich als Reaktion auf Umweltstochastizität entwickeln, werden als "Bet-Hedging" -Strategien definiert. Das Ziel dieser Arbeit ist es, die koevolutionäre Dynamik von Wirt und Parasiten als eine neue Ursache für die Evolution des Samenbankings als temporale Bet-Hedging-Strategie, zu untersuchen.

Zyklen der Koevolution fördern eine sich allmählich verändernde Umgebung für Wirte und Parasiten, die im Gegenzug von Änderungen in der Seed-Banking-Strategie wechselseitig angetrieben wird. Über einen Modellierungsansatz untersuchen wir die Entwicklung der Wirtskeimung als quantitatives, adaptives Merkmal unter Verwendung verschiedener Simulationsmethoden. Wir testen, wie die genetische Interaktion zwischen Wirten und Parasiten, nämlich die Gene-for-Gene- (GFG) und die Matching-Allele (MA) Modelle, die Evolution der Seed-Banking-Strategie beeinflusst. Wir untersuchen auch, wie die altersspezifische

Samenrekrutierung und die Persistenz der Samenbank die koevolutionäre Dynamik beeinflussen. Zusätzlich testen wir den Effekt von Kopplungsgleichgewicht/-ungleichgewicht zwischen dem GFG- und MA-Locus und den Keimungsloci auf die Entwicklung von Bet-Hedging-Strategien. Schließlich wird die Evolution des Samenbankings in komplexeren Modellen untersucht, die neben der Koevolution auch mehrere Variationsquellen wie die dichteabhängige Regulation der Wirtspopulation und Umweltstochastizität beinhalten.

Wir zeigen zum ersten Mal, dass Koevolution zwischen Wirten und ihren Parasiten die Evolution mehrerer optimaler Samenbank-Strategien fördern kann. Die optimale Bet-Hedging-Strategie hängt von der Geschwindigkeit und Amplitude der Koevolutionierungszyklen ab. Wir zeigen auch das komplexe Wechselspiel zwischen koevolutionärer Dynamik und der Evolution des Samenbankings auf der ökologischen und evolutionären Zeitskala. Wir diskutieren ebenso die Allgemeingültigkeit unserer Ergebnisse und wie man diese neuen theoretischen Ergebnisse in verschiedenen Pflanzenpathogensystemen, aber auch in Populationen von Daphnia sp. (ein Krebstier) mit gut dokumentierter, fortschreitender Koevolution mit Mikroparasiten, testet.

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Authors contributions

Chapter 2

M.V. and **A.T.** designed the study, performed the analytical computations, and wrote the manuscript. **M.V.** performed the numerical simulations and analysed the results.

Chapter 3

M.V. and **A.T.** designed the study. **M.V.** performed the numerical simulations, analysed the results and wrote the manuscript.

Chapter 4

M.V. designed the study, performed the numerical simulations, analysed the results and wrote the manuscript.

Introduction

1. Introduction

Natural environments vary over time and space, consequently living beings are constantly facing heterogeneous and varying selective pressures. We are for instance accustomed to seasonality or dramatic natural phenomenons such as hurricanes, flooding and fires. These physical changes define abiotic factors. In contrast, antagonistic or synergistic interactions such as predation, parasitism and symbiosis, describe the competition or cooperation, within and between individuals and species evolving in communities. These interactions define the biotic environment and can shape or be shaped by the abiotic environment.

Temporal and spatial variations have consequences on the life history characteristics of organisms; by altering reproductive success, growth and survival, such changes negatively impact the fitness of genotypes. Depending on the predictability of variations, organisms may adapt to changes in different ways. If reliable cues indicating future conditions exists, adaptive (predictive) phenotypic plasticity is expected to evolve (Cooper & Kaplan 1982; Pigliucci 2001), that is the expression of the most appropriate phenotype under specific circumstances. For instance a delay in maturity and/or smaller growth size of organisms facing environmental stress (Stearns & Koella 1986). However when variations are unpredictable, selection favours life history strategies allowing for temporal and/or spatial risk spreading, such as spatial dispersion. These strategies are called “bet-hedging strategies” (Slatkin 1974) since a bet-hedger genotype “bets” on the upcoming state of the environment.

The first theoretical models of bet-hedging were developed to study plant dormancy, which is one of the most studied life history strategy both empirically and theoretically (Cohen 1966; Venable & Lawlor 1980; Bulmer 1984; Ellner 1985a, b; Brown & Venable 1986; Venable & Brown 1988; Rees 1994). This strategy is defined by a plant dispersing its offspring through

time, by producing seeds expressing different dormancy phenotypes. Thus seeds produced at a given season will encounter different environmental conditions, maximising the fitness of the plant's genotype over generations. If seeds remain in the soil for many seasons and even years, the accumulations of seeds stored in the soil creates a seed bank. In this context the term "seed banking" strategy is preferred, although the term "prolonged dormancy" is common in the literature.

Most of the theoretical work on dormancy aims to understand how annual plants survive through temporal bet-hedging in drastic environment such as arid habitats (*e.g.* deserts, Mediterranean regions). As such it is often modelled as a succession of good and bad conditions, where reproduction and/or seed survival fails if bad conditions are met (due for example to the lack of precipitation). In such contexts the advantage of dormancy is obvious, as a bet-hedger genotype avoids extinction. However dormant life-history phases or stages are widespread among living organisms and found under a wide range of climates. Annual and perennial plants form seed banks (Pake & Venable 1996; Ferrandis *et al.* 2011), bryophytes, fungi and bacteria form spore banks (Furness & Hall 1981; Lennon & Jones 2011), while crustaceans and insects build egg banks (Menu & Debouzie 1993; Hairston 1996). Evans & Dennehy (2005) proposed the term "germ banking" to describe the global strategy in a review of theoretical and empirical studies in the light of both bet-hedging theory and the storage effect hypothesis. This latter hypothesis states that germ banks contribute to species coexistence (Chesson 1986), although conversely, species coexistence may contribute to the evolution of seed banking (Venable 1989). The aim of my thesis is to explore the evolution of host seed banking strategies under host-parasite antagonistic coevolution, a biotic source of variation never explored under the bet-hedging framework.

1.1 Bet-hedging theory

The theory of bet-hedging emerged in the 1960's from studies investigating the interaction of habitats spatio-temporal variations with phenotypic and developmental variations in populations (Cohen 1966; Boer 1968). Its designation changed over time and terms such as "risk spreading" and "risk avoiding" strategies (Boer 1968; Seger & Brockmann 1987), "escape in time and space" (Janzen 1971), "coin flipping strategy" (Cooper & Kaplan 1982), or more recently "developmental instability" (Scheiner 2014) all refer to sub-categories of bet-hedging. The definition of bet-hedging is still debated, however the central concept of the theory is easily understood with the following idiom "Don't put all your eggs in one basket"[1], an advice about the risk of failure when concentrating all your resources in a single investment. In biology terms, resources correspond to energy while investments correspond to the allocation of this energy to life history traits, such as the number of offspring produced by an organism or how often they reproduce. The central question that bet-hedging theory aims to answer is, how organism should maximise their fitness under variable and unpredictable environments?

Indeed the fitness of a genotype fluctuates through time, and is defined by the mean arithmetic fitness μ across individuals of this genotype and the variance δ^2 around this mean. A life history strategy, or an allele, reducing genotypic fitness variance δ^2 is expected to evolve under unpredictable environments (Slatkin 1974). The geometric mean fitness is thus an appropriate metric to measure the evolutionary success of a genotype, since selection is a multiplicative process and is sensitive to low values (Childs *et al.* 2010). A strict definition of bet-hedging implies that a reduction of genotypic variance comes at the cost of a reduced arithmetic mean fitness; the selected strategies realising the best trade-off between both factors (Proulx 2000; Childs *et al.* 2010).

1 Attributed to the book Don Quixote written by Miguel Cervantes in the early 1600s.

It has since been demonstrated that the trade-off is triple, between the arithmetic mean, the expected individual variance and fitness correlations among individuals (Starrfelt & Kokko 2012). Indeed reduction of fitness variance can be achieved in two ways. Either by decreasing the individual level of variance, defining conservative bet-hedging strategies, or by increasing the phenotypic variation within the offspring of an individual, defining diversified bet-hedging strategies. In the first case a generalist phenotype that does well on average is produced, for instance producing clutch of constant egg size, in the latter case several genotypes are produced at one generation, such as clutches of different egg size, each matching different environmental conditions (Seger & Brockmann 1987; Philippi & Seger 1989). Both mechanisms represent extreme ends of a continuum of strategies (Starrfelt & Kokko 2012), since a strategy can simultaneously reduce individual variance and increase phenotypic variation (see **Box 1**).

Variance reduction in genotypic fitness is achieved through time or space depending on the grain of the environment (Levins 1968). Time dispersal strategy such as iteroparity (Verin *et al.* 2017) or dormancy, also referred to as between-generation strategies are expected to evolve under coarse grained environments (Gillespie 1974, 1975; Hopper 1999). This is defined as variation occurring at very large time scale, such that an individual experiences the same environment over its life time while its offspring might encounter different conditions. In contrast, spatial dispersal strategies, or within-generation bet-hedging strategies such as partial migration or dispersed oviposition, are expected to evolve under fined grained environments (Levins 1968; Seger & Brockmann 1987), that is when an individual can experience all possible variations throughout its lifetime. However, many environments are medium-grained, and once again Starrfelt and Kokko (2012) argue that within-generation and between-generation bet-hedging actually represent two ends of a continuum.

Box 1: The classic example of bet-hedging from (Seger & Brockmann 1987), temporally varying rainfall.

In this example, the number of seeds produced by a plant is influenced by the state of the environment, which is either wet or dry with probability $P_{dry}=Pwet=1/2$. We consider haploid plants with four different genotypes: a dry-specialist A_{dry} genotype producing more seeds under dry years, the opposite wet-specialist A_{wet} producing more seeds under wet years, a conservative genotype A_{cons} producing an equal amount of seeds under each environmental state and a diversified genotype A_{divers} producing both dry and wet-year specialist morphs. Inheritance is asexual, such that offspring have the same genotype as their plant parent.

	Genotypes			
	A_{dry}	A_{wet}	A_{cons}	A_{divers}
Dry, $P_{dry}=1/2$	1	0.6	0.785	0.776
Wet, $P_{wet}=1/2$	0.58	1	0.785	0.815
Expected arithmetic fitness	0.79	0.8	0.785	**0.796**
Geometric mean fitness	0.762	0.775	0.785	**0.795**

Table 1.1*: Genotypic absolute fitnesses, the details of calculation can be found in (Seger & Brockmann 1987; Starrfelt & Kokko 2012).*

The expected arithmetic fitness μ and the geometric mean fitness of each genotype given in table 1 shows that (i) both bet-hedger genotypes have higher fitnesses than wet and dry-specialist genotypes, (ii) the diversified bet-hedger shows the highest geometric mean fitness, thus should be selected for, and (iii) bet-hedging does not always come at the cost of a reduced arithmetic fitness.

Comparing the conservative bet-hedger to both wet and dry-specialists, shows that the variance in offspring production (*i.e.* the measure of fitness here) is reduced at the individual level. Conversely by producing wet and dry-specialists morphs, the reduction of variance occurs between individuals of the diversifier genotype through a reduction of the correlation between individuals: A_{divers} plants of the two morphs have different fitness in a given reproduction season.

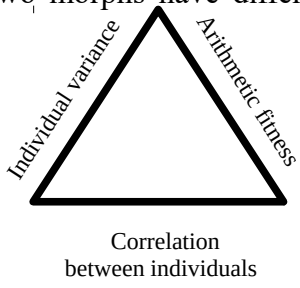

Figure 1.1*: Bet-hedging is a triple trade-off between the arithmetic mean, the expected individual variance and fitness correlations among individuals.*

1.2 Models of bet-hedging and dormancy strategies

The purpose of mathematical models in evolutionary biology is to describe and understand evolutionary changes. More specifically, bet-hedging models aim to estimate and predict how environmental variability shapes fitness changes of genotypes. Various approaches exist that I will quickly review through a history of bet-hedging models of dormancy.

1.2.1 Bet-hedging models of dormancy

Optimization models

Most of the theoretical studies regarding dormancy or developmental delay (Templeton & Levin 1979; Charlesworth 1980; MacDonald & Watkinson 1981; Bulmer 1984; Ellner 1985a, b, 1987; Tuljapurkar 1989) are based on the pioneer work of Cohen (1966). In his study focusing on annual plants in a desert environment, he developed a model investigating the optimal germination fraction of seeds *G*. This life history trait corresponds to the fraction of seeds with a non-dormant genotypes versus dormant genotypes produced by a plant. He investigated the fraction maximizing the geometric mean fitness of plants, schematically represented in **Figure 1.2** (left side), corresponding to the optimal strategy. According to his and all the studies cited above, the optimal germination fraction *G* is expected to reflect the magnitude of environmental variations, and low germination fraction (*i.e.* high ratio of dormant seeds) are expected to evolve under highly variable environments. Such optimization models consider that population sizes are infinite, hereby neglecting the effect of competition for space and resources. Strategies are thus analysed separately as the fitness of a given strategy is independent of its frequency in the population, and of the presence of other strategies within the same population.

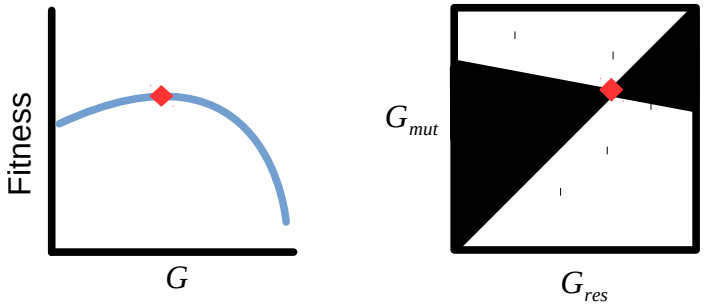

***Figure 1.2**: Graphic representations of optimal strategies. Left – Each value of the germination fraction G is plotted against the corresponding fitness of the organism, the red dot indicates the value of the trait maximising the geometric mean fitness. Right – Example of a pairwise invasibility plot (PIP), showing the result of the invasion of a mutant strategy G_{mut} (y-axis) in a resident population with strategy G_{res} (x-axis). In a large (effectively infinite) population, only mutants with trait values for which the invasion fitness is positive are able to successfully invade a resident population (black part). The red dot indicates the evolutionary stable strategy, or evolutionarily steady strategy (ESS), a strategy that can not be invaded by a mutant strategy.*

Adaptive dynamics

Including density-dependent or frequency-dependent processes requires a different mathematical framework such as adaptive dynamics (AD). The AD framework aim to understand the long-term consequences of small mutations in traits expressing a phenotype of interest (*e.g.* bet-hedging strategies). To identify the optimal strategy, the evolutionary trajectory of a trait is investigated (*i.e.* the germination fraction *G*) within the entire parameter space (*i.e.* from no seeds being dormant to all seeds being dormant) through successive processes of mutation and invasion. The evolutionary success of a mutant, which defines a new strategy, depends on the probability (i) to appear in a population, (ii) to replace the current strategy of the population (*i.e.* resident strategy), and (iii) to remain in the population.

The central hypothesis of AD is the separation between ecological times and evolutionary times. Namely, the time for a mutant to invade the population and for the population to reach demographic stability must be short, whereas the time for a new mutant to appear in a resident

population must be long enough so that only one mutant can appear at a time (Geritz *et al.* 1998). This further lead to two fundamental assumptions of AD, the population is assumed at equilibrium when a mutant appears and considering large population, the fate of a mutant can be inferred from its initial growth rate (*i.e.* the invasion fitness, usually called *sr(m)*, see Diekmann (2004) for details of the calculation). Finally the evolutionary trajectory is a sequence of successfully established mutations assuming small mutational steps, that can be studied graphically via pairwise invasibility plots (PIPs), an example is shown above (**Fig. 1.2** right side) but see Brännström et al. (2013) for a complete introduction on the utilisation of PIPs.

Moving back to the context of bet-hedging, density-dependency has been shown to amplify the influence of environmental stochasticity, thus contributing to the evolution of such strategies (Seger & Brockmann 1987; Cohen & Levin 1991; Hopper 1999; Rajon *et al.* 2009). However, several studies also demonstrated that density-dependent processes can promote sufficient temporal fitness variation for dormancy to evolve even under deterministic conditions (*i.e.* constant favourable conditions) (Bulmer 1984; Ellner 1985a; Lalonde & Roitberg 2006). As such dormancy evolves in response to other sources of variation than stochasticity, and is also advantageous to mitigate the effects of sibling competition, overcrowding and inbreeding (Westoby 1981; Kobayashi & Yamamura 2000). Studies incorporating multiples sources of variation requires more realistic and complex approaches than AD in order to explicitly model both the evolutionary process and the life cycle of organisms at the individual level.

Individual based models

Individual based models (IBM) include a clear life cycle incorporating different stages (*e.g.* juvenile, adult) and transitions (*e.g.* germination, dispersion), explicit resources dynamics (*e.g.* density-dependence), have a defined finite population size and finally involve variability

among individuals of a same stage (*e.g.* demographic stochasticity) (Uchmański & Grimm 1996).

In the context of bet-hedging, IBM allowed to investigate the expected trade-off between two bet-hedging strategies and between-population variability of bet-hedging strategies. Den Boer (1968) expected a negative correlation between bet-hedging strategies, assuming that two strategies evolving in response to the same selective pressure could not evolve jointly, as the evolution of one strategy should limit the evolution of a second one. This prediction is generally supported by theoretical studies concerning the joint evolution of temporal bet-hedging together with spatial bet-hedging (Buoro & Carlson 2014). However, the joint evolution of two temporal strategies seems likely to occur in some cases. For instance Wilbur et Rudolf (2006) demonstrated that stochastic environments favour both developmental delay (*i.e.* delayed maturity) and degree of iteroparity (*i.e.* multiple reproducing events over an organism life time), consequently selecting for “long” life-history cycles. Finally, bet-hedging strategies are expected to evolve in response to local environmental variations. IBM including a meta-population structure identified for instance that dormancy is locally adapted when spatial dispersal is restricted (Rajon *et al.* 2009; Vitalis *et al.* 2013).

In order to better reflect ecological reality, IBM show an increasing amount of complexity by assuming trade-off between life history strategies or between strategies and life history traits while including complex spatial structures (see Vitalis *et al.* 2013). However the way environmental stochasticity is modelled remains close to former models of optimisation (Cohen 1966). Although the influence of environmental autocorrelation raised interests (Rajon *et al.* 2009), the fluctuations assumed remain of extreme amplitude – with a probability of local extinction or complete reproductive failures happening at a short time scale.

1.2.2 Dormancy or Seed banking?

As we have seen above, a wide range of bet-hedging models exist and each model vary in the degree of complexity and number of assumptions. A second component of a model requiring strong assumptions is how the evolving trait or strategy is actually modelled. Most bet-hedging models are phenotype based, for instance considering dormant versus non-dormant phenotypes, hereby ignoring the underlying mechanisms and biology of dormancy. From the point of view of the strategy, dormancy corresponds to the ability of an organism to delay the germination of its offspring through time. But from a biology point of view, dormancy is defined as a state at which germination cannot occur even if conditions are favourable, thus preventing a seed to exit the soil seed bank. In that respect, dormancy takes part in generating a soil seed bank but is not the only element defining the seed banking strategy (Fenner & Thompson 2005).

Indeed the strategy of seed banking also relies on the ability of seeds to persist in the soil once maturity is reached. Seed surviving less than a year form transient seed banks, and less and more than five years, respectively form short term persistent and long term persistent seed banks (Fenner & Thompson 2005). Persistence is influenced by the physical and physiological characteristics of the seed together with the biotic and abiotic environment. Seed persistence can be viewed as a form of “resistance” to ageing, germination, predation and decay (Long *et al.* 2014). From an evolutionary point of view, plants are expected to produce an amount of seeds with characteristics (*i.e.* germination patterns, morphology) reflecting their environment (*i.e.* seasonality, predation risks) (Dalling *et al.* 2011).

The influence of dormancy on persistence is debated (*e.g.* Thompson *et al.* 2003; Honda 2008) however it is assumed to be beneficial since dormant seeds take a longer time to germinate, hereby persisting longer in soil seed banks. The most common types of dormancy are

physiological (endogenous) corresponding to hormonal inhibition and physical also described as mechanical (exogenous) where a coat isolates the embryo from water (Long *et al.* 2014). Other traits such as the seed size also influence persistence, large seed tend to be shorter lived (Bekker *et al.* 1998) but small seeds can be more easily buried in the soil explaining the positive correlations found between smaller seed and longer persistence (Hodkinson *et al.* 1998). Finally dormancy is most likely an ancestral trait of angiosperms (Willis *et al.* 2014), and few species lost the ability to form seed banks.

All together these characteristics confer an age-structure to the soil seed bank; a feature commonly overlooked by bet-hedging models following the idea that a constant germination fraction across years of the remaining seeds is optimal (Ellner 1985a). However, theoretical and empirical studies contradict this result. Philippi (1993) demonstrated that the germination behaviour is age-dependent, and that selection for an optimal distribution of germination across years is most probably weak. Secondly seedling recruitment, corresponding to the succession of seed germination, seedling survival and growth, which result in the addition of a new individual in the above ground population, is age-specific. Seedling emergence of freshly collected seeds of more than a 100 species was followed over 5 years and analyses showed that age-specific recruitment, termed seedling recruitment curve can be constant, increasing or decreasing with age depending on the species (Rees & Long 1993).

An explicit seed bank is necessary to appropriately study the evolution of temporal bet-hedging, and must assume (i) that the probability of a seed to germinate at a given time *t* depends on its age, and (ii) that the persistence of a seed in the bank is limited in time. This latter assumption has potential to strongly influence the evolution of temporal bet-hedging with regard to gradually changing environment of variable amplitude in time. Such gradual changes over

long time scales are expected, for example, from the variation of parasite pressure in time but also changes of abiotic factors due to long term climatic trends.

1.3 Host-parasite coevolution

Parasitism is a complex biotic factor of temporal variation, representing a major source of selection in natural and domesticated species. Parasites are found among many taxonomic groups (*e.g.* viruses, bacteria, eukaryotes, nematodes, arthropods and plants) and their presence at the surface on inside hosts alters the host development by reducing growth and reproduction. The fitness cost of infection results in selection for resistance mechanisms in hosts. However this selection for host resistance imposes high selection on parasite, to overcome resistance. This lead to reciprocal changes of allele frequencies over time at genes involved in the host-parasite interaction, which is referred to as coevolution (Dawkins & Krebs 1979).

Most studies assume that one or few loci with major influence govern the interaction of hosts and parasites, these genes are commonly termed resistant (R) for hosts and infective (INF) (or virulent as defined in plant pathology) for parasites (Flor 1971; Frank 1992). Other studies assume that the relation is not only based on resistant/infective genes but mediated by quantitative traits (Ridenhour & Nuismer 2007; Yang et al. 2008), more specifically the degree of phenotype matching (Nuismer *et al.* 2005) or the extent to which the phenotype of either the host or parasite exceeds that of the other (Nuismer *et al.* 2007). A classic example being plant-insects interaction mediated by the concentration of defensive compounds in the plants and detoxifying enzymes in insect (Bergelson *et al.* 2001). As many R and INF genes have been found responsible for mutual plants-pathogens recognition and cloned for many plants, see (Martin *et al.* 2003) for a complete overview, I chose to assume a relation based only on resistant/infective genes.

1.3.1 Coevolutionary scenarios

Two scenarios are discussed concerning how reciprocal changes of allele frequencies over time do occur (**Fig. 1.3**). Firstly the “arm race” scenario. An allele that confers an advantage to either hosts or parasites can potentially spread and fix in the population (Buckling & Rainey 2002), leading to recurrent selective sweeps. This corresponds to a succession of fixation events occurring either sequentially or in parallel in hosts and parasites. However, selection may also be frequency-dependent (*i.e.* balancing selection). In other words a positively selected allele can lose its advantage as its frequency increases in the population and when common be counter-selected. Allele frequency changes in the host population would then cause a corresponding allele frequency change in the parasite population and vice versa, leading to indirect negative frequency-dependent allele oscillations (niFDS). This scenario is referred to as “trench warfare”, or “red queen dynamics” (Stahl *et al.* 1999; Decaestecker *et al.* 2007). I however prefer the term “trench warfare” as I find “red queen dynamics” highly misleading. The original hypothesis of Van Valen (1973) was that changes in one species (here a host) could lead to the extinction of other species (here a parasite), such that the probability of extinction (due to coevolution) is relatively constant over millions of years and independent of the age of species. He named it "the Red Queen hypothesis," after the Chapter 2 “Through the Looking Glass” of Lewis Caroll (1872), since species had to “run” in order to stay in the same place, like Alice and the Red Queen. In my view this long term “run” involves evolution in the sense of a succession of mutation and fixation which may not be compatible with the hypothesis of balancing selection.

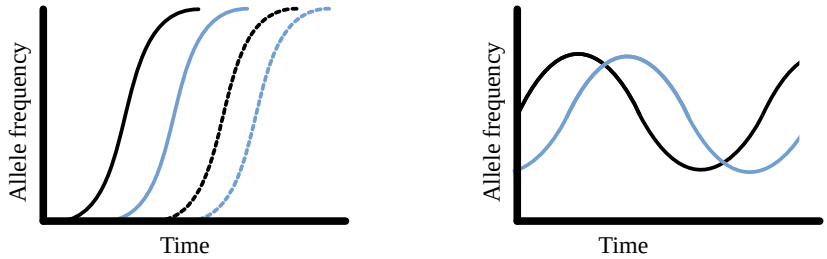

***Figure 1.3**: Dynamics of allele frequencies under host-parasite coevolutionary scenarios. Left - "arm race" scenario. Right - "trench warfare" scenario.*

1.3.2 Models of coevolution

Locus-based models

Population genetics models of host-parasite coevolution aim to reflect the genetic basis of infection, and in particular the incompatibility of interactions, in plants and animals. Classically two types of models have been proposed, gene-for-gene (GFG) models describing plant-pathogens interactions and matching alleles (MA) models describing animal-parasites interactions. The terminology of plant pathology, animal pathology and evolutionary biology is rather confusing. Here I define infectivity as the ability of a parasite to infect a host, which is named virulence in plant pathology. However I use virulence to describe the detrimental effect of parasite on host fitness, which is named aggressiveness in plant pathology.

The key feature of GFG is that genotypes vary in their degree of specialization, from specialists to generalists, such that one parasite can infect every host genotype (*e.g.* Flor 1971; Leonard 1977; Frank 1992; Thompson & Burdon 1992; Agrawal & Lively 2002; Tellier & Brown 2007; Dybdahl et al. 2014). Moreover, for each parasite locus involved in the interaction, a corresponding host locus exists. Considering single corresponding locus, hosts carry either a "susceptible" allele or a "resistant" allele. Resistant host are able to recognise parasite carrying a "non infective" allele, while parasite carrying the "infective" allele can infect the two host types (Burdon 1997). Both infective and resistant genotypes are costly generating the coevolutionary

cycles and preventing their fixation under the infinite population assumption. In a pure GFG model, hosts carrying the resistant allele are only infected by the infective parasites. If multiple loci are involved in the interaction, a host can resist a parasite if the host has a resistant allele at any locus for which the parasite has a non-infective allele (Sasaki 2000; Gilchrist N W & Sasakiz 2002; Salathé *et al.* 2005; Tellier & Brown 2007).

In opposition, MA models are based on self/non-self recognition systems in invertebrates (*e.g.* Grosberg & Hart 2000), where host and parasite genotypes are specific to one another and must match for the infection to be successful, pathogens must recognise specific proteins at the host surface to start the infection (Thrall *et al.* 2016). These two views although classically opposed, are most probably two end points of a continuum (Agrawal & Lively 2002). Along this continuum two other models are gaining interest: the inverse matching alleles model (IMA) and the inverse gene for gene models (IGFG). The IMA model is predicted on hosts having a set of recognition molecules able to bind with a specific set of molecules of pathogen antigens (similar to the vertebrate MHC system, Frank 2002). Considering the IGFG model, infection requires the host recognition by the pathogen, and the host gains resistance through the loss of the receptors targeted by the pathogen (Fenton *et al.* 2009).

Building the model

The type of interaction (GFG, MA, IGFG, IMA) is described by the infection matrix α_{ij} (see **Box 2**), in which host and parasite correspond to vectors of genotype frequencies H and P. The outcome of the interaction between each specific host i and parasite j genotypes is noted as 1 for infection or 0 for unsuccessful infection. The fitness of infected host is reduced by s representing the cost of infection (or disease severity). In some context (*i.e.* GFG),

carrying a specific genotype (*i.e.* Resistant and infective allele) comes at a cost, noted cH_i and cP_j respectively for hosts and parasites. These costs do not affect the interaction matrix.

Once the infection matrix and the coevolutionary costs are defined, the fitness of host *i* infected by a parasite of genotype *j* fitness can be written following the general equation in an infinite population size model (see **Box 3** for the derivation of a GFG model):

$W_{H(i,j)}=(1-cH_i)(1-s\alpha_{ij}W_{P(ji)})$ and the fitness of a parasite *j*,

$W_{P(j,i)}=(1-cP_j)(\alpha_{ij}W_{H(ij)})$

Epidemiology

Population genetic models, as seen in the coupled equation above, consider that the outcome of the interaction between hosts and parasites depends entirely upon the host's genes for resistance and the parasite's genes for infection, omitting epidemiological processes such as density-dependent disease transmission rate. That is when the force of infection (*i.e.* the rate at which hosts are infected) depends on the parasite prevalence (*i.e.* the proportion of infected individuals in a population) thus is function of the population density (May & Anderson 1983). Such epidemiological feedbacks lead to fluctuation of the host and parasite population sizes, outbreaks of infection, host or parasite extinctions, which strongly affect the outcome of host-parasite interactions (Boots & Haraguchi 1999; Boots *et al.* 2009).

Epidemiological models, for instance the classic Susceptible-Infected model (SI, May & Anderson 1983) describe the change of population densities with differential equations, considering continuous birth and death rate. The disease transmission rate, commonly named β, is explicitly modelled by a transmission function relating β_{ij} to the density of susceptible

host H_i (*i.e.* in the epidemiological sense that is non-infected) and the density of already infected host I_i in the population at a given time t (*e.g.* mass action).

A population genetic approach can be combined together with an epidemiological model by considering *i* strains of hosts and *j* genotypes of parasites with corresponding costs cH_i and cP_j. The outcome of infection will then depend on the type of interaction assumed and defined by the infection matrix α_{ij}. If $1 \leqslant i, j \geqslant A$ The system of equation can be written as (simplified version of May & Anderson 1983; Boots et al. 2014):

$$\frac{dH_i}{dt} = H_i\left(b_i(1-c_{Hi}) - d_i - \sum_{j=1}^{A} \alpha_{ij}\beta_{ij}(1-c_{Pj}) \sum_{k=1}^{A} I_{kj}\right)$$

$$\frac{dI_{ij}}{dt} = H_i\left(\alpha_{ij}\beta_{ij}(1-c_{Pj}) \sum_{k=1}^{A} I_{kj}\right) - I_{ij}(d_i + s_{ij})$$

Where b_i and d_i are the birth and natural death rate of hosts of genotype *i* and s_{ij} is the death rate of the host genotype *i* infected by a parasite of genotype *j*.

Box 2: Infection matrices determining the outcome of the interaction between host genotypes (rows) and parasite genotypes (columns) considering a haploid single locus models.

$$\begin{array}{c} \\ P_1 \\ P_2 \end{array}\!\!\begin{array}{c} \begin{array}{cc} H_1 & H_2 \end{array} \\ \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} \end{array} \qquad \begin{array}{c} \\ P_1 \\ P_2 \end{array}\!\!\begin{array}{c} \begin{array}{cc} H_r & H_R \end{array} \\ \begin{bmatrix} 1 & 0 \\ 0 & 0 \end{bmatrix} \end{array} \qquad \begin{array}{c} \\ P_1 \\ P_2 \end{array}\!\!\begin{array}{c} \begin{array}{cc} H_1 & H_2 \end{array} \\ \begin{bmatrix} 0 & 1 \\ 1 & 0 \end{bmatrix} \end{array} \qquad \begin{array}{c} \\ P_{ninf} \\ P_{inf} \end{array}\!\!\begin{array}{c} \begin{array}{cc} H_R & H_r \end{array} \\ \begin{bmatrix} 1 & 0 \\ 1 & 1 \end{bmatrix} \end{array}$$

MA IGFG IMA GFG

Matching infection Targeted recognition

The infection matrix gives the probability that the encounter between specific host and parasite genotypes leads to infection. Infection is noted as 1, while 0 indicates unsuccessful infection. For both matching-allele (MA) and inverse gene-for-gene (IGFG) matrices, infection is successful for matching genotypes, while a mismatch is observed as resistance. In contrast, for both inverse matching-allele (IMA) and gene-for-gene (GFG) matrices, infection is not successful when genotypes match. The IGFG matrix is given for consistency, as this model does not hold considering a haploid single locus interaction, where P2 would ultimately go extinct. Models considering diploids generate similar behaviour and dynamics (Ye *et al.* 2003).

1.3.3 Host-parasite coevolutionnary dynamics

The dynamics of a general population genetic models, such as the one derived in **Box 3**, are driven by negative indirect frequency-dependent selection (niFDS). Assuming a GFG interaction, the host/parasite coevolutionary cycle can be detailed as following:

Any increase of the resistant allele in hosts will select for the infective allele in parasite, but once resistance becomes common its cost outweighs its advantages and the allele is counter-selected. Consequently the susceptible host allele is selected for, and once it is common the cost of being infective outweighs its advantage and is counter-selected. niFDS thus maintains unstable cycling of host and parasite frequencies over time (**Fig. 1.4**, middle panel). The costs associated with carrying resistance or infectivity have consequences on the speed of the coevolutionary cycles. Increasing costs fastens the decline of resistant hosts and infective parasites, which in analogy with environmental stochasticity, is associated with drastic environments of extreme amplitudes.

In some contexts, coevolutionary dynamics can oscillate towards stable frequencies of host and parasite alleles; defining long term stable polymorphism (**Fig. 1.4**, right panel). This is due to the introduction of negative direct frequency dependence (ndFDS), such that the strength of natural selection for resistant/infective alleles declines with increasing frequency of the alleles themselves. Studies suggests that ndFDS can be reached when hosts and parasites life cycles are separated in time scale or spatial scale (Brown & Tellier 2011). Seed banking strategies on host plants, for instance, allow for time scale separation and may lead to stable polymorphism (Tellier & Brown 2009). How the characteristics of the coevolutionary cycles influence the evolution of seed banking strategies and how, conversely, the evolution of seed banking strategies affect the coevolutionary dynamics is the main focus of my thesis.

Box 3: A classic one locus GFG model

Assuming an infinite population size model describing a GFG interaction between hosts and parasites, both organisms being haploid. The generation time is discrete and there is one parasite generation per plant generation. One genetic locus with two alleles describes the GFG interaction, with the host exhibiting a resistant *R* or a susceptible *r* allele, and the parasite an infective *A* or a non-infective *a* allele. At generation *g* the resistant and susceptible hosts have respectively for frequencies R_g and r_g, and the infective and non infective parasites, respectively a_g and A_g. Carrying resistance or infectivity alleles come with the costs c_H and c_P. The cost *c* describes the strength of resistance againts the parasite *A*: if $c=1$, *R* hosts are fully resistant. Once infected, the fitness of hosts is reduced by s. The following recurrence equations describe changes in allele frequencies, as a projections of frequencies at time *g+1* from frequencies at time *g*:

$$
\begin{aligned}
R_{g+1} &= (1-c_H)(1-s(1-A_g c))R_{g-i}/\overline{W_H} \\
r_{g+1} &= (1-s)r_{g-i}/\overline{W_H} \\
A_{g+1} &= A_g(r_{g-i}+(1-c)R_{g-i})/\overline{W_P} \\
a_{g+1} &= a_g(1-c_P)(r_{g-i}+R_{g-i})/\overline{W_P}
\end{aligned}
\qquad [1]
$$

Host frequencies are scaled by the mean fitness of the host population $\overline{W_H}$, and parasite frequencies by the mean fitness of the parasite population $\overline{W_P}$, such that $R_{g+1}=1-r_{g+1}$ and $A_{g+1}=1-a\,g+1$.

The behaviour of this model is investigated in **Box 4**.

Box 4: Analysis of the dynamical system, one locus-based GFG model.

The system of equation [1] (**Box 3**) can be written in the form of difference equations, describing the change in abundance from time t to time t+1:

$$\Delta R = R_{g+1} - R_g = f(R_g, A_g)$$
$$\Delta A = A_{g+1} - A_g = g(R_g, A_g)$$

This system admits trivial equilibrium points $(R_{g+1}, A_{g+1}) = (R_g, A_g) = (\hat{R}, \hat{A})$ at which either host or parasite are absent (0,1), (1,0) or both (0,0) and non trivial equilibrium points where both are present.

Around each equilibrium point, the system can be rewritten as:

$$\begin{pmatrix} \Delta R \\ \Delta A \end{pmatrix} = J \begin{pmatrix} R \\ A \end{pmatrix} \quad \text{with} \quad J = \begin{pmatrix} a = \frac{\partial f \Delta R}{\partial R} & b = \frac{\partial f \Delta R}{\partial A} \\ c = \frac{\partial g \Delta A}{\partial R} & d = \frac{\partial g \Delta A}{\partial A} \end{pmatrix}$$

The matrix J is the matrix of partial derivatives evaluated at the equilibrium, also called the Jacobian matrix of the original system of equations. The form of J determines the stability of the dynamics near a given equilibrium point.

The eigenvalues of J (λ_1, λ_2) satisfy the characteristic equation: $\lambda^2 - tr(J) + det(J) = 0$ where $tr(J) = a + d$ and $det(J) = ad - bc$.

The discriminant of the characteristic equation is $\Delta_J = tr(J)^2 - 4\,det(J)$ and has two real roots if $\Delta_J > 0$ (i) two complex roots if $\Delta_J < 0$ (ii) or one root only if $\Delta_J > 0 = 0$ (iii). The eigenvalues of J are the roots of Δ_J:

$$\text{(i)} \quad \lambda_{1,2} = \frac{tr(J) \pm \sqrt{(\Delta)}}{2} \quad \text{(ii)} \quad \lambda_{1,2} = tr(J) \pm i\sqrt{(|\Delta|)} \quad \text{(iii)} \quad \lambda_0 = tr\frac{(A)}{2}$$

The equilibrium point is stable when λ_1 and λ_2 lie within a unit circle centred on (-1,0) on the complex plane (Roughgarden 1979).

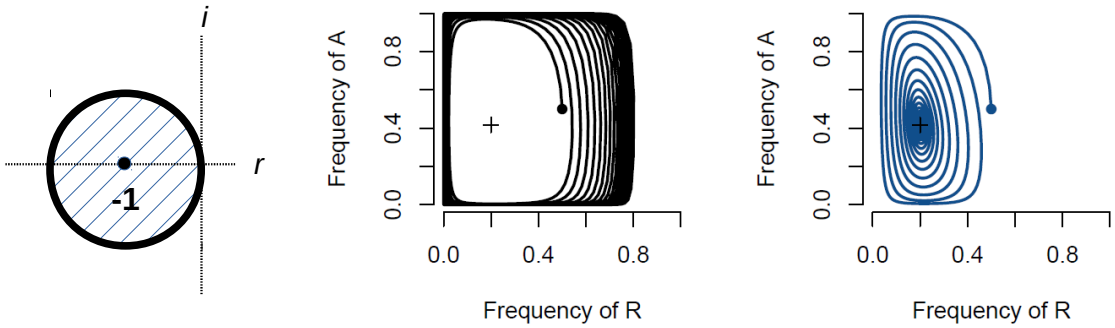

***Figure 1.4**: Unit cycle of the complex plane centred at the point -1 with corresponding stability conditions. The cross correspond to the non trivial equilibrium of the system. Outside the unit cycle, the equilibrium is unstable (middle panel), inside the cycle, the equilibrium is stable (right panel).*

1.4. Thesis outline and goal

The aim of my thesis is to explore host-parasite coevolutionary dynamics as a novel hypothesis for the evolution of seed banking in hosts as a temporal bet-hedging strategy, via a modelling approach.

Following this **introductory chapter**, the manuscript is organised in 4 chapters. Chapters 2 to 4 are each composed of an introduction, a description of the methods and corresponding results with a final discussion. The general population genetics model of host-parasite coevolution integrating the hypothesis of bet-hedging, is described in the **chapter 2**, together with analytical and simulation methods of the evolution of seed banking strategies with the corresponding results. The focus of this chapter is to study in depth the influence of the characteristics of the coevolutionary cycles on the evolution of bet-hedging strategies in resistant and susceptible hosts and how it interacts with the considered seed persistence. In the **chapter 3**, I explore eco-evolutionary feedbacks between the evolution of seed banking strategies in hosts and the host-parasite coevolutionary dynamics, arising from the introduction of a constant or increasing age-specific seed recruitment. I extend the general model of coevolution by introducing density-dependent regulation of the host population and then environmental stochasticity in the **chapter 4**, and observe how seed banking evolves under each context while assuming different age-specific seed recruitment. A complete summary of the results obtained throughout the thesis is given in the last part of the thesis **chapter 5**, followed by a discussion of future perspectives.

The **chapter 2** is accepted for publication in Evolution.

Verin, M. and **Tellier, A**. (2018), Host-parasite coevolution can promote the evolution of seed banking as a bet-hedging strategy. Evolution. Accepted Author Manuscript. doi:10.1111/evo.13483

Host-parasite coevolution can promote the evolution of seed banking as a bet-hedging strategy

Host-parasite coevolution can promote the evolution of seed banking as a bet-hedging strategy

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First published: 20 April 2018 | https://doi.org/10.1111/evo.13483

Abstract

Seed (egg) banking is a common bet-hedging strategy maximizing the fitness of organisms facing environmental unpredictability by the delayed emergence of offspring. Yet, this condition often requires fast and drastic stochastic shifts between good and bad years. We hypothesize that the host seed banking strategy can evolve in response to coevolution with parasites because the coevolutionary cycles promote a gradually changing environment over longer times than seed persistence. We study the evolution of host germination fraction as a quantitative trait using both pairwise competition and multiple mutant competition methods, while the germination locus can be genetically linked or unlinked with the host locus under coevolution. In a gene-for-gene model of coevolution, hosts evolve a seed bank strategy under unstable coevolutionary cycles promoted by moderate to high costs of resistance or strong disease severity. Moreover, when assuming genetic linkage between coevolving and germination loci, the resistant genotype always evolves seed banking in contrast to susceptible hosts. Under a matching-allele interaction, both hosts' genotypes exhibit the same seed banking strategy irrespective of the genetic linkage between loci. We suggest host-parasite coevolution as an additional hypothesis for the evolution of seed banking as a temporal bet-hedging strategy.

Keywords: Host-parasite coevolution, gene-for-gene, matching-allele, germination fraction, life-history trait evolution

AUTHORS CONTRIBUTIONS: M.V. and A.T. designed the study, performed the analytical computations, and wrote the manuscript. M.V. performed the numerical simulations and analyzed the results.

2.1 Introduction

Seed (egg) banking consists in the variation in the timing of emergence of viable seeds or eggs from a single clutch that are stored in the soil, river or lake sediments (Evans & Dennehy 2005). This life history strategy is common to many plants (Venable 1989; Philippi 1993; Clauss & Venable 2000) insects (Hanski 1988; Gourbière & Menu 2009) and crustaceans (Moustakas & Evans 2013). Only a fraction of seeds germinates each year, decreasing the population growth rate when environmental conditions are favourable while avoiding extinction when conditions are drastic. Seed banking is expected to evolve in stochastic and unpredictable environments as a temporal bet-hedging strategy (Boer 1968; Slatkin 1974; Seger & Brockmann, 1987; Philippi & Seger, 1989), spreading the risk of reproductive failure through time dispersion. This strategy is also advantageous to mitigate the negative impact of sibling competition, overcrowding and inbreeding (Westoby 1981; Kobayashi & Yamamura 2000).

In the context of bet-hedging theory, classical models investigate the evolution of the optimal germination fraction (which we denote here b_0). This fraction defines the proportion of seeds produced by a plant that germinates each season, as opposed to the proportion of seeds entering the bank ($1-b_0$) (Cohen 1966; Bulmer 1984; Ellner 1985; Valleriani 2005). According to these studies, the optimal germination fraction is expected to be low (high fraction of seeds entering the seed bank) for highly variable environments, such as variation in water availability or other abiotic factors, but also disturbance of habitats. The models cited above consider drastic environmental contexts, in particular annual plants growing in deserts, where the environment is represented as a fast succession of good and bad conditions. The reproduction and/or survival of plants is optimal during good seasons and strongly reduced (as far as null) during bad seasons.

However, seed banking plants are widespread and observed in temperate climate where environmental changes are more gradual.

Another interesting life history characteristic generating variable environments over time is the interaction between hosts and their parasites or parasitoids. It has been well documented that such interactions promote coevolutionary dynamics resulting in fluctuating selection, fixation of alleles or stable polymorphism in both species (*e.g.* (Leonard 1977; Gandon *et al.* 1996; Tellier & Brown 2007b; Ashby & Boots 2017). The dynamics are driven by negative indirect frequency-dependent selection (niFDS) – rare alleles exhibiting fitness advantages as selection in the host population depends on allele frequencies in the parasite population, and *vice versa* (Clarke 1964; Tellier & Brown 2007b). We investigate in this article the context of unstable coevolutionary dynamics, with cycles increasing over time in their amplitude and period (Leonard 1977; Tellier & Brown 2007b, 2009). These unstable cycles are therefore unpredictable for hosts and parasites in time, and their characteristics are further dependent on the genetic interaction between host and parasite. In the theoretical literature, the genetic determinism of host-parasite interactions has traditionally been modelled in two ways (but see van Baalen 1998; Gandon *et al.* 2002; Boots *et al*. 2014; Ashby & Boots 2017). Either host and parasite genotypes are specific to one another and must match for the infection to be successful (Matching allele, MA, (*e.g.* Gandon *et al*. 1996; Agrawal & Lively 2002; Dybdahl *et al*. 2014), or genotypes vary in their degree of specialization, from specialists to generalists (Gene for gene, GFG) resulting in more complex coevolutionary dynamics (*e.g.* Flor 1971; Leonard 1977; Frank 1992; Agrawal & Lively 2002; Dybdahl *et al*. 2014). We hypothesize here that unstable coevolutionary dynamics generate the unpredictable variation necessary for evolving seed banking as a bet-hedging strategy in hosts.

This specific co-evolutionary mechanism for the evolution of temporal bet-hedging has not yet been explored, while it was suggested in a review article by Hanski (1988). He indeed questioned whether cyclic or chaotic host/parasitoid and predator/prey dynamics can promote the evolution of extra long diapause of insects (*i.e.* equivalent to seed banking). Note that the reverse mechanism has been well studied, namely that prolonged diapause (or seed banking) can stabilise host-parasitoid and host-parasite dynamics (Tellier & Brown 2009, Ringel *et al*. 1998). Though, the stabilizing property relies on the germination of seeds to be geometrically distributed and the time in the bank to be unbounded, such that an egg or a seed can remain infinitely dormant with a constant germination probability (Corley *et al*. 2004). Few theoretical studies assume a bounded seed bank (of more than two years) (Templeton & Levin 1979; Valleriani & Tielbörger 2006), however, in biology, seed banks are characterised by their persistence and accordingly classified in three categories (Fenner & Thompson 2005). Transient seed banks are formed by seeds surviving less than a year in the soil before their decay. Short term and long term persistent banks are, respectively, formed by seeds surviving less and more than five years. Three characteristics compose a seed banking strategy: the germination fraction (b_0), the shape of the germination function (often assumed to be geometric) and whether the life spans of a seed is finite (bounded to a maximum value).

We propose a general model to test Hanski's intuition in the context of host-parasite coevolution and aim to demonstrate an additional mechanism generating the evolution of seed banking as temporal bet-hedging, namely the evolution of a strategy that maximises the geometric fitness of hosts under parasite pressure. Our model assumes an infinite host and parasite population, each composed of two types (resistant/susceptible and infective/non-infective), and includes a short or long term persistent bank for the host. We firstly assess that the geometric mean fitness criteria, determining which allele goes to fixation in an infinite

population model (Templeton and Levin 1979, Starrfelt & Kokko 2012) holds for models assuming the competition of two alleles (*i.e.* two hosts types). Then using both pairwise competition and multiple competition simulations of seed banking strategies, we show that distinct germination fractions emerge depending on the genetic linkage between the germination and coevolutionary loci and the considered model of genetic interaction (MA or GFG). We expose a complex interaction between the properties of the coevolutionary cycles (period and amplitude of the fluctuations) and the characteristics of the seed banking strategies. Finally, this allows us to extend previous classical results in finite size models, and to study temporal bet-hedging as an eco-evolutionary adaptation to slow but sustained biotic environmental fluctuations.

2.2 Materials and methods

2.2.1 Model description

We assume an infinite population size model describing a GFG interaction between hosts and parasites (*e.g.* Tellier & Brown 2009). For simplicity, both organisms are haploid as models with diploids generate similar behaviour and dynamics (Ye *et al.* 2003). One genetic locus with two alleles describes the GFG interaction, with the host exhibiting a resistant (*R*) or a susceptible (*r*) allele, and the parasite an infective (*INF*) or a non-infective (*ninf*) allele. At generation g the resistant and susceptible hosts have frequencies R_g and r_g respectively, and the infective and non-infective parasites, a_g and A_g respectively. Host frequencies are scaled by the mean fitness of the host population $\overline{W_H}$, and parasite frequencies by the mean fitness of the parasite population $\overline{W_P}$. Carrying resistance or infectivity alleles comes with the costs c_H and c_P. When infected, host fitness is reduced by s, the so-called disease severity. Non-infective parasites cannot infect resistant hosts. We assume that each host is exposed to parasites at each generation (Leonard 1977, Tellier and Brown 2007). The following difference equations describe changes in allele frequencies with a seed bank for the host.

$$R_{g+1}=(1-c_H)(1-s(1-A_g))[\sum_{i=0}^{min(m_R,g)} b_{Ri}(1-d)^i R_{g-i}]/\overline{W_H} \quad [1]$$

$$r_{g+1}=(1-s)[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}]/\overline{W_H} \quad [2]$$

$$A_{g+1}=A_g[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}]/\overline{W_P} \quad [3]$$

$$a_{g+1}=a_g(1-c_P)[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}+\sum_{i=0}^{min(m_R,g)} b_{Ri}(1-d)^i R_{g-i}]/\overline{W_P} \quad [4]$$

The seed bank is modelled based on a forward in time adaptation of the model from (Kaj *et al.* 2001). In general terms, a distinct quantitative locus determines the rate b_i at which seeds produced i generations ago germinate at a given generation g. The maximum amount of time seeds can remain in the bank is fixed to m. A key parameter of the seed bank is the fraction b_0 of newly produced seeds that germinate in the next generation, if b_0 =1 all seeds are non-dormant and the bank is empty. A germination function describes the relative contribution of non-dormant and dormant seeds to the above ground population, b_0 versus $b_{i\neq 0.}$ We assume a geometric memoryless function of germination, $b_i = b_0(1-b_0)^{(i-1)}$ (**Fig. S2.1**). In other words, the time spent in the bank does not affect the germination rate of the seed *per se*, but older seeds contribute less to the above ground population. Note that by definition, $\sum_{i=0}^{m} b_i = 1$ and the germination function is truncated. The germination function is applied to the resistant and susceptible hosts which thus have the respective rate and seed bank persistence b_{Ri} and m_R, and b_{ri} and m_r. Seed banking comes with a cost d, the rate at which seed dies per generation. We choose an intuitive geometric seed death rate function, so the probability for a seed to be viable and able to germinate after i generations is $(1\text{-}d)^i$. The rate of seed death, d, is identical for R and r alleles.

Our model is analogous to a modifier model (e.g. Blanquart & Gandon 2011), in which the host genome is composed of two distinct loci, either linked by being physically close on a single chromosome, or unlinked by being far apart on the same chromosome or being on different chromosomes. The modifier locus is quantitative and controls the germination strategy of the host, and the second locus is under fluctuating selection due to coevolution. We do not assume a seed bank for the parasite, its genome being reduced to the single corresponding locus under fluctuating selection.

The questions we address are 1) whether the coevolutionary dynamics select for the evolution of a seed banking strategy, 2) whether an optimal germination rate exists in that case, and 3) if the optimal strategy differs for resistant and susceptible hosts. The evolution of the seed banking strategy is the result of a change in either the germination fractions b_{R0} and/or $b_{r0,}$ or the length of the seed bank m_R and/or m_r. Both changes lead to a new distribution of the germination function b_i (based on the geometric function) and modify the characteristics of the coevolutionary cycles. We focus here on the evolution of b_0, as we later explain (and had confirmed in preliminary simulations, data not shown) that the seed bank persistence m would raise to extremely high values, that may not be physiologically relevant for a seed. Due to the feedback between the evolving germination rate b_0 and the coevolutionary dynamics, we cannot approximate analytically the period and amplitude of the unstable coevolutionary cycles and therefore we use two methods of simulations.

2.2.2 Evolution of seed banking strategy: pairwise competition

Our first method investigates the fate of one mutant, denoted b_0*, in a resident population with strategy b_0. We perform pairwise competition between the mutant and the resident drawing an analogy to pairwise invasion plots used in adaptive dynamics approaches. The mutant b_0* is introduced with frequency 0.01 in the population after a burn in phase of 20,000 generations. The burn in phase allows to replenish the seed bank and run several coevolutionary cycles. Susceptible and resistant hosts have here the same mutant (b_0 *=b_{r0} *=b_{R0} *), and resident (b_0 =b_{r0} =b_{R0}) strategies. Note however, that due to the coevolutionary dynamics a given mutant may invade only one or both host types. We perform competitions between the whole continuum of seed banking strategies, with b_0 ranging from 0.01 to 1, and simulate the dynamics for 1,500,000 generations. This time is chosen to be long enough for fixation or loss of the mutant strategy and

is much longer than that used for studying only coevolutionary cycles (Tellier & Brown 2009). We consider the mutant invasion to be successful when its mean frequency is higher than 0.9 over the last 10,000 generations. The mutant and resident strategies are denoted as coexisting when both are present with mean frequency higher than 0.1 and lower than 0.9 in this same time interval.

2.2.3 Evolution of seed banking strategy: multiple mutant competition

As an alternative method to investigate the process of successive mutation events, we perform simulations of competition between multiple mutants (see for example Boots *et al*. 2014). After a burn in phase of 20,000 generations, a number N of mutants with different strategies (different values of b_0*) are introduced in a resident population (with initial value b_0). We use here N=5 (in Boots *et al*. 2014, N=1). Each mutant has a different seed banking strategy, b_0 *, which is sampled in a Normal distribution with mean the resident value b_0 and a small standard deviation σ=0.05. The N mutants are introduced with equal frequencies summing to 0.01 (so in effect an introduction frequency of 0.01/N for each mutant). The resident and the mutants compete during a fixed number of generation T=1,000, and we then compute the geometric mean fitness over T for each strategy. The strategy with the smallest fitness amongst the $N+1$ genotypes present (N b_0* mutants and the b_0 resident) is removed from the population, and a new mutant with frequency 0.01/N is introduced. We make the assumption that hosts with the highest fitness have higher chances to produce mutants, and that the mutational step is small. Thus the new mutant strategy b_0* to be introduced is sampled in a Normal distribution with a mean equal to the population average strategy $\bar{b_0}$ and a small standard deviation σ=0.05. The

average strategy $\bar{b}_0$ is defined as the sum of the remaining strategies (N values of b_0) times their geometric mean of frequency in the population.

Two cases are investigated, (i) independence and (ii) non-independence of germination and resistance loci (*e.g.* linkage equilibrium or disequilibrium). In other words, either resistant and susceptible types have the same germination strategy which is then the strategy of the population ($b_0=b_{r0}=b_{R0}$), or these alleles can evolve each their own strategy ($b_{r0}\neq b_{R0}$). The method described above corresponds to the first case (linkage equilibrium). In the case of linked loci, we amend the above simulation protocol by removing and then adding at a given time point one resistant and one susceptible mutant. The mutant strategies are sampled around the average strategy of each type $\bar{b}_{R0}$ and $\bar{b}_{r0}$, respectively.

We investigate the evolution of seed banking under coevolution using the two simulation methods described above. We contrast the evolution of seed banking for different parameter combinations: high and low costs c_H and c_P (for values of 0.05, 0.1, 0.2 and 0.4), low to high disease severity (s=0.1, 0.3, 0.6, and 0.9) and under short term or long term seed bank persistence m (5 or 15 years). In addition, we test two hypotheses regarding the genetic linkage between the host locus for coevolution and the locus for the seed banking trait (determining the germination rate b_i). For each set of parameters, we perform 50 repetitions, and record over $2\text{x}10^6$ generations the population mean strategy $\bar{b}_0$, or the resistant and susceptible mean strategies $\bar{b}_{R0}$ and $\bar{b}_{r0}$ to account for the variability of the mutation sampling procedure. The code for simulation is available in the SI files.

2.3 Results

2.3.1 Preliminary analysis: bet-hedging in infinite population models

In infinite population models, the geometric mean fitness of a genotype determines which allele goes to fixation (Templeton and Levin 1979, Starrfelt & Kokko 2012). This criterion holds for models of competition between two alleles. Indeed an allele 1 exhibiting randomly varying fitness over time outcompetes an allele 2 with constant fitness if the geometric mean fitness of allele 1 is higher than the relative fitness of allele 2 (**SI text S1**). If both alleles show stochastic fitnesses over time but exhibit different seed banking strategies (**SI text S2**), we show that the allele reaching fixation has the strategy maximizing its geometric mean fitness. Our key assumption states that in a deterministic seed bank, the relative proportion of seeds from a given past generation depends on the population fitness at that generation. These results are partially described in the literature (see the more rigorous work by Templeton and Levin 1979). We nevertheless recapitulate them here to introduce our system of notations and extend the previous results in the finite population models by Cohen (1966) and Lewontin & Cohen (1969) to an infinite population.

Templeton and Levin (1979) further highlight the consequences of cyclically varying environments (*i.e.* deterministic environmental cycles) on the fitness of two competing alleles. They identify a key parameter: the ratio between the seed bank persistence and the period of the environmental cycles. They investigate rather short cycling periods (*e.g.* 3 years), however environmental fluctuations in temperate climate may likely vary over longer periods of time. Assuming a cyclic environment and a single host population with a seed banking strategy, we investigate the influence of the seed bank persistence m on the optimal germination fraction b_0 as

a function of the period and amplitude of environmental variations (**SI text S3**). As expected (Templeton and Levin 1979), for seed bank persistence exceeding the period of environmental variation the host population shows a high investment in the seed bank seen as a higher geometric mean fitness and a low optimizing germination fraction (**Fig. S2.2A**). However, when assuming that seed persistence is smaller than the period of drastic environmental variation, we find that low germination fraction can still be advantageous (**Fig S2.2B**). With cycles of reduced amplitude, the fitness differences between the various persistence values are drastically reduced, and low germination fractions are only likely to evolve when *m* is equal to the environmental period (**Fig. S2.2C**). In the special case of a short term persistent bank (*m*=5), we observe high optimal germination fractions corresponding to weak investment up to no (b_0 =1) investment in the bank, irrespective of the amplitude of the environmental variations (**Fig S2.2B**, and **C**).

In contrast to these cases with regular cycles, optimality theory can not be applied in our model of host-parasite coevolution, since the coevolutionary cycles show 1) increasing period and amplitude over time, and 2) these are continuously shaped by the evolution of the seed banking strategies.

2.3.2 Dynamical system behavior and seed bank

The system of equations (1) of GFG coevolution with seed banking has several equilibrium points defined for any germination function, which occur as $R_g = R_{g+1} = \hat{R}$ and $A_g = A_{g+1} = \hat{A}$. Four so-called trivial points exist as the four monomorphic outcomes (0,0), (1,0), (0,1) and (1,1) for the frequency of *R* and *ninf* (Leonard 1977). There is also one non-trivial equilibrium at which all four host and parasite alleles are maintained:

$\hat{A}=(\varepsilon-1+c_H)(1-s)/s(1-c_H)$ and $\hat{R}=\varepsilon c_P/(1+c_P(\varepsilon-1))$.

The ratio of the relative germination functions for *R* and *r* alleles including seed mortality is:

$$\varepsilon=\sum_{i=0}^{m_r} b_{ri}(1-d)^i/\sum_{i=0}^{m_R} b_{Ri}(1-d)^i,$$

which is equal to one if the resistant and susceptible alleles have the same germination function and seed bank persistence. The internal equilibrium point exists if the parameters fulfil two conditions. First, $c_H>1-\varepsilon$ meaning that the susceptible allele has a germination rate (including seed mortality) equal or slightly smaller than that of the resistant allele (as c_H is assumed to be realistically small, Leonard 1977, Tellier and Brown 2007). Second, $c_P(1-\varepsilon)<1$ which is always true based on the definitions of a gene-for-gene model (*e.g.* Leonard 1977; Tellier & Brown 2007). The value of the internal equilibrium point defines which allele has a higher frequency in host and parasite populations during the cycles. In the typical GFG parameter space, susceptible hosts and infective parasites have higher frequencies (Leonard 1977, Tellier and Brown 2007).

Only negative indirect frequency-dependence (niFDS) occurs under a geometric seed bank (Tellier & Brown 2007b, 2009), thus the internal equilibrium is unstable (**Fig. S2.3A**). This means that allele frequencies are diverging from it, and the coevolutionary cycles progressively increase in period and amplitude (**Fig. S2.3B**). As such, the unstable coevolutionary cycles are unpredictable for the plant population at a given generation. The coevolutionary parameters impact the characteristics of the unstable cycles as follows. An increase of the costs c_H (and respectively c_P) reduces the advantage of resistance (respectively infectivity), resulting in a shorter period and smaller amplitude of the cycles (see Tellier & Brown 2011, for an approximation of the period of cycles). A higher disease severity *s* also accelerates the cycles (shorter period) but increases their amplitude. Secondly, the seed bank strategy shapes the cycles;

a long seed banking persistence *m* increases the period of the cycles, as does an increase of the germination fraction b_0 (**Fig. S2.3B**).

2.3.3 Seed banking under GFG coevolution: unlinked loci

Assuming the host locus driving coevolution being independent (genetically unlinked) from the locus determining the germination fraction, means that the susceptible and resistant hosts evolve the same seed banking strategy. In this case, a striking result is the loss of the seed bank when studying the competition of multiple mutants b_0* in a resident population b_0 (**Fig. 2.1**) for moderate disease severity (s=0.3 or 0.2) and intermediate or low costs (c_H=c_P=0.2 or 0.05). This outcome is observed irrespectively of the initial resident strategy and the persistence of the seed bank *m*, with no variance between repetitions (**Fig. S2.4, S2.5**).

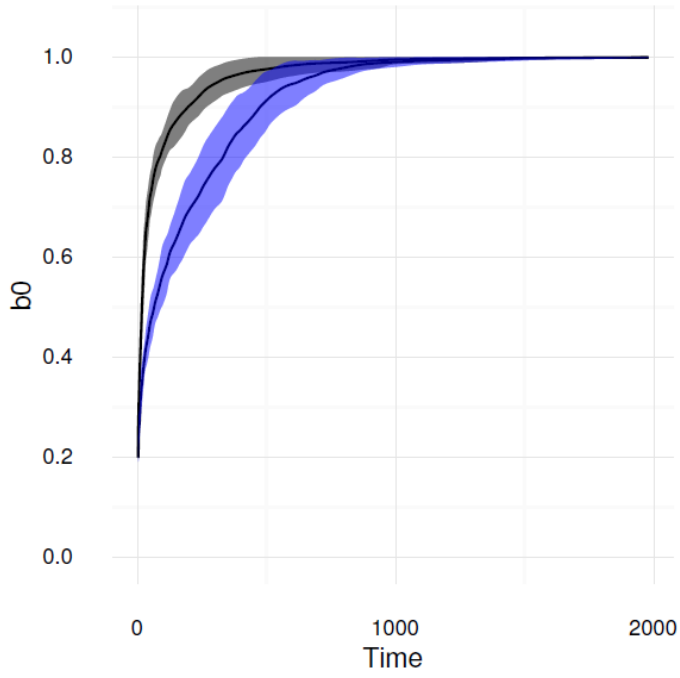


***Figure 2.1**: Evolution of the host population mean germination fraction $\bar{b}_0$ (+-sd) under multiple competition (GFG model, unlinked loci), for short term persistence m=5 (black) and long term persistence m=15 (blue) over 50 repetitions. Costs are fixed to c_H=c_P=0.2, s=0.3, and d=0.002. The initial resident value is b_0=0.2.*

2.3.4 Seed banking under GFG coevolution: linked loci

Considering non-independent (genetically linked) loci of coevolution and germination and the same coevolutionary costs as the above section, the results are fundamentally different. We observe that resistant and susceptible hosts systematically evolve towards distinct strategies. Surprisingly, a susceptible host does not appear to have any advantage in evolving a seed bank, whereas several germination strategies do evolve for the resistant host. Susceptible hosts always evolve towards a mean population strategy $\bar{b}_{0r}$ strictly equal to 1 for all persistence values (**Fig. 2.2B-C**, **Fig. 2.3B-C**), and the pairwise and multiple competition results are fully consistent with each other. We interpret this result as the absence of a seed-bank to be an evolutionary stable strategy for the susceptible host.

The outcome of seed banking evolution for the resistant host is more complex, and depends on the coevolutionary parameters and on the persistence of the seed bank. For moderate costs (c_H=c_P=0.2), the result of the pairwise competition shows a range of values, from b_{0R}=0.72 to b_{0R}=0.74, where germination strategies are able to invade and be invaded by one another (**Fig. 2.2A**). We interpret this region $0.72 < b_{0R} < 0.74$, as consisting of strategies optimising the resistant host's geometric mean fitness. This supposition is confirmed by simulations under the multiple mutants competition method (**Fig. 2.2C**, **Fig. S2.6A-B**), in which the resistant host evolves towards germination fractions ranging from $b_0 \simeq 0.62$ to $b_0 \simeq 0.88$, with a mean strategy fluctuating around b_{0R}=0.75 (**Fig. 2.2C**). Note that the boundaries of the region are wider than under the pairwise competition prediction (**Fig. 2.2A**). We explain it as a consequence of noise inherent to the sampling of mutants in the second method. Although not measured here, as we only account for the resistant mean strategy, the combination of the two approaches suggests that

polymorphism could evolve, with the coexistence of close strategies belonging to the optimising region ($0.71 < b_{0R} < 0.76$). A similar pattern is observed with the increased persistence of the seed bank (m=15 in **Fig. 2.3**), but the optimum strategy shifts towards the value b_{0R}=0.47 both for the pairwise competition method (**Fig. 2.3A**), and for the multiple mutant competitions (**Fig. 2.3C**, **Fig. S2.6C-D**), albeit being more variable in the latter. The investment in the seed bank is thus stronger with increased persistence. Here, around half of the seeds produced enter the long term bank compared to less than a quarter for a shorter bank (m=5).

Considering small costs (**Fig. S2.7-9**) the range of germination fractions strategies optimizing the resistant fitness is in line with those observed for moderate costs (**Fig. 2.2-3**), though our simulations show a greater variability around the mean resistant strategy in **Fig. S2.7C**, **S2.8C**, **S2.9** compared to **Fig. 2.2C**, **2.3C**, **S2.6**. As small costs give a greater advantage to resistant hosts, slower coevolutionary cycles are generated. Consequently, when considering resistant genotypes, the relative advantage of a given strategy against another one is small, explaining the observed variability. Furthermore, for high germination fraction values (>0.79), the frequency of resistant hosts becomes negligible (under 10^{-300}). This latter outcome is biologically unrealistic, and we exclude this range of values from our results (**Fig. S2.7-8**).

To summarize our results so far, we show the loss of the seed bank at moderate disease severity in the case of unlinked loci, while this loss is observed only for the susceptible host when considering linked loci. This is due to the fitness asymmetry between the two host types that is inherent to the GFG model and acting on two levels. Firstly, the susceptible host is constantly infected by the two types of parasites. As we demonstrated in the **SI text S1 model A3** for reduced fitness fluctuations a weak to no investment in the bank is optimal. Secondly, the susceptible host is the most common type over time, whereas the resistant host is only infected by the virulent parasite and undergoes extreme fitness fluctuations – fast increase of fitness but

of short period (**Fig. S2.3B**). Thus the evolution of the seed banking strategy is governed by the susceptible host under unlinked loci. The fitness asymmetry between resistant and susceptible genotypes depends on the different coevolutionary parameters (costs and disease severity), and is released by switching to a Matching Allele (MA) interaction.

We run simulations of multiple mutant competitions for increasing coevolutionary costs (c_H=c_P up to 0.4) together with increasing disease severity s (0.1, 0.3, 0.6 and 0.9). Each combination of parameters results in specific coevolutionary cycle characteristics, for instance the cycling periods are short for high costs c_H=c_P=0.4 in comparison with small costs c_H=c_P=0.05. In the first case, the fitness asymmetry between host types is reduced. The speed of the coevolutionary cycles has a direct incidence on the evolution of seed banking strategies (**Fig. 2.4, S2.10**) as we now observe contexts where both susceptible and resistant hosts evolve a seed banking strategy. This is observed for moderate to strong costs of resistance and infectivity (c_H=c_P>0.1) and for strong disease severity (s=0.6 and/or 0.9). In these contexts, the coevolutionary cycles are fast. Again, the investment in the seed bank is stronger for long term seed banks. The results under unlinked loci now change drastically (**Fig 2.4, S2.10**), as now the host population can evolve seed banking. Indeed the host population strategy is still mostly influenced by the susceptible host type, which can evolve b_0 <1.

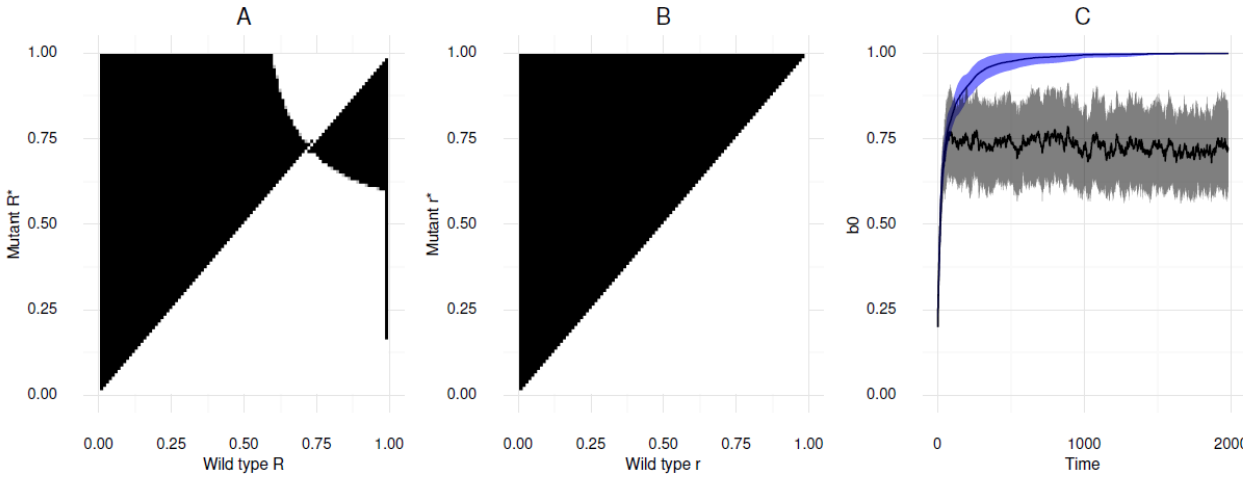


***Figure 2.2**: Pairwise invasibility plots (PIP) for GFG model with linked loci, for A) resistant and B) susceptible hosts, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed short term persistent bank m=5, and costs c_H=c_P=0.2, s=0.3, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions), inferior to 0.1 (white regions), or coexists with the resident phenotype (grey regions). C) Evolution of the mean germination fractions (+-sd) of the susceptible $\bar{b}_{r0}$ (blue) and resistant $\bar{b}_{R0}$ (black) host under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

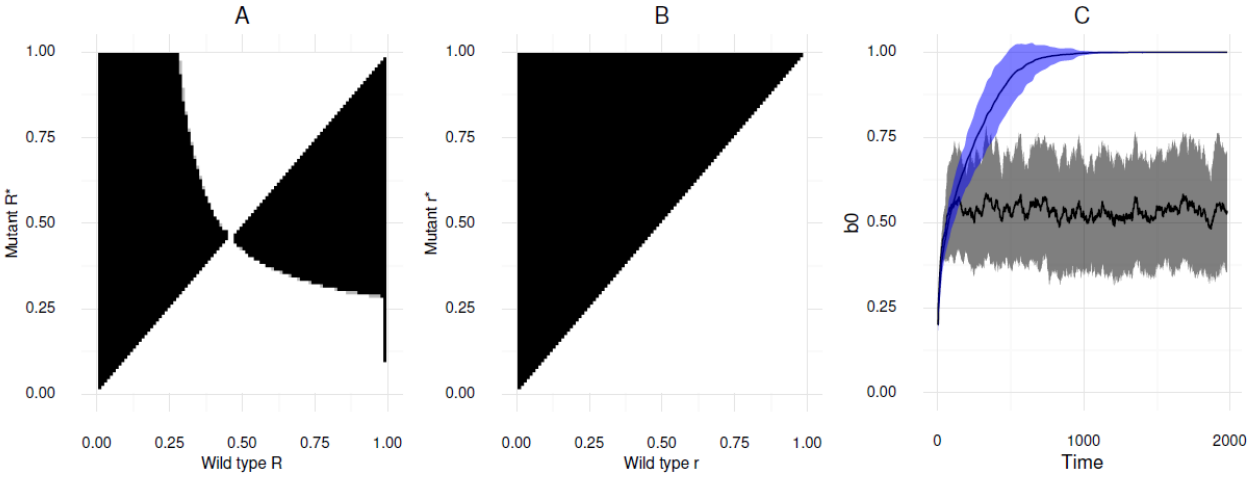


***Figure 2.3**: Pairwise invasibility plots (PIP) for the GFG model with linked loci, for A) resistant and B) susceptible hosts, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed long term persistent bank m=15, and costs c_H=c_P=0.2, s=0.3, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions), inferior to 0.1 (white regions), or coexists with the resident phenotype (grey regions). C) Evolution of the mean germination fractions (+-sd) of the susceptible $\bar{b}_{r0}$ (blue) and resistant $\bar{b}_{R0}$ (black) host under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

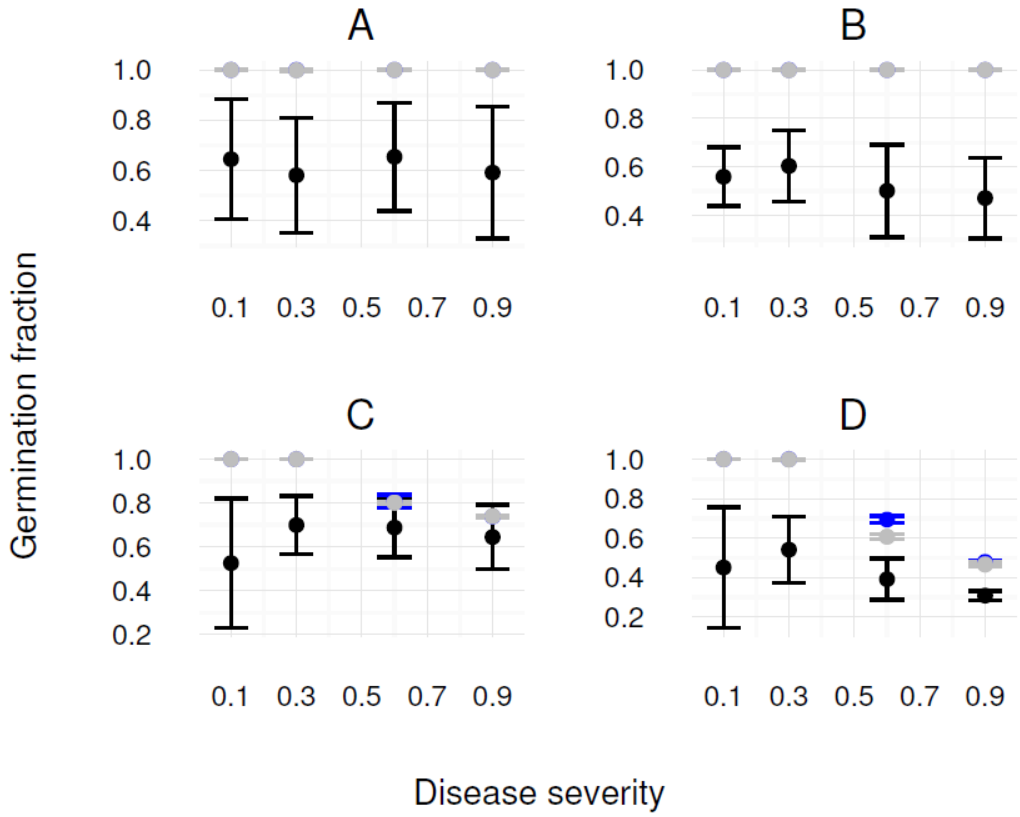


***Figure 2.4**: Population mean germination fraction (grey), resistant mean germination fraction b_{0R} (black) and susceptible mean germination fraction b_{0r} (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations) and function of the disease severity s (x-axis). The GFG model has costs of resistance and infectivity fixed to $c_H=c_P=0.05$ (A-B) and $c_H=c_P=0.2$ (C-D). The seed bank persistence m is fixed to 5 years (A-C) and 15 years (B-D). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{0R}=b_{0r}=0.2$.*

2.3.5 Seed banking under Matching allele coevolution

We modify our model to include a Matching Allele recognition matrix (**SI; text S3**) in which both hosts are infected by their specific parasite and have equivalent fitness fluctuations. We then observe that hosts evolve a seed banking strategy (**Fig. S2.11**), and moreover evolve towards the same region of optimal strategies (**Fig. S2.12-14**) depending on the persistence of the seed bank. The results are consistent for both types of simulations considered (details in **SI**). These extended results under the MA model demonstrate that the evolution of seed banking is indeed due to coevolutionary dynamics.

2.4 Discussion

Our results show that seed banking in hosts can evolve in response to parasite pressure and coevolutionary dynamics as a temporal bet-hedging strategy, especially if costs of the resistant and infectivity alleles and disease severity are strong. We have chosen not to separate between the ecological (coevolution) and the evolutionary (evolution of seed banking) time scales in our model, as there is a feedback between the evolution of the germination rate b_0 and the coevolutionary dynamics – they do not reach a stable equilibrium or limit cycles stable in period and amplitude. We therefore use simulation methods to demonstrate our hypothesis.

Host genotypes undergo different coevolutionary dynamics depending on their asymmetric or symmetric genetic interaction with parasites (GFG or MA), and as a result genotypes can evolve distinct seed banking strategies. In the GFG model, generalist (*i.e.* susceptible) hosts are found to be the most common type with higher geometric mean frequency than specialist (*i.e.* resistant) hosts. Therefore, under genetic independence of the coevolutionary and germination loci, selection is driven by the generalist host. In addition, the range of seed banking strategies evolving is shaped by the characteristics of the coevolutionary cycles (period and amplitude). Hosts evolve a strict non seed banking strategy (b_0=1) for slow coevolutionary cycles due to small costs of resistance and infectivity (c_H and c_P) together with low disease severity (s), while various strategies evolve for fast cycles (b_0<1) due to moderate to high costs and disease severity. However, under genetic disequilibrium, the resistant host always evolves seed banking while only the susceptible strategy is influenced by the speed of the coevolutionary cycles. Hosts undergoing identical period and amplitude of their coevolutionary dynamics (MA) evolve towards the same seed banking strategies (b_0<1).

The fixed seed bank persistence constrains the optimal investment in the bank. Seed banking keeps memory of past selective events, and is most effective when the temporal window covered by the seed bank length *(i.e.* persistence *m*) is large enough with regard to the period of the environmental cycles (Templeton & Levin 1979 and our results in SI text S3). For both GFG and MA models, cycles of host-parasite coevolution show periods much larger than the fixed seed bank persistence *m*. Under our GFG model, the investment in the seed bank is then stronger (*i.e.* higher fraction of seeds entering the bank) for long term persistent banks (m=15) than for short term banks (m=5); this is particularly visible for moderate to high coevolutionary costs. Decreasing the cost of the specialist genotypes or the disease severity increases the frequency of resistant hosts over time, which in return extends the periods of the coevolutionary cycles and increases the time that allele frequencies remain along the boundaries (defined as frequency of 0 or 1). Although the range of optimal germination fractions is the same as for higher costs, our simulations show hence more variation. As coevolutionary cycles are faster for MA, the investment in the bank is found to be higher (b_0=0.6 and b_0=0.4 for long and short term persistence, respectively). Host fitnesses being strictly equal with respect to the interaction with parasites, the optimal range of strategy is in this case independent of the genetic linkage. Unstable coevolutionary dynamics can therefore generate bet-hedging.

We make further predictions about the evolution of persistence. If seeds are not constrained physiologically or mechanically, persistence would probably evolve towards extreme values together with low germination fractions to maximise the temporal coverage of the cycling dynamics. An age structure due to perenniality or to a seed bank with different trade-off shapes between the age of a seed and its germination probability, does affect the coevolutionary dynamics (Tellier & Brown 2009) and would influence the evolution of the bet-hedging strategy.

Disease prevalence can also be stochastic and generate equivalent pressures as the abiotic variability usually assumed (Lewontin & Cohen 1969). A strong dependence between the environment, the disease prevalence and disease severity is a common feature to many plant and also invertebrate hosts (the so-called disease triangle in plant disease, Agrios 2005). We thus raise the question whether it is more efficient to evolve resistance to a pathogen or to invest in seed banking. In our infinite size model, assuming a stochastic disease prevalence (but fixed each year) is equivalent to the model of two competing alleles exhibiting stochastic fitness derived in the supplement (SI Text S2). As previous results for abiotic variable environments demonstrated that bet-hedging evolves only when extreme variations in fitness are observed between years (Cohen 1966, text S3), we predict that seed banking would show more benefits than resistance only for extreme stochastic prevalence together with strong disease severity, or assuming high costs of resistance. A second question regards the potential for evolving bet-hedging strategy in parasites under coevolution. Indeed bet-hedging strategies also evolve in parasites, such as low virulence in parasites transmitted by vectors in fluctuating environments (Nguyen *et al.* 2015). Thus we speculate that unstable cycles of coevolution could also generate bet-hedging in parasites, promoting the existence of dormant survival strategies within or outside hosts.

We finally derive the following predictions to test our model. Firstly, an experimental coevolution set up with phage and bacteria, could investigate whether bacteria under coevolutionary pressures evolve a bet-hedging strategy for dormancy depending on their resistance genotype (*e.g.* combining approaches by (Poullain *et al.* 2008; Beaumont *et al.* 2009; Betts *et al.* 2014). Secondly, species in disturbed habitats forming transient populations (*e.g.* metapopulations), may evolve seed banks as bet-hedging strategy for persistence and/also in trade-offs with dispersal (Vitalis *et al.* 2013). Such species may not be in tight coevolution with

pathogens, due to their inherent transient nature. However, candidate species to evolve bet-hedging in response to coevolution would be found in more stable habitats (temperate climate) which are pervasive for infections (Jousimo *et al.* 2014). In addition, for seed banking species showing more stable populations in time and a spatial structure, we predict that populations under higher pathogen pressure should exhibit higher resistance (Soubeyrand *et al.* 2009; Jousimo *et al.* 2014) alongside with longer seed banking persistence or lower germination fraction.

A key step to further test our predictions is to disentangle the effect of different selective pressures in driving the evolution of bet-hedging in space (dispersal) or in time (seed banking), even though these belong to a continuum of strategies but rely on different physiological adaptations (Buoro & Carlson 2014). Obviously, several selective pressures due to variation of biotic and abiotic factors generate the condition for bet-hedging to occur and are acting concomitantly on species, perhaps even with fluctuating strength or importance over time and space. Coevolution, as we described, is a slow mechanism, probably acting at longer evolutionary scales (over several cycles) than drastic environmental stochasticity, but we expect that combinations of several variable abiotic and biotic factors would accelerate the evolution of bet-hedging.

The choice for a modelling approach assuming fixed population size is dictated by the core assumption of bet-hedging theory: the environmental unpredictability. Our model ensures that coevolutionary dynamics are strictly driven by niFDS, leading to unstable cycles, which constitute an unpredictable environment for hosts. However, our results may not apply to all host-parasite systems because our current framework dismisses epidemiological and population dynamics and the potential for eco-evolutionary feedbacks. The latter are known to generate direct frequency-dependent selection, resulting in coevolutionary dynamics exhibiting stable

limit cycles or damping off towards a stable polymorphic internal equilibrium point (Tellier & Brown 2009; Ashby & Boots 2017). As such, an epidemiological approach cannot generate the evolution of bet-hedging in the strict sense (Boer 1968), and is outside the scope of this study. Epidemiological dynamics produce strong eco-evolutionary feedbacks when parasites survive only within their hosts and either kill them to be transmitted or induce large fitness damage to their host (e.g. Gokhale *et al.* 2013, Ashby & Boots 2017). Considering the diversity of parasite life cycles and life history traits as well as the influence of the abiotic environment on host-parasite interactions, it is conceivable that strong eco-evolutionary feedbacks may not always apply. As a consequence, short term epidemiological models may not accurately predict the longer term coevolutionary dynamics and cycles over thousands of generations, the time scale at which seed banking strategies may evolve. The simple model based on fixed population sizes and longer time scale we use here represents a first necessary step in order to disentangle the effect of coevolutionary dynamics *sensu stricto* (*i.e.* changes in allele frequencies in interacting species) from that of eco-evolutionary feedbacks on the evolution of seed banking. Further studies should include the effect of population dynamics and epidemiology in our framework, to investigate not only if seed banking evolves under such conditions, but also the interaction between epidemiological dynamics and the buffering effect of the seed bank on population sizes.

AUTHORS CONTRIBUTIONS:

Supplementary informations

Content:

- **Supplementary figures**
- **Text S1**: Model A1, Stochastic fitness in infinite population size.
- **Text S2**: Model A2, Advantage of seed banking in infinite population size.
- **Text S3**: Model A3,: Optimal seed banking strategy with environment fluctuations of constant amplitude and periods.
- **Text S4:** Model B, Host-parasite interaction model with Matching Allele.

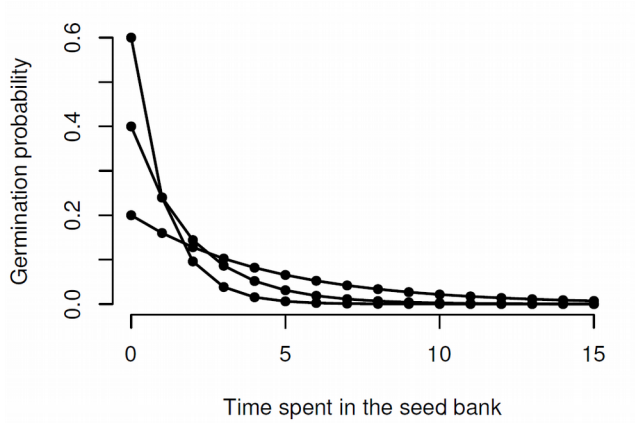


Figure S2.1*: Geometric germination function for varying germination fraction* b_0 *(0.6, 0.4 and 0.2, in x-axis at time 0) and a fixed seed bank persistence m of 15 years. The germination probability of a seed is independent of its age but the contribution of a dormant seed to the above ground decreases with the time spent in the bank.*

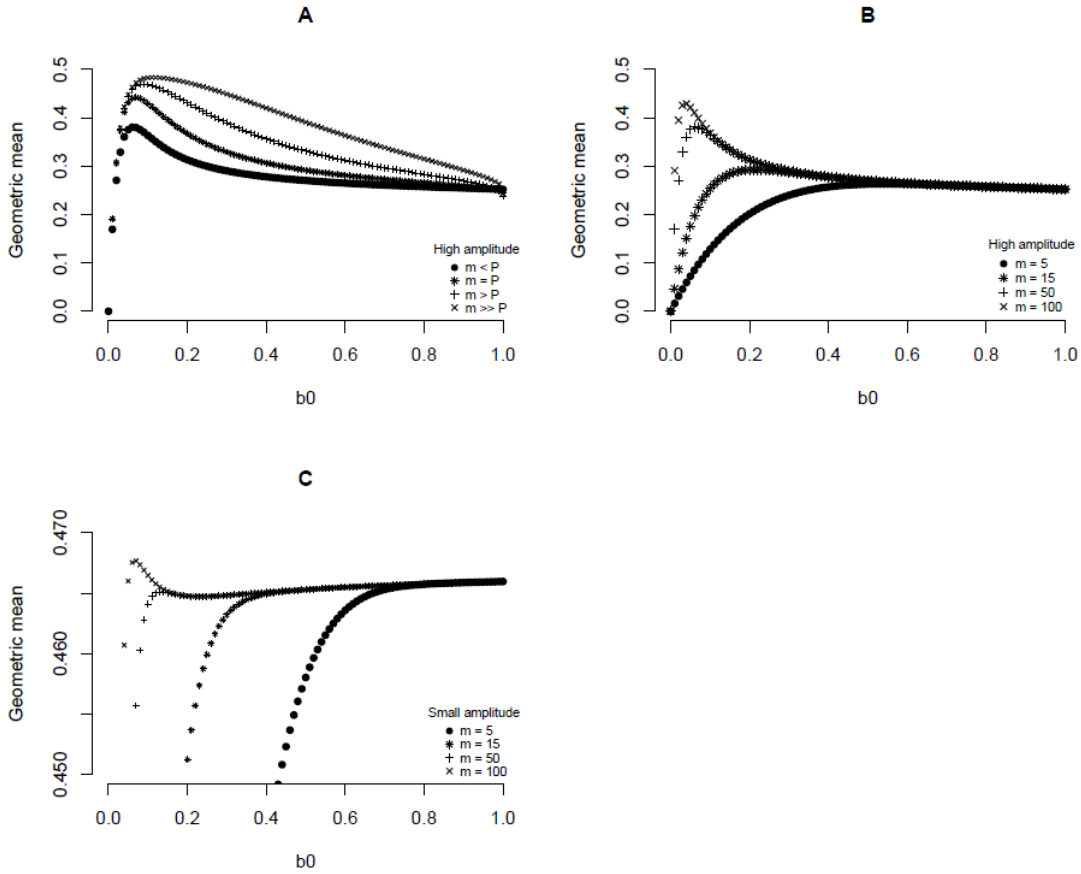


Figure S2.2*: Host geometric mean fitness (y-axis) over the continuum of germination fraction (x-axis), under a cycling environment of constant cycling frequency and period. A) The seed bank persistence m is fixed to 50 generations, the environment cycles have high amplitudes (drastic) and periods of 100, 50, 25 and 12.5 generations. B) The environment cycles have high amplitudes (drastic) and the period is fixed to 100 generations, the seed bank persistence m is 5, 15, 50, 100 generations. C) The environment cycles have small amplitudes (mild) and the period is fixed to 100 generations, the seed bank persistence m is 5, 15, 50, 100 generations.*

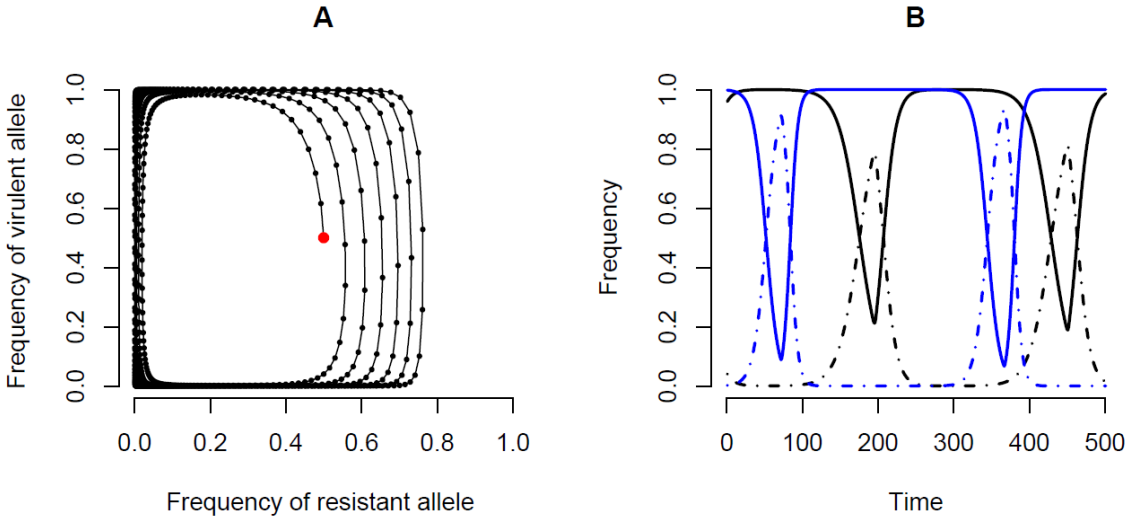


***Figure S2.3**: Examples of coevolutionary dynamics under the GFG model. A) Dynamics of relative allele frequencies over time, with a host seed bank persistence m=5 and a germination fraction* $b_0=0.5$*, for costs* $c_H=c_P=0.2, s=0.3$ *and d=0.002. Initial frequencies (red point) are* $R_0=a_0=0.5$*. The model is run for 3,000 generations. The allele frequency spiral outward the internal equilibrium point. B) Susceptible host (solid line) and resistant host (dot-dashed line) relative frequencies over time, with a seed bank persistence m=5 and germination fractions* $b_0=0.5$ *(black) and 0.9 (blue) for costs* $c_H=c_P=0.2, s=0.3$ *and d=0.002.*

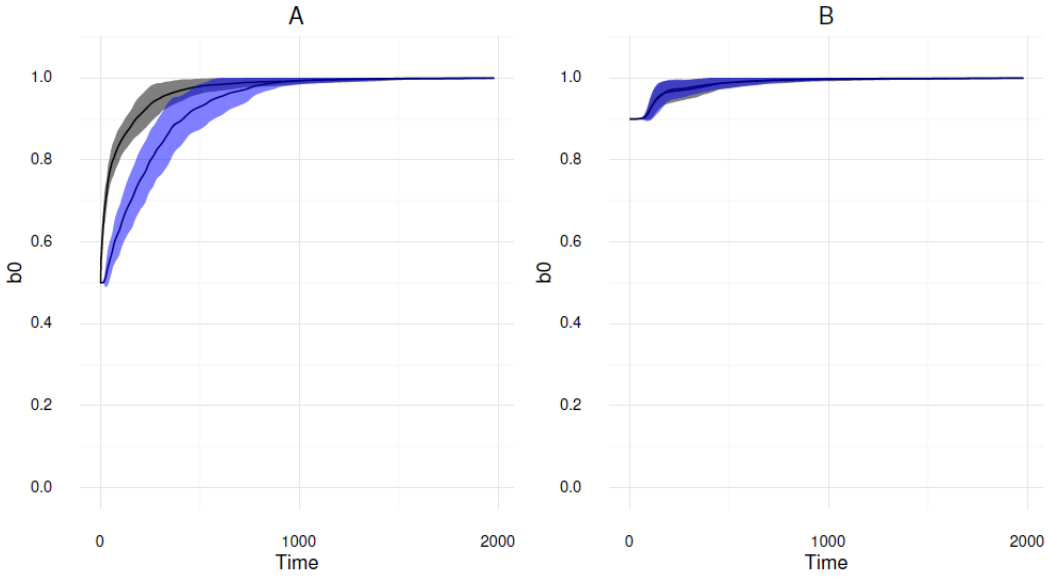


***Figure S2.4**: Evolution of the host population's mean germination fraction (+- sd) under multiple competition (GFG model, unlinked loci), for short term persistence m=5 (black) and long term persistence m=15 (blue) over 50 repetitions. Costs are fixed,* $c_H=c_P=0.2, s=0.3$ *and d=0.002. The initial resident value is* $b_0=0.5$ *(A) and* $b_0=0.9$ *(B).*

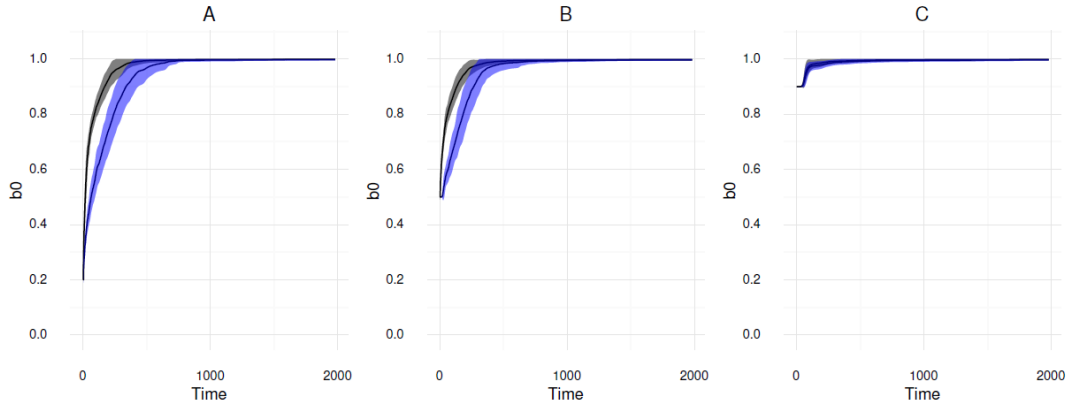


***Figure S2.5**: Evolution of the host population's mean (+- sd) germination fraction under multiple competition (GFG model, unlinked loci), for short term persistence m=5 (black) and long term persistence m=15 (blue) over 50 repetitions. Costs are fixed, c_H=c_P=0.05, s=0.2 and d=0.002. The initial resident value is b_0=0.2 (A), b_0=0.5 (B) and b_0=0.9 (C).*

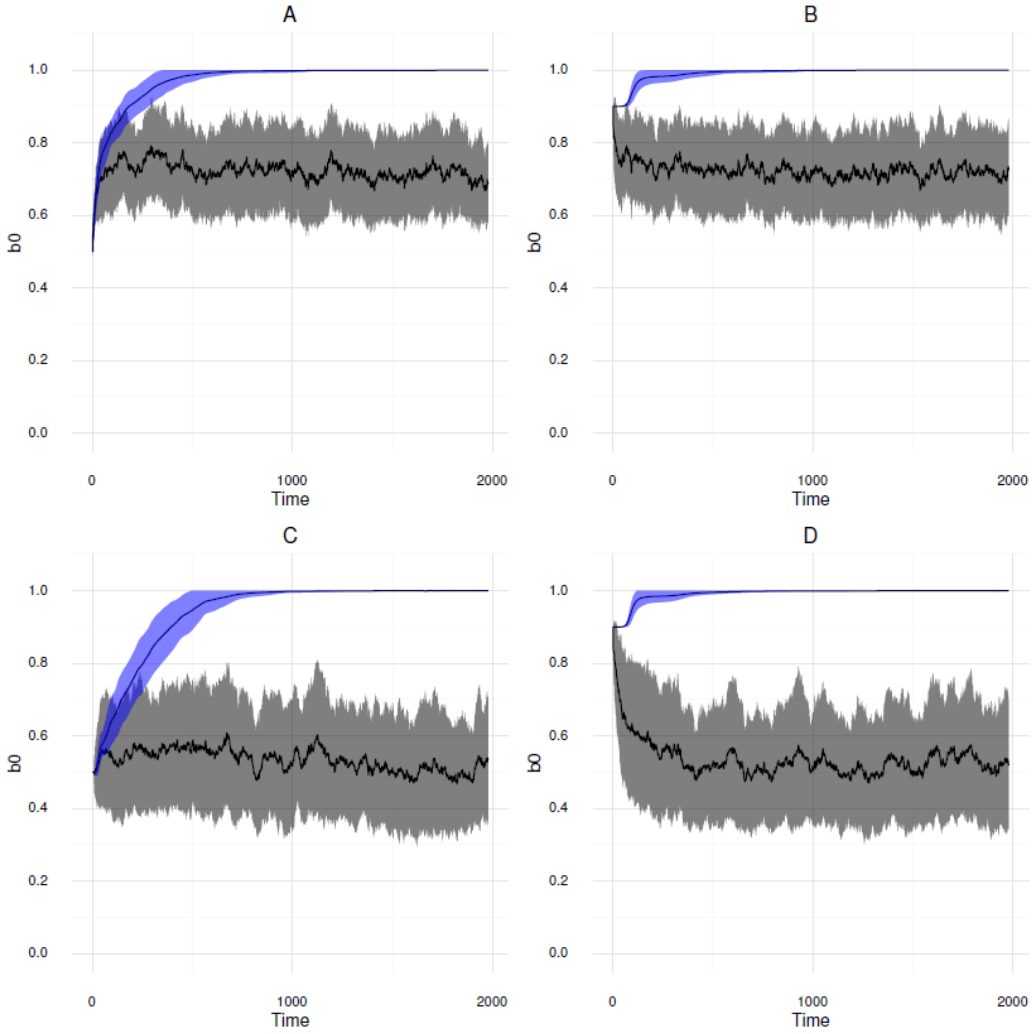


***Figure S2.6**: Evolution of the mean germination fractions (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for short term persistence m=5 (A,B) and long term persistence m=15 (C,D) over 50 repetitions. Costs are fixed, c_H=c_P=0.2, s=0.3, d=0.002. The initial resident value is b_0=0.5 (A,C) and b_0=0.9 (B,D).*

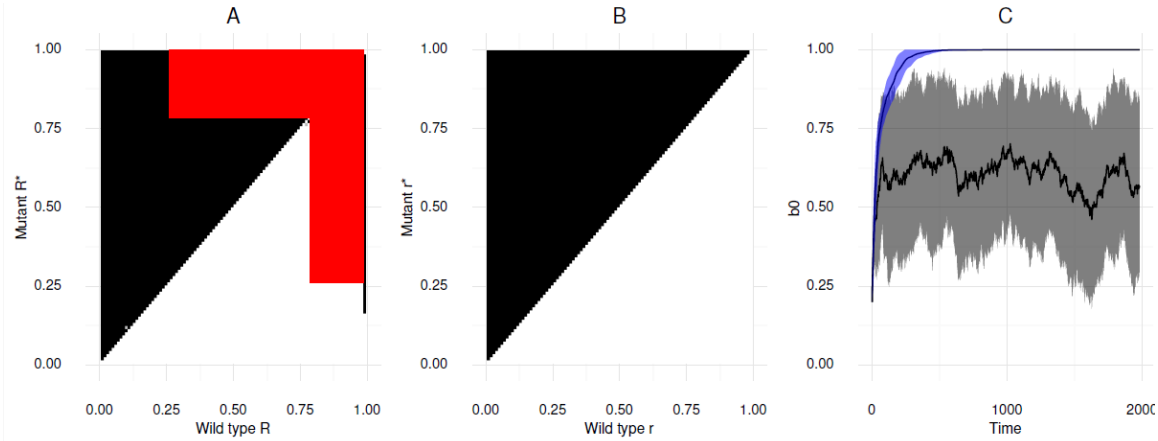


Figure S2.7*: Pairwise invasibility plots (PIP, GFG model, linked loci), for A) resistant and B) susceptible hosts, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed short term persistent bank* ***m=5****, and costs, c_H=c_P=0.05, s=0.2, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions), inferior to 0.1 (white regions), or coexists with the resident phenotype (grey regions). The red region covers the range of value excluded from the study (due to extremely high frequency of the host alleles along the boundaries during the cycles). C) Evolution of the mean germination fractions (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

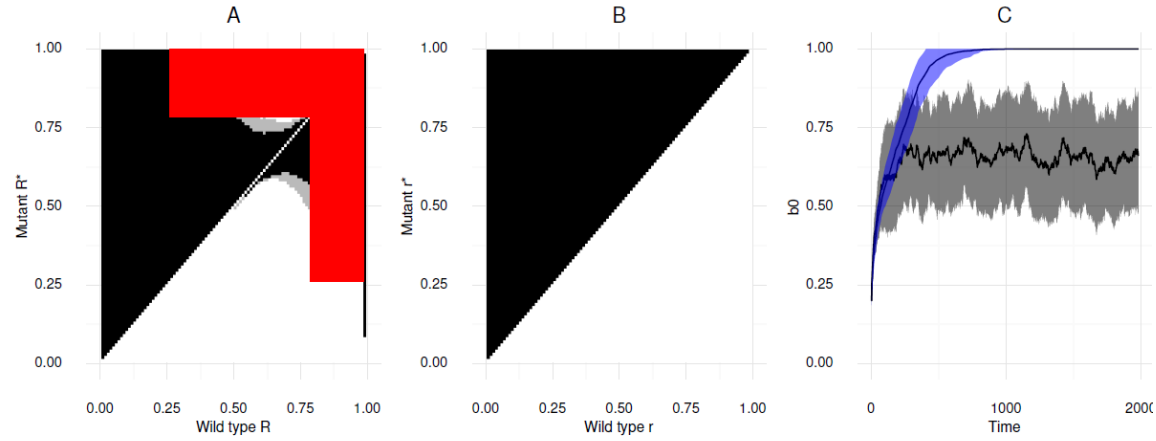


Figure S2.8*: Pairwise invasibility plots (PIP, GFG model, linked loc), for A) resistant and B) susceptible hosts, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed long term persistent bank* ***m=15****, and costs, c_H=c_P=0.05, s=0.2, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions), inferior to 0.1 (white regions), or coexists with the resident phenotype (grey regions). The red region covers the range of value excluded from the study (due to extremely high frequency of the host alleles along the boundaries during the cycles). C) Evolution of the mean germination fractions (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

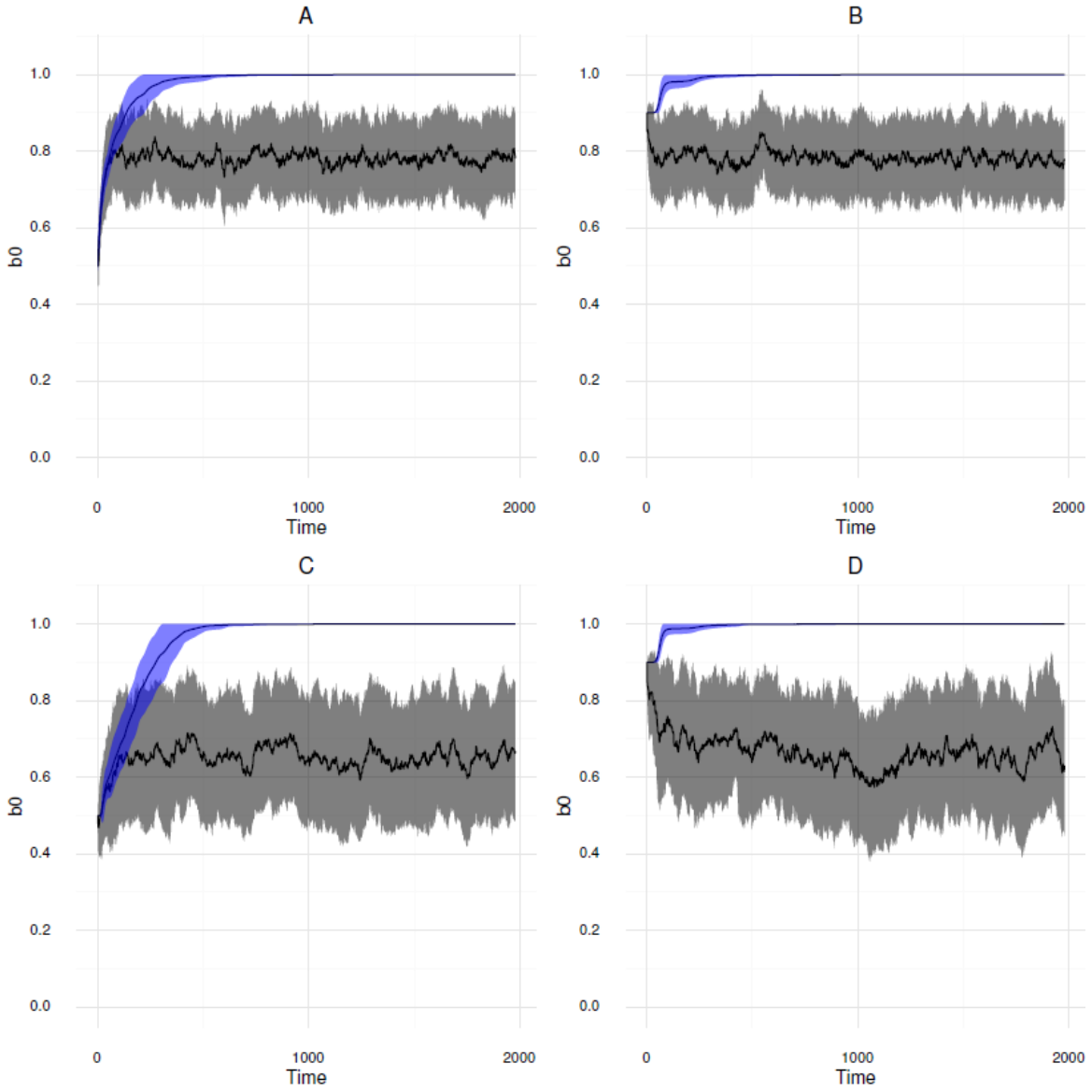


***Figure S2.9**: Evolution of the mean germination fraction (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for short term persistence m=5 (A,B) and long term persistence m=15 (C,D) over 50 repetitions. Costs are fixed, c_H=c_P=0.05, s=0.2, d=0.002. The initial resident value is b_0=0.5 (A,C) and b_0=0.9 (B,D).*

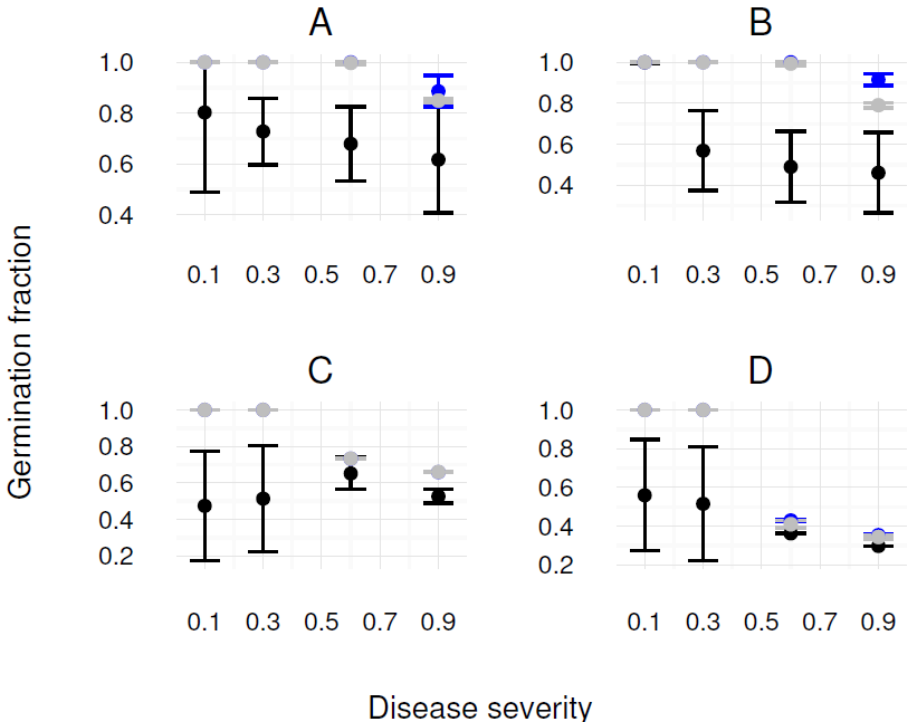


***Figure S2.10**: Population mean germination fraction (unlinked loci, grey), resistant mean germination fraction b_{0R} (linked loci, black) and susceptible mean germination fraction b_{0r} (linked loci, blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations) as a function of the disease severity s (x-axis). The GFG model has the costs of resistance and infectivity fixed to $c_H=c_P=0.1$ (A-B) and $c_H=c_P=0.4$ (C-D). The seed bank persistence m is fixed at 5 years (A-C) and 15 years (B-D). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{0R}=b_{0r}=0.2$. In the case (B) s=0.1, the resistant allele is lost as $s=c_H=c_P$.*

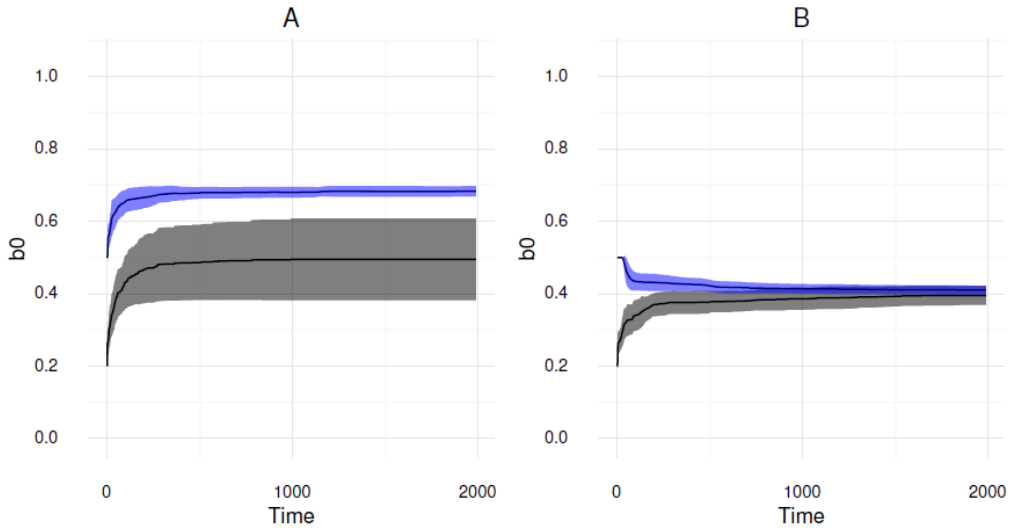


***Figure S2.11**: Evolution of the host population's mean germination fraction (+- sd) under multiple competition (MA model, unlinked loci), for short term persistence m=5 (A) and long term persistence m=15 (B) over 50 repetitions. Disease severity is fixed to s=0.3, and d=0.002. The initial resident value is $b_0=0.2$ (black), $b_0=0.5$ (blue).*

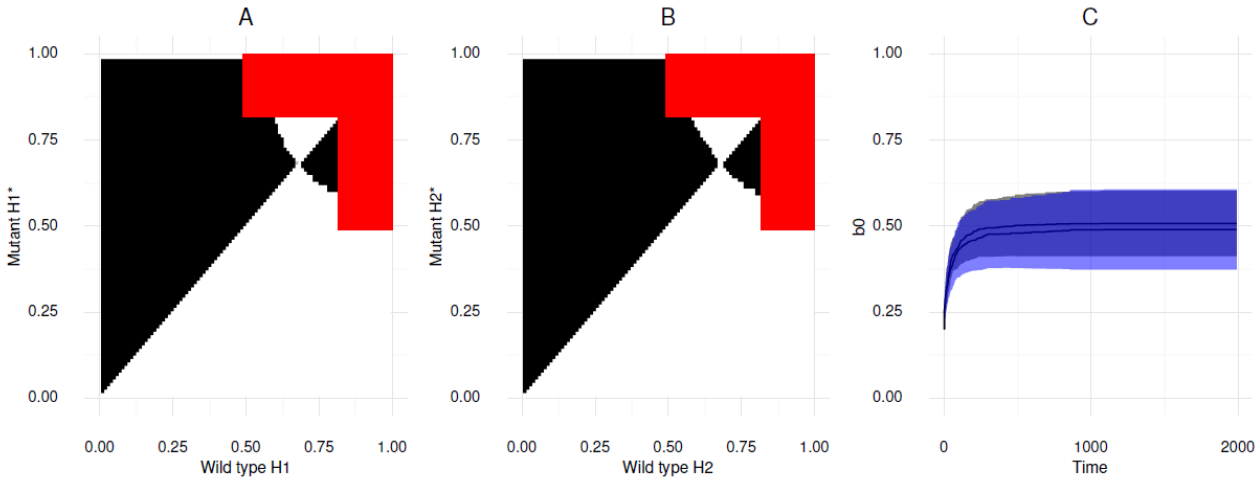


***Figure S2.12**: Pairwise invasibility plots (PIP, MA model, linked loci), for A) host type one and B) host type two, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed short term persistent bank **m=5**, s=0.3, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions) or inferior to 0.1 (white regions). The red region covers the range of value excluded from the study (due to extremely high frequency of the host alleles along the boundaries during the cycles). C) Evolution of the mean germination fractions (+- sd) of host one (black) and host two (blue) under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

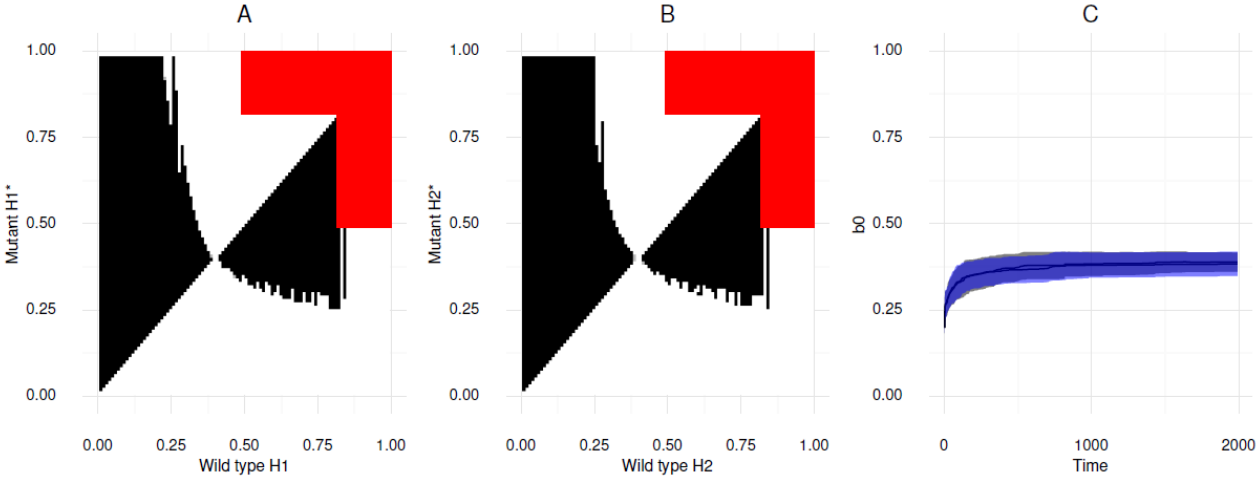


***Figure S2.13**: Pairwise invasibility plots (PIP, MA model, linked loci), for A) host type one and B) host type two, with germination fraction b_0* of invading phenotypes on the vertical axis, and resident phenotypes b_0 on the horizontal axis. Hosts have a fixed long term persistent bank **m=15**, s=0.3, and d=0.002. The dynamics are simulated for 1,000,000 generations, the mutant genotype frequency over the last 10,000 generations is superior to 0.9 (black regions) or inferior to 0.1 (white regions). The red region covers the range of value excluded from the study (due to extremely high frequency of the host alleles along the boundaries during the cycles). C) Evolution of the mean germination fractions (+- sd) of host one (black) and host two (blue) under multiple competition with corresponding parameters over 50 repetitions. The initial resident value is b_0=0.2.*

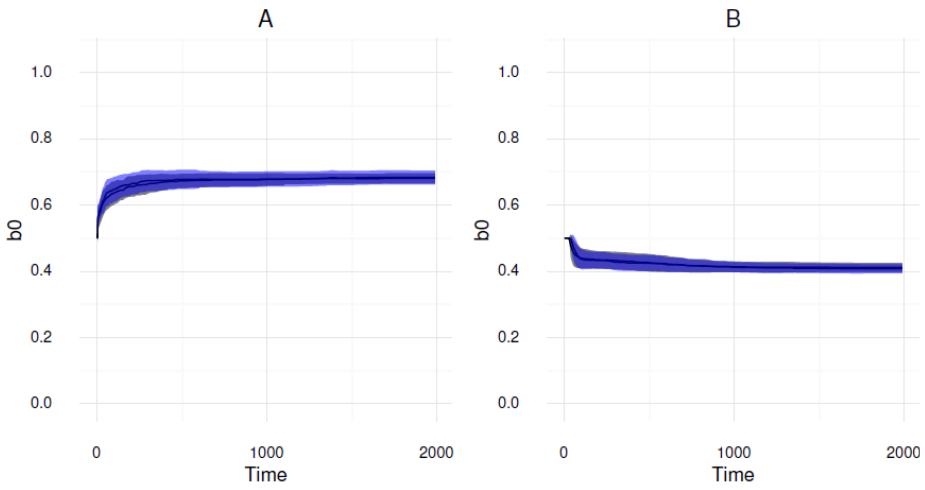


***Figure S2.14**: Evolution of the mean germination fraction (+- sd) of host one (black) and host two (blue) under multiple competition (MA model, linked loci), for short term persistence m=5 (A) and long term persistence m=15 (B) over 50 repetitions. Costs are fixed to s=0.2, and d=0.002. The initial resident value is b_0=0.5.*

Text S1 - Model A1: stochastic fitness in infinite population size

Aims:

The aim of model A1 is to demonstrate that in a variable stochastic environment, the mean of the geometric fitness determines 1) the relative fitness of alleles, and 2) which allele reaches fixation in the case of competition between two alleles. In other words, we show that the results of Lewontin and Cohen (1969) in finite population are also valid in our model of two competing alleles in an infinite population. Our results are analogous to those from Templeton and Levin (1979) who provided a more rigorous demonstration.

Model description:

This is a simple model of competition, discrete time, in infinite population size, with two alleles *P* and *Q*. p_t and q_t are the frequencies of allele *P* and *Q*, respectively, at generation *t*. We can write the frequency of allele *P* and *Q* at the next generation (*t*+1) defined by the following recurrence equations.

$$p_{t+1} = \frac{p_t \rho}{p_t \rho + q_t \omega_t} = \frac{p_t \rho}{w_t}$$

$$\frac{p_{t+1}}{q_{t+1}} = \frac{p_t \rho}{q_t \omega_t}$$

With w_t being the fitness of the whole population at generation *t*, ρ the fitness of allele *P* which is chosen as constant over time, and ω_t is the fitness of allele *Q* at generation *t* which can vary in time. The fitnesses are defined as $1 \geq \rho, \omega > 0$. We assume throughout the article that the fitness per year (ω) cannot be null, to be able to use the approximation of the geometric mean (see below eqs. S1, S2).

Model analysis:

Let us compute the changes in the ratio of frequencies of *P* over *Q* between two consecutive generations (*t* and *t*-1; *t*-1 and *t*-2, …). We use the logarithm of the ratio of frequencies, similar to

the logit function used in stats, log(x/(1-x)) where 1> x > 0. Note that *ln* is a monotonous increasing function.

$$\ln\left(\frac{p_{t+1}}{q_{t+1}}\right)-\ln\left(\frac{p_t}{q_t}\right)=\ln\left(\frac{\alpha}{\omega_t}\right)=\ln\rho-\ln\omega_t$$

So we can write for *t* generations:

$$\ln\left(\frac{p_t}{q_t}\right)-\ln\left(\frac{p_{t-1}}{q_{t-1}}\right)=\ln\left(\frac{\rho}{\omega_{t-1}}\right)$$

$$\ln\left(\frac{p_{t-1}}{q_{t-1}}\right)-\ln\left(\frac{p_{t-2}}{q_{t-2}}\right)=\ln\left(\frac{\rho}{\omega_{t-2}}\right)$$

...

$$\ln\left(\frac{p_1}{q_1}\right)-\ln\left(\frac{p_0}{q_0}\right)=\ln\left(\frac{\rho}{\omega_0}\right)$$

Summing up the changes in allele frequencies over these *t* consecutive generations, we define and obtain:

$$\Delta_t \ln\left(\frac{p}{q}\right)=\ln\left(\frac{p_t}{q_t}\right)-\ln\left(\frac{p_0}{q_0}\right)$$

$$=\sum_{i=0}^{t-1}\ln\left(\frac{\rho}{\omega_i}\right)=t\ln\rho-\sum_{i=0}^{t-1}\ln\omega_i$$

So, the average change per generation between generation 0 and *t* is:

$$\frac{1}{t}\left(\ln\left(\frac{p_t}{q_t}\right)-\ln\left(\frac{p_0}{q_0}\right)\right)=\ln\rho-\frac{1}{t}\sum_{i=0}^{t-1}\ln\omega_i \qquad \text{[S1]}$$

Simple check: if we assume $\omega_t = \omega$, that is the fitness of allele *Q* is constant over time, then eq. S1 becomes:

$$\left(\ln\left(\frac{p_t}{q_t}\right)-\ln\left(\frac{p_0}{q_0}\right)\right)=t(\ln\rho-\ln\omega)$$

It is clear that for allele *Q* to get to fixation, the difference on the left has to be negative such that

$\rho < \omega$. We find the obvious and known results from the literature.

We now assume that ω_t varies in time as a random variable, so that the ω_t are i.i.d. between generations (mimicking Lewontin and Cohen 1969). In eq. S1 we note that is the expectation of

the logarithm of *t* i.i.d. random variables. The expectation of the logarithm of a random variable is the logarithm of the geometric mean, so that we find (if $\omega_i > 0$):

$$\frac{1}{t}\sum_{i=0}^{t-1}\ln\omega_i = \ln\left(\sqrt[t]{\prod_{i=0}^{t-1}\omega_i}\right) \qquad [S2]$$

We have now demonstrated that for allele *Q* to get to fixation against allele *P*, the geometric mean of the ω_t should be higher than ρ, which is analogous for an infinite population model to the results by Lewontin and Cohen (1979). The geometric of the fitness over several years is the quantity which is maximized by evolution (Cohen 1966, Lewontin and Cohen 1969, Templeton and Levin 1979).

Special case of uniform i.i.d. fitness function:

As a special case, if ω_t are uniform random variables $(\omega_0,...,\omega_{t-1} \sim U(\alpha,\beta))$,

we can compute the expectation and variance of the geometric mean (if *t* is large enough):

$$E\left(\sqrt[t]{\prod_{i=0}^{t-1}\omega_i}\right) = \left[\frac{t}{t+1}\left(\beta^{\frac{t+1}{t}} - \alpha^{\frac{t+1}{t}}\right)\right]^t$$

$$V\left(\sqrt[t]{\prod_{i=0}^{t-1}\omega_i}\right) = \left[\frac{t}{t+2}\left(\beta^{\frac{t+2}{t}} - \alpha^{\frac{t+2}{t}}\right)\right]^t - \left[\frac{t}{t+1}\left(\beta^{\frac{t+1}{t}} - \alpha^{\frac{t+1}{t}}\right)\right]^{2t}$$

Text S2 - Model A2: advantage of seed banking in infinite population size

Aims:

The aim of model A2 is to demonstrate how seed banking can evolve as a bet hedging strategy in a model with infinite population size (thus adapting the results of Cohen 1966, Cohen 1993). Our model is also similar to that of Templeton and Levin (1979), whereas they do not investigate the evolution of seed banking strategy itself.

Model description:

Now we extend the model A1 to the situation with a seed bank, and both alleles *P* and *Q* have a fitness ω_g variable in time due to the changing environment at each generation *g*.

We define, *m* as the length of the seed bank, that is the maximum amount of time seeds can stay in the bank. We assume for simplicity *m* to be identical for both *P* and *Q* alleles. The germination rate of seeds of allele *P* and *Q* from *i* generation ago is respectively $b_{P,i}$, and $b_{Q,i}$. Seeds can also die at rate *d* per generation, and for simplicity, *d* is assumed equal for both alleles. The seed death rate follows thus a geometric function with rate *d*.

We also take into account the fact that seeds produced at a given generation *i* in the past have different fitness due to the variable environment quality. The seed at generation *g* have fitness ω_g, and the fitness of seeds produced *i* generations ago is denoted as ω_i. Note here that both alleles have the same fitness at any given generation *g*. In fact, this parameter ω_g (or ω_i) defines the population fitness at generation *g* (or *i*), namely being proportional to the amount of seeds which were produced at that generation by all plants (*P* and *Q*). If the environment is of good quality, more seeds are produced and ω_i is high, while if the environment is very poor, ω_i is very low or even zero in extreme cases. We therefore keep memory of the variation in the quality of the environment over time in the seed bank in our infinite population model (by analogy with the finite population model of Cohen (1966, 1993)).

We write a simple two allele discrete time model to compute the allele frequency of allele *P* and *Q* at generation *g*+1 based on their frequency in the previous *m* generations (p_{g-i},or q_{g-i}):

$$p_{g+1} = \frac{\omega_g \sum_{i=0}^{m} p_{g-i} b_{P,i} (1-d)^i \omega_i}{w_g} \quad \text{and} \quad q_{g+1} = \frac{\omega_g \sum_{i=0}^{m} q_{g-i} b_{Q,i} (1-d)^i \omega_i}{w_g},$$

where $w_g = \omega_g \sum_{i=0}^{m} P_{g-i} b_{P,i} (1-d)^i \omega_i + \omega_g \sum_{i=0}^{m} q_{g-i} b_{Q,i} (1-d)^i \omega_i$

Note that by definition $\sum_{i=0}^{m} b_{P,i} = \sum_{i=0}^{m} b_{Q,i} = 1$, and ω_g simplifies in numerator and denominator so that it does not influence the value of p_{g+1}.

Model analysis:

In order to simplify the calculations, it is assumed that the seed bank for both alleles *P* and *Q* is fully occupied, with frequencies p_0 and q_0 respectively, for each of the *m+1* generations. So we define $\forall i \in [1, m], p_i = p_0$ and $q_i = q_0$. We can sum up the changes in allele frequencies between *m+1* consecutive generations as in model A1:

$$\Delta_{m+1} \ln\left(\frac{p}{q}\right) = \ln\left(\frac{p_{m+1}}{q_{m+1}}\right) - \ln\left(\frac{p_0}{q_0}\right).$$

Note here that for all subsequent models with seed bank (A2, A3, B and C), we assume the sum over *M=m+1* successive generations, because we define the germination rate b_0 for seeds which do not enter the seed bank and germinate the year after they were produced. Seeds enter the seed bank, and are amenable to death only when being for at least a full generation in the seed bank (*i* > 0).

We find:

$$\Delta_{m+1} \ln\left(\frac{p}{q}\right) = \ln\left(\frac{\sum_{i=0}^{m} p_{m-i} b_{P,i} (1-d)^i \omega_i}{\sum_{i=0}^{m} q_{m-i} b_{Q,i} (1-d)^i \omega_i}\right) - \ln\left(\frac{p_0}{q_0}\right)$$

So that the increase/decrease in frequency of allele *P* compared to *Q*, depends on the sign of the above difference. When averaging over m+1 generations, we find:

$$\frac{1}{m+1}\left(\Delta_{m+1} \ln\left(\frac{p}{q}\right)\right) = \frac{1}{m+1} \ln\left(\sum_{i=0}^{m} b_{P,i} (1-d)^i \omega_i\right) - \frac{1}{m+1} \ln\left(\sum_{i=0}^{m} b_{Q,i} (1-d)^i \omega_i\right) \qquad [S3]$$

We recognize again the expectation of the logarithm of a random variable, and we can take the logarithm of the geometric mean. To determine which allele reaches fixation, we compute the difference in the geometric means of fitnesses, so that allele *Q* reaches fixation if:

$$\ln\left(\sqrt[m+1]{\prod_{i=0}^{m} b_{P,i}(1-d)^i\omega_i}\right) < \ln\left(\sqrt[m+1]{\prod_{i=0}^{m} b_{Q,i}(1-d)^i\omega_i}\right) \quad \text{[S4]}$$

For the variance, it is expected following Cohen (1993) that allele *Q* can invade if it has a smaller variance of the geometric mean than *P*, so we find:

$$V\left(\sqrt[m+1]{\prod_{i=0}^{m} b_{P,i}(1-d)^i\omega_i}\right) > V\left(\sqrt[m+1]{\prod_{i=0}^{m} b_{Q,i}(1-d)^i\omega_i}\right)$$

$$\Rightarrow \prod_{i=0}^{m} {b_{P,i}}^{2/(m+1)}(1-d)^{2i/(m+1)} > \prod_{i=0}^{m} {b_{Q,i}}^{2/(m+1)}(1-d)^{2i/(m+1)}$$

Conclusion:

We can analyze the competition between alleles *P* and *Q* as being equivalent to the competition between one resident allele with a given seed bank strategy (*P*) and an introduced mutant with a different seed bank strategy (*Q*). A stronger seed bank strategy for allele *a* versus *P*, is equivalent to a shift of the distribution towards higher values of $b_{Q,i}$ for higher values of *i*, compared to $b_{P,i}$. The geometric of the fitness over the *m* years stored in the bank is the quantity which is maximized by evolution (Cohen 1966, Lewontin and Cohen 1969, Templeton and Levin 1979).

We find two key results which defines the evolution of seed banking as a bet hedging strategy:

1) when the environment varies stochastically in time, a mutant with a longer seed bank invades if its geometric mean of fitness in the seed bank is higher than that of the resident, and

2) when the environment is constant over time, seed mortality ensures that the mutant with shorter seed bank then invades.

Text S3 - Model A3: Optimal seed banking strategy with environment fluctuations of constant amplitude and periods

Aims:

Our aim is to determine the germination fraction b_0 optimizing the geometric mean fitness of a host when its reproduction is influenced by a cycling environment of fixed amplitude and period. We investigate the interaction between the period of the environmental cycle and the fixed persistence *m* of the seed bank. Then we investigate the optimal investment in the seed bank for various persistence *m* with fixed period of the environment with high and reduced amplitudes cycling amplitudes.

Model description:

We consider one population. The rate b_i (at which seeds produced *i* generations ago germinates at a given generation *g*) is reduced by a survival rate ω_i depending on the environment state at the time *i* of their production (as in model A2). The population fitness at generation *g*+1 is:

$$w_{g+1} = \sum_{i=0}^{m} b_i w_{g-i} (1-d)^i \omega_i$$

The value ω_i is taken in the environmental sequence ω(t), corresponding to a succession of cycles of constant amplitude s and period θ, following the equation:

$$\omega(t) = \sigma \sin(\frac{1}{\theta} t) + 0.5$$

With s=0.5, the amplitude is high and the rate ω_i varies between 0; corresponding to the death of all seeds produced *i* generations ago, and 1; corresponding to the germination of all seeds. With s=0.25, the amplitude is halved. We define 4 periods for the cycles, 100, 50, 25 and 12.5 generations
(θ = {1/16; 1/8; 1/4; 1/2}). The environmental sequence is of length T=2,000 generations, and we calculate the geometric mean fitness of the host over T. For each set of parameters tested, the

value b_0 corresponding to the highest geometric mean is the optimal strategy.

Results:

Fixing the persistence *m* to 50 years, we observe that the geometric means become higher with decreased environmental periods (**Fig. S2.2A**). Optimal germination fractions are low (values around 0.1) for each period tested, although the investment in the bank is slightly stronger when the environmental period exceeds that of the persistence.

Fixing the period to 100 years, we observe an increase of the optimal germination fraction when the seed bank persistence is shorter (**Fig. S2.2B**). With halved amplitudes, we observe higher fitness values independently of the persistence *m* (**Fig. S2.2C**). The highest germination fraction (b_0=1), is now optimal for persistence of 5, 15 and 50 years, corresponding to a non-seed banking strategy. For the longest persistence of 100 years, a low germination fraction is optimal.

Conclusion:

When environmental cycles are drastic, seed banks should evolve towards the longest persistence possible, with a very high investment ($1-b_0$) in the bank. A high investment in the bank is optimal only if the persistence in the bank is long enough in comparison to the period of the cycling environment, considering both drastic and mild environments. The advantage of a seed bank is lost in a mild environment for short persistence. These results are analogous to those from Templeton and Levin (1979).

Text S4 - Model B: Host-parasite coevolution model with Matching allele

Aims:

Our aim is to compare the prediction obtained between GFG and MA allele approach to understand the impact of a symmetrical or asymmetrical infection matrix on the bet hedging evolution via the changes in coevolutionary cycles.

Model description:

In an MA approach, hosts and parasites do not carry costly resistance and infectivity traits, but parasites genotype must match the most common host genotype to infect them successfully (*e.g.* Gandon et al. 1996). We have two types of host H1 and H2 and two types of parasite P1 and P2; P1 matches the genotype of H2 and P2 the genotype of H1. The rest of the model assumptions are common to the GFG model described in the main article (***Model description and methods)***. The following difference equations describe changes in allele frequencies including a seed bank for the host.

$$H_{1,g+1} = (1 - sP_{2,g})\left[\sum_{i=0}^{\min(m_{H1},g)} b_{H_1,i}(1-d)^i H_{1,g-i}\right] / \overline{w}_{H,g}$$

$$H_{2,g+1} = (1 - sP_{1,g})\left[\sum_{i=0}^{\min(m_{H2},g)} b_{H_2,i}(1-d)^i H_{2,g-i}\right] / \overline{w}_{H,g}$$

$$\overline{w}_{Hg} = (1 - sP_{2,g})\left[\sum_{i=0}^{\min(m_{H1},g)} b_{H_1,i}(1-d)^i H_{1,g-i}\right] + (1 - sP_{1,g})\left[\sum_{i=0}^{\min(m_{H1},g)} b_{H_2,i}(1-d)^i H_{2,g-i}\right]$$

$$P_{1,g+1} = P_{1,g}\left[\sum_{i=0}^{\min(m_{H2},g)} b_{H_2,i}(1-d)^i H_{2,g-i}\right] / \overline{w}_{P,g}$$

$$P_{2,g+1} = P_{2,g}\left[\sum_{i=0}^{\min(m_{H1},g)} b_{H_1,i}(1-d)^i H_{1,g-i}\right] / \overline{w}_{P,g}$$

$$\overline{w}_{P,g} = P_{1,g}\left[\sum_{i=0}^{\min(m_{H2},g)} b_{H_2,i}(1-d)^i H_{2,g-i}\right] + P_{2,g}\left[\sum_{i=0}^{\min(m_{H1},g)} b_{H_1,i}(1-d)^i H_{1,g-i}\right]$$

Five equilibrium points exist, the four monomorphic trivial outcomes (0,0), (1,0), (0,1), (1,1), and the internal point at which all four host and parasite alleles are maintained: $\left(\hat{H}_1 = 1/2; \hat{P}_1 = 1/2\right)$. The stability of the internal point depends on the initial frequencies of hosts and parasites; when both host alleles are in equal frequencies, it is stable, otherwise it is unstable. The co-

evolutionnary cycles under MA interaction are faster than under GFG. Increasing the germination fraction b_0 or the seed bank persistence *m* shortens the periods of the cycles and increases their amplitude. This leads to numerical issues using the R software as frequencies approaches the boundaries (0 or 1). With germination fractions above the value 0.8; amplitudes are varying to such extents that it reaches the largest double of the machine (.Machine$double.xmin), meaning that allele frequencies are in the range of very close to fixation for most of the time. We decided to remove the range of value $0.8<b_0<1$ of the study. We use the same methods, pairwise competition and multiple mutant competition, as for the GFG model, to study the evolution of the seed banking strategy.

Results:

Considering genetically unlinked loci, we observe the evolution of the population mean strategy evolving towards different range of values depending on the fixed seed bank persistence (**Fig. S2.11**, multiple competition method). The investment in the seed bank is clearly stronger for long term persistent bank, with $\bar{b}_0 = 0.4$.

Considering (genetically linked) loci of coevolution and germination, the result are equivalent to those obtained for unliked loci (**Fig. S2.12-14**). Furthermore we observe that both host types evolve towards the same range of strategies (**Fig. S2.11B, S2.12C, S2.13C, S2.14B**).

Conclusion:

As host genotypes are now strictly equal for their fitnesses, we observe the evolution of strategies belonging to the same range of values for both host types, independently of the genetic assumptions (linked or unlinked loci).

Annex[1]

Content:

- **Summary**
- **Model C:** Host-parasite interaction model with stochastic disease incidence.
- **Model C1**: Evolution of seed banking under stochastic disease prevalence.
- **Model C2**: Host-parasite interaction model with seed bank.

1 This annex is not part of the publication

Summary:

The model analysed in this first section assumes that parasite prevalence is constant over time. We investigate here the influence of a stochastically varying prevalence over time using a reduced model of host-parasite coevolution and assess whether stochastic disease prevalence can select for the evolution of seed banking as a bet-hedging strategy.

We assume that hosts are coevolving with only one type of parasite (non-infective), but its prevalence varies stochastically over time. As expected, we demonstrate that when susceptible hosts genotypes with different seed banking strategies are competing, the genotype with the highest geometric fitness reaches fixation (Model C1).

As a second step, we analyse the competition between a non seed banker resistant host and a seed banker susceptible host genotype. Facing stochastic disease prevalence, the resistant genotype evolves towards fixation rather than susceptibility and seed banking – unless the efficiency of resistance is lower than its cost, which is biologically unrealistic. Overall, we show that stochastic disease prevalence cannot favor bet-hedging strategies as an alternative to evolving resistance to infection.

Model C: host-parasite interaction model with seed bank and stochastic disease prevalence

Aims:

The aim of model C is to study the conditions under which seed banking can evolve in response to simple host-parasite interactions. We assume that the only stochastic parameter depending on the environment is the disease prevalence which varies randomly over time and is defined as ϕ_g at generation g, and ϕ_i at the i^{th} generation ago.

Model description:

We simplify here the model from the main text (eq. 1) assuming two host alleles (resistant R and susceptible r) but only one parasite type (here the avirulent or non-infective, A). The disease prevalence is ϕ_i which varies over time, and is defined as a random uniform distribution taking values between 0 (no disease) to 1 (all plants are infected). The susceptible plant are always infected, while the resistant plant are infected with probability 1-c (c is the effectiveness of resistance) but resistance carries a fitness cost c_H. The fitness cost for the host of being infected is s. The seed bank is defined as in model A2 assuming that R and r alleles have different germination functions ($b_{R,i} \neq b_{r,i}$), but equal length of the seed bank m.

We can thus rewrite the difference equations from the main text (eq. 1) as to compute the allele frequencies of allele R and r at generation g+1 as a function of the previous m generations stored in the seed bank:

$$\frac{R_{g+1}}{r_{g+1}} = \frac{(1-c_H)\left[\sum_{i=0}^{m} R_{g-i} b_{R,i} (1-d)^i (1-\phi_i s(1-c))\right]}{\sum_{i=0}^{m} r_{g-i} b_{r,i} (1-d)^i (1-\phi_i s)} \qquad \text{[S5]}$$

Note that by definition $\sum_{i=0}^{m} b_{R,i} = \sum_{i=0}^{m} b_{r,i} = 1$ and to make notations easier, M=m+1.

Model analysis:

Assuming that both *RES* and *res* allele have full seed bank with fixed frequencies R_0 and r_0, so $\forall i \in [1,m], R_{g-i} = R_0$ and $r_{g-i} = r_0 \cdot$ that

We can sum up the changes in allele frequencies between *m+1* consecutive generations as in model A1 and A2, so that:

$$\Delta_M \ln\left(\frac{R}{r}\right) = \ln\left(\frac{R_{g+1}}{r_{g+1}}\right) - \ln\left(\frac{R_0}{r_0}\right) = \ln\left(\frac{(1-c_H)\sum_{i=0}^{m} b_{R,i}(1-d)^i(1-\phi_i s(1-c))}{\sum_{i=0}^{m} b_{r,i}(1-d)^i(1-\phi_i s)}\right)$$

$$\Rightarrow \frac{1}{M}\left(\Delta_M \ln\left(\frac{R}{r}\right)\right) \qquad [S6]$$

$$= \frac{1}{M}\ln\left(1-c_H\right) + \ln\left(\sqrt[M]{\prod_{i=0}^{m} b_{R,i}(1-d)^i(1-\phi_i s(1-c))}\right) - \ln\left(\sqrt[M]{\prod_{i=0}^{m} b_{r,i}(1-d)^i(1-\phi_i s)}\right)$$

From equations S5, S6, we derive in the following two cases of interest to investigate the behavior of model C:

1) assuming a model with only susceptible plants, we show how seed banking evolves in response to stochastic infection rates (model C1)

2) if *res* alleles can evolve seed bank while competing with resistant alleles without seed bank, we compute which allele reaches fixation (model C2).

Model C1: Evolution of seed banking under stochastic disease prevalence

Aims:

In model C1 we address the question of whether seed bank can evolve as a bet hedging strategy to mitigate the effect of stochastic variation for disease prevalence over time.

Model C1 analysis:

Starting with equation S5, we now define a competition model for two susceptible alleles with frequencies r by setting $c=c_H=0$. To study the evolution of seed banking, we study the condition for a mutant r^* with a different seed bank to invade and reach fixation against the resident allele r. The distribution of germination rates are then $b_{r,i}$ and $b_{r^*,i}$. We also use as above the fact that the seed bank is full with a given fixed frequency of allele r_0 and r_0^*. Note that $M=m+1$.

$$\Delta_M \ln\left(\frac{r}{r^*}\right) = \ln\left(\frac{r_{g+1}}{r^*_{g+1}}\right) - \ln\left(\frac{r_0}{r^*_0}\right) = \ln\left(\frac{\sum_{i=0}^{m} b_{r,i}(1-d)^i(1-\phi_i s)}{\sum_{i=0}^{m} b_{r^*,i}(1-d)^i(1-\phi_i s)}\right) \qquad [S7]$$

As we define $\phi_i \sim U(0,1)$, in the eq. S7 can be replaced by $\phi_i' \sim U(1-s,1)$.

Then taking the expectations of the geometric mean we find that the condition for the mutant r^* for the seed bank strategy to invade is:

$$\frac{1}{M}\Delta_M \ln\left(\frac{r}{r^*}\right) < 0$$

$$\Rightarrow \left[\frac{M}{M+1}\left(1-(1-s)^{\frac{M+1}{M}}\right)\right]^M \prod_{i=0}^{m} b_{r,i}^{\,1/M}(1-d)^{i/M} < \left[\frac{M}{M+1}\left(1-(1-s)^{\frac{M+1}{M}}\right)\right]^M \prod_{i=0}^{m} b_{r^*,i}^{\,1/M}(1-d)^{i/M}$$

$$\Rightarrow \prod_{i=0}^{m} b_{r,i}^{\,1/M}(1-d)^{i/M} < \prod_{i=0}^{m} b_{r^*,i}^{\,1/M}(1-d)^{i/M} \qquad [SX]$$

Conclusion:

The competition between two susceptible alleles with different seed banks under unpredictable variable disease prevalence (eq. S6) follows the same results (eq. S7) as for the evolution of bet hedging in a variable environment (model A2, eq. S3, S4).

Model C2: Host-parasite interaction model with seed bank

Aims:

In model C2, we address the following question. Which of the following two strategies is better: evolving resistance (*RES* allele) or a long seed bank (the *res* allele) in response to variable infection prevalence over time?

Model B2 analysis:

We start from equation S5 and assume that the resistant allele has no seed bank ($b_{R,0}$=1) and obtain:

$$\frac{R_{g+1}}{r_{g+1}} = \frac{(1-c_H)R_g(1-\phi_g s(1-c))}{\sum_{i=0}^{m} r_{g-i} b_{r,i}(1-d)^i(1-\phi_i s)}$$

Defining $R_m = R_0, r_m = r_0$:

$$\Delta_M \ln\left(\frac{R}{r}\right) = \ln\left(\frac{R_{m+1}}{r_{m+1}}\right) - \ln\left(\frac{R_0}{r_0}\right) = \ln\left(\frac{R_m(1-c_H)(1-\phi_m s(1-c))}{r_m \sum_{i=0}^{m} b_{r,i}(1-d)^i(1-s\phi_i)}\right) - \ln\left(\frac{R_0}{r_0}\right)$$

$$\Delta_M \ln\left(\frac{R}{r}\right) = \ln\left(\frac{(1-c_H)(1-\phi_m s(1-c))}{\sum_{i=0}^{m} b_{r,i}(1-d)^i(1-\phi_i s)}\right)$$

$$\frac{1}{M}\Delta_M \ln\left(\frac{R}{r}\right) = \frac{1}{M}\ln\left((1-c_H)(1-\phi_m s(1-c))\right) - \ln\left(\sqrt[M]{\prod_{i=0}^{m} b_{r,i}(1-d)^i(1-\phi_i s)}\right) \qquad [S9]$$

We solve equation S9 with $d > 0$. The sign of equation S9 indicates which allele reaches fixation (with M=m+1). As we define $\phi_i \sim U(0,1)$, $(1-s\phi_i)$ in the eq. S9 can be replaced by $\phi_i' \sim U(1-s,1)$. We then take the expectations of both sides of the equation above, and thus the susceptible allele invades (reaches fixation) if:

$$\left((1-c_H)(1-\phi_m s(1-c))\right)^{1/M} < \left[\frac{M}{M+1}\left(1-(1-s)^{\frac{M+1}{M}}\right)\right]^M \prod_{i=0}^{m} b_{r,i}{}^{1/M}(1-d)^{i/M} \qquad [S10]$$

There are few cases of interest when analyzing eq. S10.

First, if the resistance is total, c=1, then the left term of eq. S10 becomes $\left((1-c_H)(1-\phi_m s(1-c))\right)^{1/M} = \left(1-c_H\right)^{1/M}$, and if resistance is not too costly (c_H high that is c_H » 1), resistance would be favored compared to the susceptible allele with the seed bank strategy.

Second, more generally we derive two limits for the equation S10: when susceptible alleles evolve long seed banks ($M \to \infty$), or when the cost of infection is very high ($s \to 1$).

Long seed bank limit:

$$\lim_{M\to\infty}\left[\frac{M}{M+1}\left(1-(1-s)^{\frac{M+1}{M}}\right)\right]^M \prod_{i=0}^{m} b_{r,i}{}^{1/M}(1-d)^{i/M} = s^M \prod_{i=0}^{m} b_{r,i}{}^{1/M}(1-d)^{i/M}$$

and,

$$\lim_{M\to\infty}\left((1-c_H)(1-\phi_m s(1-c))\right)^{1/M} = 1$$

In the case of very long seed bank, the susceptible allele cannot invade.

High disease severity limit:

$$\lim_{s\to 1}\left[\frac{M}{M+1}\left(1-(1-s)^{\frac{M+1}{M}}\right)\right]^M \prod_{i=0}^{m} b_{r,i}{}^{1/M}(1-d)^{i/M} = \left(\frac{M}{M+1}\right)^M \prod_{i=0}^{m} b_{r,i}{}^{1/M}(1-d)^{i/M}$$

and,

$$\lim_{s\to 1}\left((1-c_H)(1-\phi_m s(1-c))\right)^{1/M} = \left((1-c_H)(1-\phi_m(1-c))\right)^{1/M}$$

Conclusion:

Overall, susceptible alleles have a disadvantage to evolve a seed bank strategy as an alternative to total resistance against the pathogen. In fact, as the disease prevalence varies in time between 0 and 1, the fitness of infected susceptible individuals varies between 1-s and 1, to be compared to the fitness of infected resistant individuals which lies between $(1-c_H)(1-s(1-c))$ and $(1-c_H)$. Thus the advantage for susceptible alleles with a seed bank against the resistant alleles occurs only if resistance is costly (c_H»1) and is not total (c<1). In addition, even if c<1, resistance is

advantageous compared to a very long seed bank ($M \to \infty$). In the only case where s is very high, i.e. when the parasite is very aggressive, the susceptible allele may invade if the cost of resistance is high ($c_H \gg 1$) and its effectiveness is low ($c < 1$).

Eco-evolutionary feedbacks between coevolutionary dynamics and host seed banking strategy

3.1 Introduction

Parasitism affects the growth, survival and fecundity of infected hosts, hereby modifying hosts life cycles. As an evolutionary answer to the fitness costs of infection, hosts may alter their life history traits (e.g. experimental studies of Michalakis & Hochberg 1994; Richner 1998; Agnew *et al.* 2000). These traits are mostly related to the reproduction component of their life cycle, such as evolving towards early reproduction in drosophilas (Gomariz-Zilber & Thomas-Orillard 1993) or smaller size at maturation observed in marine snail (Lafferty 1993). Increased clutch size have been reported in the great tits (Richner 1998), and the evolution towards an early development and timing of first reproduction in hosts is expected for high disease severity (Hochberg *et al.* 1992). Pathogens modify as well the plant resource allocation to vegetative versus sexual reproduction, shifting towards self-fertilization if infection occurs via flowers, or sexual reproduction when risks of disease transmission from parents to asexual progeny are high (Parker 1992). It is often assumed that parasites shape the host life cycle to increase their transmission. Yet parasites are bound to their host life cycle, and their development time and transmission mode must correlate with the life span of the host. For example the variability in the development time of one mosquito species correlates with the transmission mode of its parasite; mosquitoes pupating early transmitting vertically the parasite while late pupation allows horizontal transmission (Koella & Agnew 1999). The virulence and prevalence of a parasite is then function of its own life cycle and the host life history traits (Koella *et al.* 1998). Moreover, changes in host life history traits affect the evolution of parasite transmission and/or virulence (Gandon *et al.* 2002a,b, 2008).

These reciprocal life history changes of hosts and parasites– feedbacks – define coevolution in its broader sense. Coevolution can also more strictly be defined by the genetic interaction occurring at a few loci between hosts and parasites (Flor 1971; Frank 1992). This interaction is antagonistic, and hosts are being under selection for resistance and reciprocally parasite for evolving infectivity (counter-resistance). Coevolutionary dynamics are driven by indirect negative frequency dependent selection – iFDS – where rare alleles exhibit fitness advantages as selection in the host population depends on allele frequencies in the parasite population, and vice versa (Clarke 1964, Tellier and Brown 2007). When selection and life cycles of hosts and parasites are synchronised, iFDS generates unstable dynamics of allele frequencies in both populations over time, namely resulting in fixation of one host and one parasite genotype. Allele frequency changes fluctuate around the so-called polymorphic internal equilibrium point, which in this case is said to be unstable. However, several studies described epidemiological and ecological factors that can stabilise host/parasites coevolutionary dynamics. Long term stable polymorphism, *i.e.* maintenance of several genotypes in host and parasite populations and fluctuations reaching the internal equilibrium point, can be achieved via the decoupling of host and parasite life cycles in either time or space (Brown & Tellier 2011). Host overlapping generations through seed banking or perenniality (Tellier & Brown 2009) and parasite polycyclic life cycle (Leonard 1977; Tellier & Brown 2007) separate the time scale of hosts and parasites, while separation in space can occur for spatial heterogeneous parasite virulence (Nuismer 2006). Density dependent disease transmission via an epidemiological set up also generates long term polymorphism (Boots *et al.* 2009; Tellier & Brown 2009; Ashby & Boots 2017). In summary, feedbacks occur between host and parasite life cycles and history traits (see previous section), while host and parasite life history traits also shape the coevolutionary dynamics of their genetic interaction.

We illustrate in this study an eco-evolutionary feedback between the evolution of a host life history trait and the genetic coevolutionary interaction of host and parasites, by investigating the evolution of host seed banking strategies. Seed banking is a temporal strategy of bet-hedging, defined as the long term storage in the soil of a fraction of the seeds produced by a plant. The influence of a seed bank on host/parasite coevolutionary dynamics is well understood and stabilizes the coevolutionary dynamics, promoting the maintenance of polymorphism (Tellier & Brown 2009). Secondly we know that varying coevolutionary dynamics with unstable cycling amplitude and period can promote the evolution of host seed banking strategies (Verin & Tellier 2018). In the model derived in the first section of this manuscript, the probability of a seed to germinate does not depend on its age, as such the seed recruitment in the above ground population decreases with age, and the germination input from the bank does not affect the stability of the coevolutionaty dynamics, *i.e.* the non trivial equilibrium is unstable. In this study, we relax this assumption by assuming an age -specific recruitment.

We use a basic gene-for-gene (GFG) model of host/parasite interaction in plant (Leonard 1977), where plants and parasites have one interacting corresponding locus with respectively, one allele for resistance (R) and one for susceptibility (r) for the pant, and one allele for non-infective (nINF) and infective (INF) for the parasite. The seed bank strategy of a plant is determined by the seed bank persistence bounded to m and the fraction of seeds germinating each year b_i. Finally, the probability of a dormant seed to germinate is dependent of its age. We study the evolution of the germination fraction with a fixed seed bank persistence ranging from 1 to 20 years, disentangling the role of fluctuating selection – occurring at the coevolving loci – from the selective advantages of the evolving seed bank itself.

Using multiple mutant competition simulations of seed banking strategies, we show that coevolutionary dynamics can feedback on the evolving strategies. Indeed strategies, evolving in response to fluctuating selection, can stabilise polymorphism. This generates a novel (stable) environment for hosts and parasites, modifying the fitness advantages of seed banking strategies, leading to drastic and distinct shifts between the optimal strategies of the susceptible and the resistant hosts over time.

3.2 Material and methods[1]

3.2.1 Model description

We assume an infinite population size model describing a GFG interaction between haploid hosts and parasites (*e.g.* Tellier & Brown 2009). The GFG interaction is defined by one genetic locus with two alleles, hosts exhibit a resistant (*R*) or a susceptible (*r*) allele, and parasite an avirulent (*A*) or a virulent (*a*) allele. The difference equations describe the changes in allele frequencies over generations *g* for both hosts and parasites.

$$R_{g+1}=(1-c_H)(1-s(1-A_g))[\sum_{i=0}^{min(m_R,g)} b_{Ri}(1-d)^i R_{g-i}]/\overline{W_H} \quad [1]$$

$$r_{g+1}=(1-s)[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}]/\overline{W_H} \quad [2]$$

$$A_{g+1}=A_g[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}]/\overline{W_P} \quad [3]$$

$$a_{g+1}=a_g(1-c_P)[\sum_{i=0}^{min(m_r,g)} b_{ri}(1-d)^i r_{g-i}+\sum_{i=0}^{min(m_R,g)} b_{Ri}(1-d)^i R_{g-i}]/\overline{W_P} \quad [4]$$

1 The GFG model described in this paragraph is strictly identical to the model described in chapter 2.

At generation *g* resistant and susceptible hosts have respectively frequencies R_g and r_g and virulent and avirulent parasites, respectively a_g and A_g. As the population size is infinite, host and parasite frequencies are scaled by the mean fitness of the host population $\overline{W_H}$ and parasite population $\overline{W_P}$ respectively. The resistant and the infectivity alleles are costly to their carrier, c_H and c_P. Finally hosts have their fitness reduced by *s*, the disease severity, when infected.

3.2.2 Age-specific seed recruitment and see banking strategy

A quantitative locus determines the rate b_i at which seeds produced *i* generations ago germinates at a given generation *g*. The seed bank length is defined by the fixed parameter *m*. Although all seed can germinate as $\sum_{i=0}^{m} b_i = 1$, seeds die in the bank with probability *d* each generation. A fraction b_0 of newly produced seeds germinates the following generation *g* while a fraction $1\text{-}b_0$ enter the bank – the bank being empty for $b_0 = 1$.

The probability of a dormant seed to germinate depends on the age of the seed. We investigate two shapes of the age-specific seed recruitment. Firstly a constant recruitment with age $b_{i\neq 0} = 1 - b_0/m$ (**Fig. 1**, solid line) and secondly an increasing recruitment with age $b_{i\neq 0} = b_0(1-b_0)^{(m-i)}$ (**Fig. 1**, dashed line). It is important to note that for both cases, the proportion of older seeds being recruited at a given generation *g* is higher than that of a younger seed. This is opposed to the third classic assumption, assuming that the probability to germinate does not depend on the age of the seed, such that the recruitment of the seed in the above ground population geometrically decreases with age (**Fig. 1**, solid line, grey). This latter assumptions has been analysed in the chapter 2.

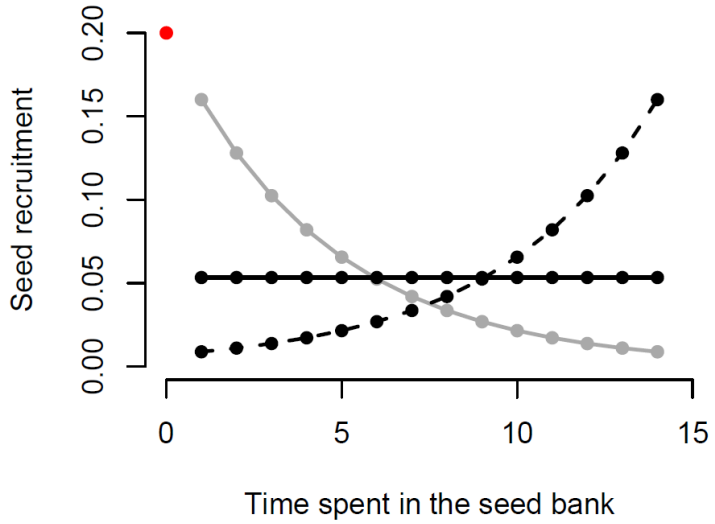


***Figure 3.1**: Age-specific recruitment patterns. The recruitment of a dormant seed is constant over time (solid line, black) or increasing with the age of the seed (dotted line, black). A decreasing recruitment with age (solid line, grey) is shown for comparison but not analyzed in the study (see section 1 for detailed results). The red dot indicates the germination fraction $b_{i=0}=0.2$.*

3.2.3 Evolution of seed banking strategy multiple mutant competition[2]

We perform simulations of competition between multiple mutants (see for example Boots *et al.* 2014). After a burn in phase of 20,000 generations, a number N of mutants with different strategies (different values of b_0*) are introduced in a resident population (with initial value b_0). We use here N=5 (in Boots *et al.* 2014, N=1). Each mutant has a different seed banking strategy, b_0*, which is sampled in a Normal distribution with mean the resident value b_0 and a small standard deviation σ=0.05. The N mutants are introduced with equal frequencies summing to 0.01 (so in effect an introduction frequency of 0.01/N for each mutant). The resident and the mutants compete during a fixed number of generation T=1,000, and we then compute the

2 The multiple mutant competition described in this paragraph is strictly identical to the method described in the chapter 2. Only the investigated parameter space changes.

geometric mean fitness over T for each strategy. The strategy with the smallest fitness amongst the $N+1$ genotypes present (N b_0* mutants and the b_0 resident) is removed from the population, and a new mutant with frequency $0.01/N$ is introduced. We make the assumption that hosts with the highest fitness have higher chances to produce mutants, and that the mutational step is small. Thus the new mutant strategy b_0* to be introduced is sampled in a Normal distribution with a mean equal to the population average strategy $\bar{b_0}$ and a small standard deviation σ=0.05. The average strategy $\bar{b_0}$ is defined as the sum of the remaining strategies (N values of b_0) times their geometric mean of frequency in the population.

Two cases are investigated, (i) independence and (ii) non-independence of germination and resistance loci (*e.g.* linkage equilibrium or disequilibrium). In other words, either resistant and susceptible types have the same germination strategy which is then the strategy of the population ($b_0=b_{r0}=b_{R0}$), or these alleles can evolve each their own strategy ($b_{r0}\neq b_{R0}$). The method described above corresponds to the first case (linkage equilibrium). In the case of linked loci, we amend the above simulation protocol by removing and then adding at a given time point one resistant and one susceptible mutant. The mutant strategies are sampled around the average strategy of each type $\bar{b_{R0}}$ and $\bar{b_{r0}}$, respectively.

I investigate the evolution of seed banking under coevolution for one parameter combination only: high costs c_H and c_P equal to 0.2 and a medium disease severity s equal to 0.3 for a fixed seed bank persistence m varying from 1 year to 20 years. For each set of parameters, I perform 50 repetitions, and record over $2x10^6$ generations the population mean strategy $\bar{b_0}$, or the resistant and susceptible mean strategies $\bar{b_{R0}}$ and $\bar{b_{r0}}$ to account for the variability of the mutation sampling procedure.

3.3 Results

3.3.1 Stability outcome for fixed seed banking strategies

Seed banking provides a memory of past selective events and modify the response of the GFG loci to natural selection. This generates negative direct FDS that can stabilise polymorphism. Whether the dynamics cycle towards the internal equilibrium depends on the delay between the production of the seed and its potential germination.

Considering a constant seed recruitment with age (**Fig. 3.1**, solid line) we observe that under 7 years of persistence (**Fig.3.2**, left panel), the dynamics is driven by iFDS and does not reach an equilibrium. From 7 to 12 years of persistence we observe cycles of shorter amplitude, indicating that the seed bank generates direct FDS but does not balance out iFDS. Above the persistence threshold of 12 years, stable dynamics may arise, depending on the ratio $b_0/(1- b_0)$ – the ratio of seeds germinating following production to seeds going in the bank. When very little to no seeds enter the bank the dynamics are unstable, as the germination input from the seed bank is negligible. On the other hand, if too many seeds enter the bank, dynamics are also unstable, since the seed bank genetic constitution is then too similar to the above ground population. In other words the seed bank does not act as a memory of past selective events.

It is for high germination fractions (*e.g.* b_0=0.8) that we observe stabilised polymorphism. Indeed the genetic composition of the seed bank is clearly distinct from the above ground population and the amount of dormant seeds germinating is sufficient to generate direct FDS. This mechanism acts in interaction with the persistence *m* and the age-specific seed recruitment, as the range of germination rate leading to stable dynamics is wider for increasing persistence, showing again the importance of a sufficient time delay.

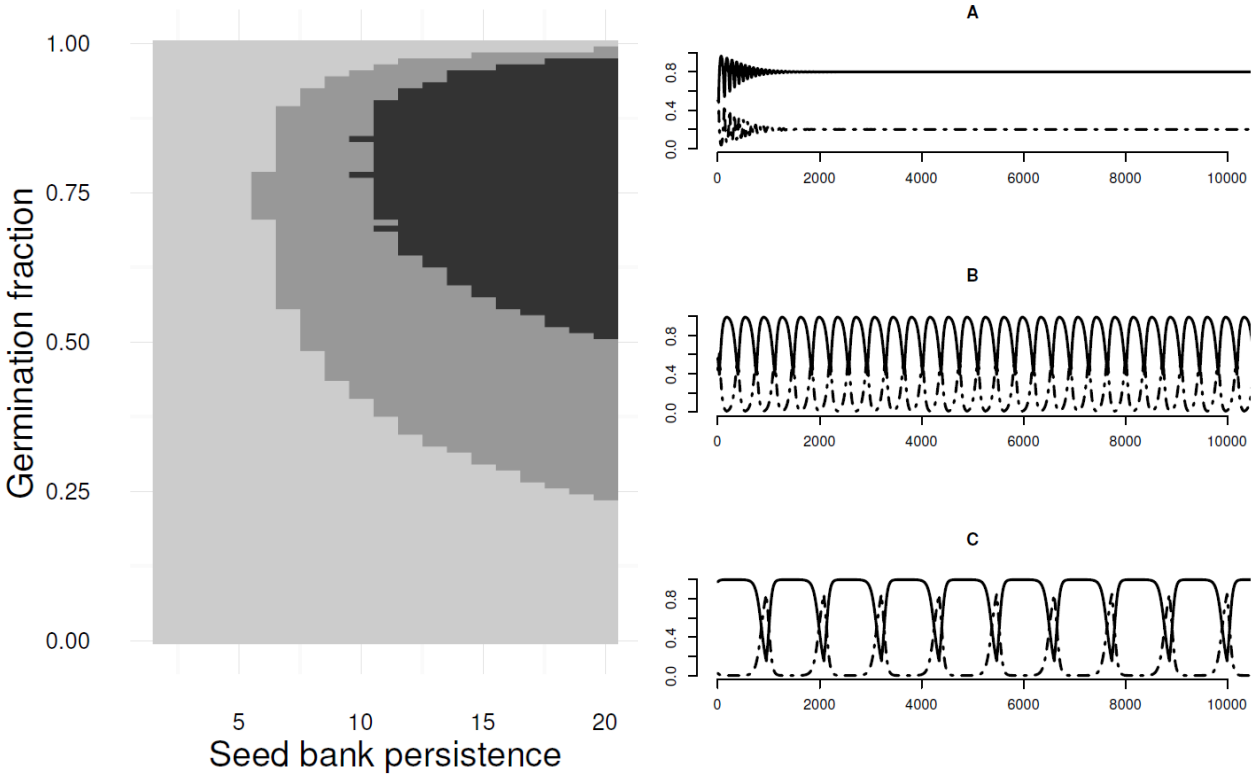


***Figure 3.2**: Host/parasite coevolutionary dynamics for varying host seed bank persistence m (x axis) and germination fraction of the host population b_0 (yaxis). The age-specific recruitment pattern is constant. Costs are fixed, c_H=c_P=0.2, s=0.3 and d=0.002. The dynamics is unstable (light grey), reaches a limit cycle (grey) or the internal equilibrium state (black). Susceptible host (solid line) and resistant host (dashed line) relative frequencies over time, for a seed bank persistence m of 15 years and germination fractions, A) b_0=0.8 B) b_0=0.4 C) b_0=0.2 for fixed costs c_H=c_P=0.2, s=0.3 and d=0.002*

Now, allowing strategies to differ between hosts types – same persistence *m* but b_{r0} different from b_{R0} – changes the balance between indirect and direct frequency dependent selection (**Fig. 3.3**, for fixed *m*=15 years). The direct FDS generated by a susceptible seed bank stabilises the dynamics in a population composed of strictly non seed banking resistant b_{R0}=1. Yet this is constrained by the previously described mechanisms, as for susceptible strategies above a threshold (here b_0=0.78) the seed bank selective does not generate ndFDS and dynamics are unstable. In the opposite configuration – non seed banking susceptible b_{r0}=1 – resistant seed banking strategies show a reduced influence on the dynamics, with stable polymorphism

observed for reduced range of germination rates ($0.48<b_0<0.95$). Otherwise when host types have close germination values, the mechanisms described in the previous section apply.

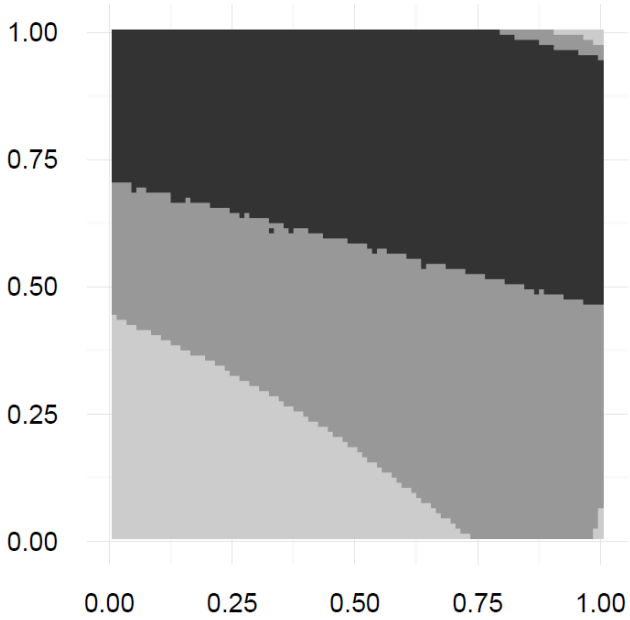


***Figure 3.3**: Host/parasite coevolutionary dynamics for varying susceptible germination fraction (x-axis) and resistant host germination fraction (y-axis). The seed bank persistence m is fixed to 15 years. The age-specific recruitment pattern is constant. Costs are fixed, $c_H=c_P=0.2$, $s=0.3$ and $d=0.002$. The dynamics is unstable (light grey), reaches a limit cycle (grey) or the internal equilibrium state (black).*

Similar outcomes are observed when considering an increasing recruitment with age (F**ig. 3.1**, doted line), but the influence of seed banking is stronger. Firstly, when host types have the same strategy, stabilised polymorphism is observed for a persistence of 7 years (**Fig. S3.1**) compared to 12 years previously (**Fig. 3.2**). Secondly, when host strategies differ, we observe a wider range of strategies combinations leading to stabilised dynamics (**Fig. S3.3**).

3.3.2 An eco-evolutionary feed back

We observe contrasted results for the simulations of the evolution of the seed banking strategies under coevolution, allowing us to understand the complex interplay between (i) fluctuating slow selection generating indirect FDS, and (ii) direct FDS and germination delay. The most telling results occurs for non-independent loci of coevolution and germination, for which we perform simulations of the evolution of the germination rate b_0 in hosts with fixed seed bank lengths m varying from 1 year to 20 years. In the global picture **Fig. 3.4**, we present the mean germination rates $\bar{b}_{R0}$ and $\bar{b}_{r0}$ of host populations at the end of the simulation process, with corresponding variance over the 50 repetitions of each parameter set. The detailed evolution over time for each value of m is given in (**Fig. S3.3-4**).

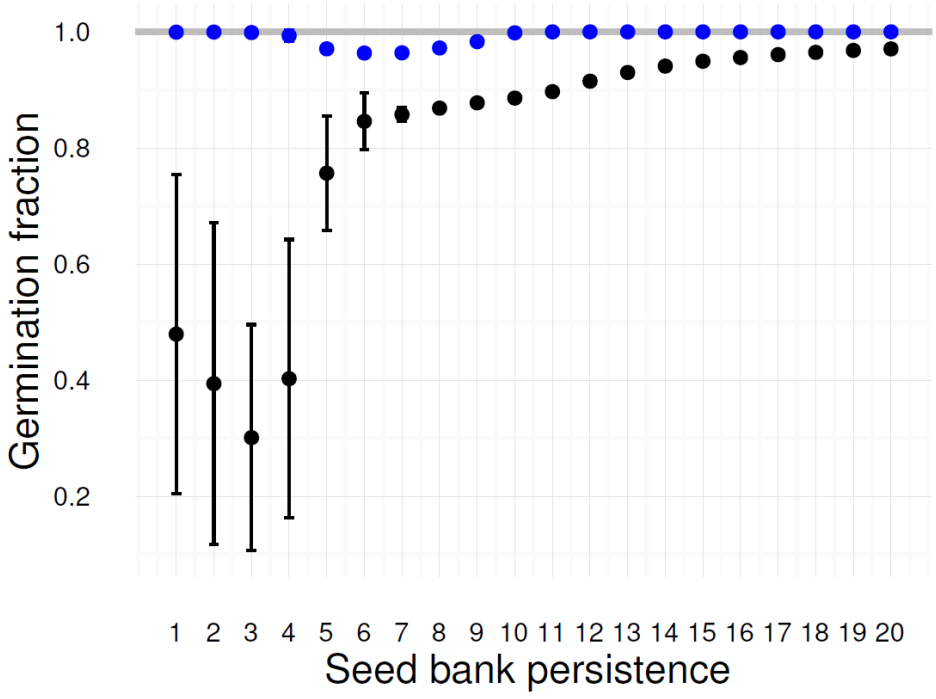


***Figure 3.4**: Resistant mean germination fraction $\bar{b}_{r0}$ (black, yaxis) and susceptible mean germination fraction $\bar{b}_{R0}$ (blue, y-axis) at the end of the multiple mutant simulation process for varying seed bank persistence m (x-axis). Errors bars indicate variation over the 50 repetitions per parameter set. The age-specific recruitment pattern is constant. Costs are fixed, cH=cP=0.2, s=0.3 and d=0.002. The initial germination fraction is $b_{0R}=b_{0r}=0.2$.*

We classify our results in three categories depending on the selective forces driving the evolution of the strategy. In the first category, the seed bank length is short (< 4 years), and the germination delay is not sufficient to provide direct FDS to the host population. This result in the loss of the seed bank for the susceptible host b_{r0}=1 and the evolution of highly variable seed banking strategies for the resistant host. Above 4 years, the seed bank length shows benefits, but the direct FDS cannot balance out the indirect FDS when under 12 years. This defines the second category of results, where both host type evolve a seed bank. We can note that the investment in the seed bank is stronger for resistant than for susceptible hosts (*i.e.* higher fraction of seeds entering the bank). We observe an increasing resistant mean strategy $\bar{b}_{R0}$ with increasing persistence, while the susceptible investment in the bank is slightly stronger for a persistence of 6 year than compared to 5 or 8 years.

For 12 years of persistence and above, the germination delay may lead to stable polymorphism, defining our third category of results. Now negative direct FDS may overcome iFDS, revealing an eco-evolutionary feed back between the evolving strategies and coevolution. The global results (**Fig. 3.4**) show a strict loss of the seed bank for susceptible hosts, and the evolution of very high germination rates $\bar{b}_{R0}$ for resistant hosts, with rates increasing with *m*. This is only a partial view of the results, the detailed evolution of the strategy over time (**Fig. S3.3-4**) reveal an interesting pattern: The resistant strategy firstly evolves towards very high b_{R0} fractions – resistant hosts loosing their seed bank in many cases – then the susceptible strategy evolves towards high b_{r0} too, yet this is shortly followed by a decrease of the resistant host strategy – resistant hosts replenishing their seed bank again. This succession of evolutionary events is due to an eco-evolutionary feed-back. To better understand it, we need to take into account the stability shifts of the coevolutionary dynamics occurring over time, resulting from the movement of the resistant and susceptible strategies in the parameter space – as illustrated in

Fig. 3.3 for a fixed persistence *m* of 15 years, drawing the movement of the pair (b_{R0} , b_{r0}) over time from one simulation (red line, **Fig. 3.5**). Initially the coevolutionary dynamics are unstable **(1)**, the resistant strategy rapidly evolves towards higher germination rates (fast jumps along the y-axis, high b_{R0}) and the dynamics switches to stability **(2)**. As soon as a stable polymorphic state is reached, we observe high jumps of the susceptible strategy (x-axis, b_{r0}), while the resistant progressively looses its seed bank (b_{R0} = 1). Then, only the susceptible strategy moves towards high rates, shifting back the dynamics to instability **(3)**. This latter shift is followed by the decrease of the resistant strategy (b_{R0} <1), moving towards the stable parameter space, while the susceptible host progressively looses its seed bank. This results in several push backs alongside the stability/instability border, until the susceptible reaches its optimal germination fraction, the loss of its seed bank b_{r0}=1 **(4)**. Consequently the resistant host strategy evolves towards its local optimum, that is the germination fraction b_{R0} = 0.98. This strategy of the resistant hosts lies at the border between stability and instability.

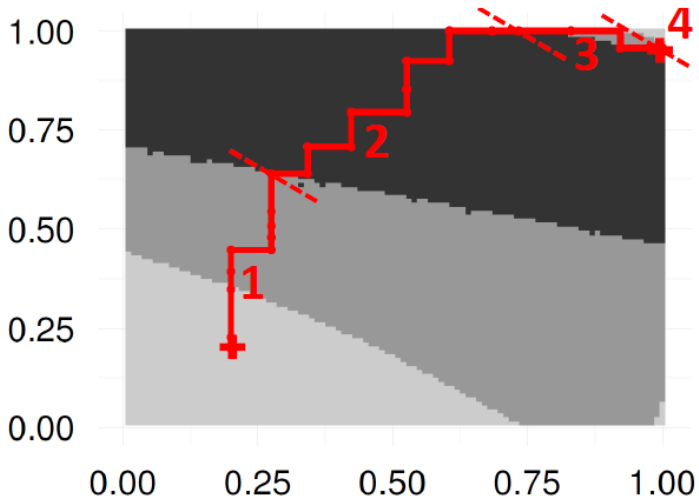


***Figure 3.5**: Stability shifts through the multiple mutant simulation process. Host/parasite coevolutionary dynamics for varying susceptible germination fraction (x-axis) and resistant host germination fraction (y-axis). The seed bank persistence m is fixed to 15 years. The age-specific recruitment pattern is constant. Costs are fixed, c_H=c_P=0.2, s=0.3 and d=0.002. The dynamics is unstable (light grey), reaches a limit cycle (grey) or the internal equilibrium state (black).*

We obtain corresponding results considering an increasing seed recruitment with age (**Fig. 3.6**). As this implies stronger fitness advantages of the seed bank, we observe the eco-evolutionary feedback for a wider range of persistence *m*, from 7 years to 20 years. Between 3 and 7 years of persistence both hosts evolve a seed bank in the same manner as described above. Finally under 3 years of persistence the susceptible host looses its seed bank while the resistant shows highly variable seed banking strategies (both over time and between repetitions). The detailed evolution over time for each value of *m* is given in (**Fig. S3.5-6**).

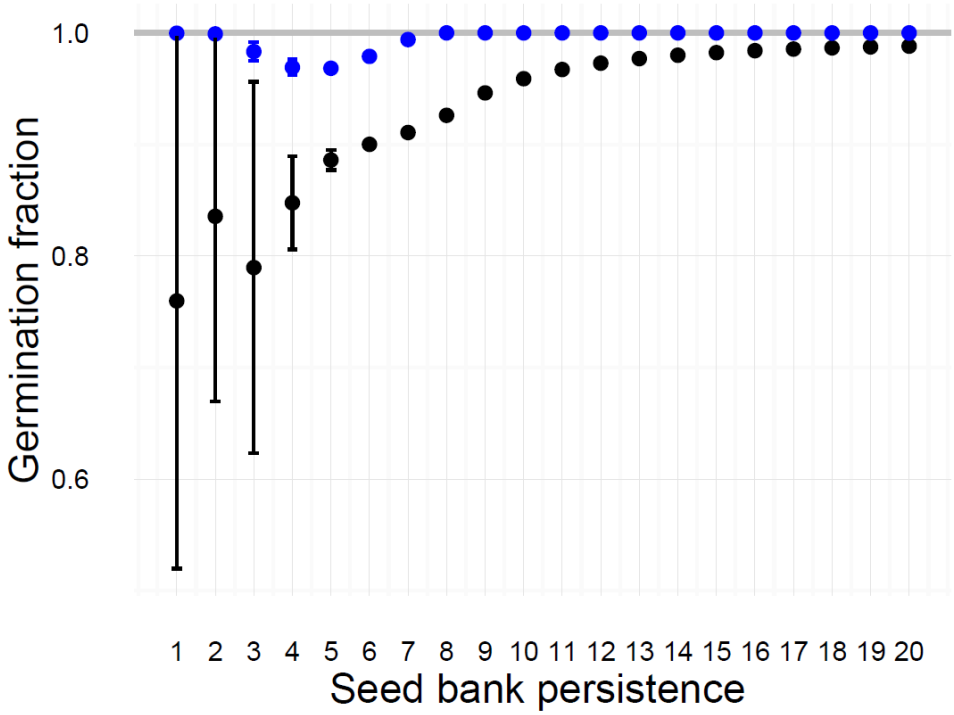


***Figure 3.6**: Resistant mean germination fraction $\bar{b}_{r0}$ (black, yaxis) and susceptible mean germination fraction $\bar{b}_{R0}$ (blue, y-axis) at the end of the multiple mutant simulation process for varying seed bank persistence m (x-axis). Errors bars indicate variation over the 50 repetitions per parameter set. The age-specific recruitment pattern is increasing. Costs are fixed, c_H=c_P=0.2, s=0.3 and d=0.002. The initial germination fraction is b0R=b_{0r}=0.2.*

Unlinked loci

By considering unlinked loci, we reduce the parameter space in which strategies evolve, we now investigate the evolution of the strategy at the level of the population such that $\bar{b}_{R0} = \bar{b}_{r0} = \bar{b}_0$. We show results for fixed seed bank length of 5 and 15 years only. We observe the evolution towards a small investment in the bank $\bar{b}_0$ =0.95 for a short term persistent bank of 5 years and for both age-specific seed recruitment investigated. As for a long term persistent bank of 15 years, the seed bank is strictly lost $\bar{b}_0$ =1, again for both age-specific seed recruitment. In both context the evolution of the population mean strategy is driven by the susceptible host, which is the most common host in the population over time. The loss of the seed bank for a persistence of 15 years is then due to the feedback between the coevolutionary dynamics and the evolution of seed banking (**Fig. 3.7**)

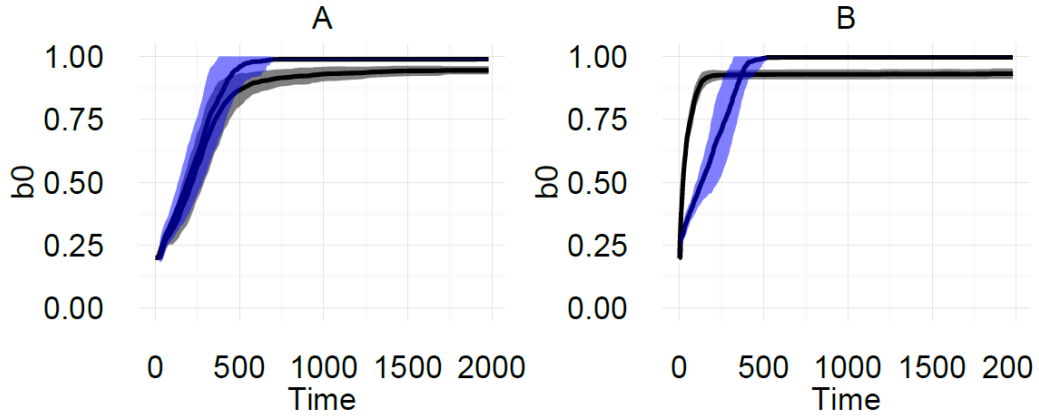


***Figure 3.7**: Evolution of the host population mean germination fraction $\bar{b}_0$ (+-sd) under multiple competition considering A) constant B) increasing recruitment of seed with age (GFG model, unlinked loci), for short term persistence m=5 (black) and long term persistence m=15 (blue) over 50 repetitions. Costs are fixed to c_H=c_P=0.2, s=0.3, and d=0.002. The initial resident value is b_0=0.2.*

3.3.3 Seed banking under Matching allele coevolution

We modify our model to include a Matching Allele recognition matrix in which both hosts are infected by their specific parasite and have equivalent fitness fluctuations[3]. Both host types H_1 and H_2 share the same germination fraction optimum, and always evolve a seed banking strategy independently of the age-specific seed recruitment assumed (**Fig. 3.9**). Initially the coevolutionary dynamics are unstable, then the strategies of H_1 and H_2 evolve towards higher germination rates which switches the dynamics to stability, for both short term and long term persistent banks when assuming a constant age-specific recruitment, but only for short term persistent banks when assuming an increasing age-specific recruitment. Indeed for long term persistent banks the dynamics exhibits cycles of small amplitudes. The results are strictly equivalent when assuming unliked loci (**Fig. S3.7**), however the simulation process did not reach a plateau in the context of long term persistent banks with an increasing age-specific recruitment.

3 The MA model described in this paragraph is strictly identical to the model described in chapter 2

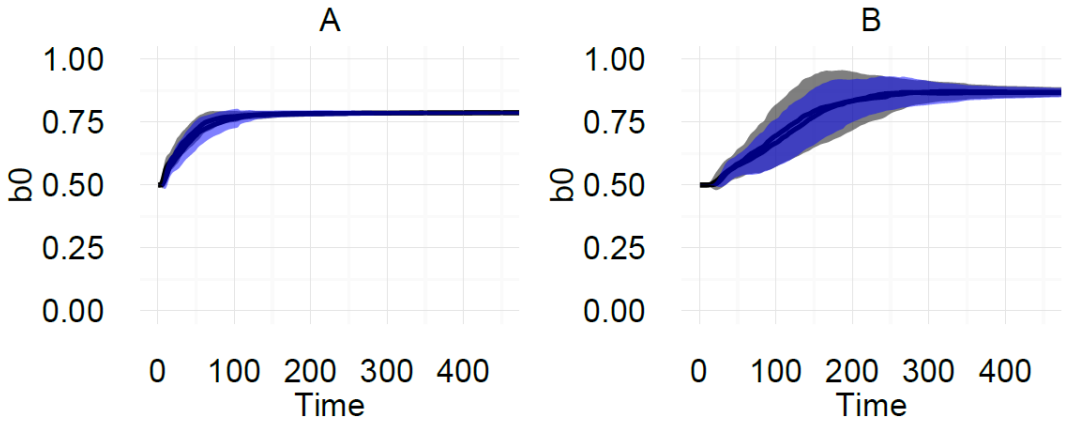


Figure 3.8: *Evolution of the mean germination fraction (+- sd) of the host one (black) and host two (blue) under multiple competition (MA model, linked loci), the age-specific seed recruitment is constant with age. The fixed seed bank persistence is A) 5 years B) 15 years. Costs are fixed to s=0.3, d=0.002. The initial resident value is* b_0*=0.5.*

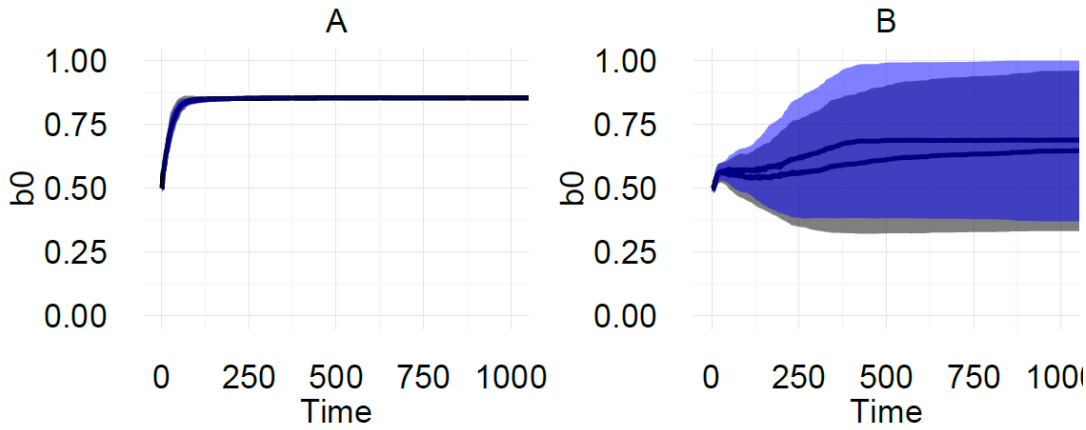


Figure 3.9: *Evolution of the mean germination fraction (+- sd) of the host one (black) and host twoe (blue) under multiple competition (MA model, linked loci), the age-specific seed recruitment is increasing with age. The fixed seed bank persistence is A) 5 years B) 15 years. Costs are fixed to s=0.3, d=0.002. The initial resident value is* b_0*=0.5.*

3.4 Discussion

We show here a feedback between the evolution of host seed banking strategies and the host-parasite coevolutionary dynamics. Hosts seed banking strategies are evolving in response to fluctuating selection acting on the resistance/infectivity loci involved in the host-parasite interaction. Fluctuating selection is generated by negative indirect frequency-dependent selection niFDS, in which the strength of natural selection acting on resistance genes depends on the frequencies of parasite genes and vice-versa, leading to unstable fluctuations of allele frequencies over time. Previous work show that host seed banking evolves as a bet-hedging strategy in such context (Verin & Tellier 2018, see chapter 2), meaning that hosts should evolve strategies that dampens the fluctuations of allele frequencies. Yet fluctuations could not converge towards a stable equilibrium as the seed bank was assuming a decreasing seed recruitment with age.

The novelty of our study is to relax this latter hypothesis, enabling the evolution of seed banking strategies that promote sufficient negative direct frequency-dependent selection (in which the contribution of an allele to fitness declines as its frequency increases) so that coevolutionary dynamics can converge towards a stable equilibrium. The stability of the internal equilibrium depends on the strength of ndFDS versus FDS (Tellier & Brown 2007, 2009). In the context of seed banking, we show that the strength of ndFDS is function of a long seed bank persistence *m* and an age-specific seed recruitment of a constant or increasing shape.

Stabilised coevolutionary dynamics generate a novel environment for hosts and parasites which has huge consequences on their fitness and the evolution of bet-hedging. In a stable environment, organisms are expected to maximise their arithmetic mean fitness, and not their

geometric mean fitness as previously under unpredictable environmental fluctuations (Cohen 1966). At such stable equilibrium state, the best strategy for the susceptible host is to loose the seed bank (b_{r0}=1), as we observe in **Fig.3.4-5** considering linked loci, and in **Fig.3.6** for unlinked loci. In return this switches back the state of the coevolutionary dynamics to instability. As such, stabilised coevolutionary dynamics feed back on the evolving seed banking strategies. We can observe this feedback throughout the course of the multiple mutant simulation process. Once the susceptible strategy jumps towards the seed bank loss, it shifts back the dynamics to instability, with has high consequences on the resistant host fitness. The resistant strategy is then attracted towards the stable parameter space, while the susceptible host definitely looses its seed bank, maintaining unstable coevolutionary dynamics. Consequently the seed banking strategy evolves towards its local optimum, which is positioned at the border between stability and instability of the coevolutionary dynamics.

Host/parasite interactions relying on fitness asymmetry between host and parasite types, such as interactions involving genotypes of varying specialisation degrees (GFG, iGFG), will promote such feedbacks. The reasoning being that the various genotypes will show distinct optimum strategies that might be conflictual depending on the equilibrium state of the coevolutionary dynamics. In the GFG model we describe here, the resistant host undergo extreme frequency fluctuations over time – fast frequency increase but of short periods – and both its arithmetic and geometric mean fitness are maximised when dynamics are stable. It is the evolution of the resistant host strategy that results in stabilised coevolutionary dynamics and initiate the feedback, but the evolutionary response in the susceptible host constrains the resistant host strategy to a sub-optimal strategy. When the GFG and germination loci are unliked, the evolution of the seed banking strategy is driven by the susceptible host evolving towards its respective optimum. If the fitness asymmetry is released, as in the MA model, the two hosts

types share the same optimum and evolve towards it. Therefore once a constant or an increasing age-specific recruitment is assumed, seed banking is more likely to evolve under MA than under GFG in response to coevolution.

This work further highlights the issue of separating the ecological time scale – the coevolutionary dynamics – from the evolutionary time scale – the evolving seed bank. In our system, the life history traits and the dynamics are both evolving continuously, requiring simulations methods of analyse. We also show that maintenance of polymorphism in the host and parasite populations can vary over time, depending on the evolution of seed banking which affects the stability of the coevolutionary dynamics.

Supplementary figures

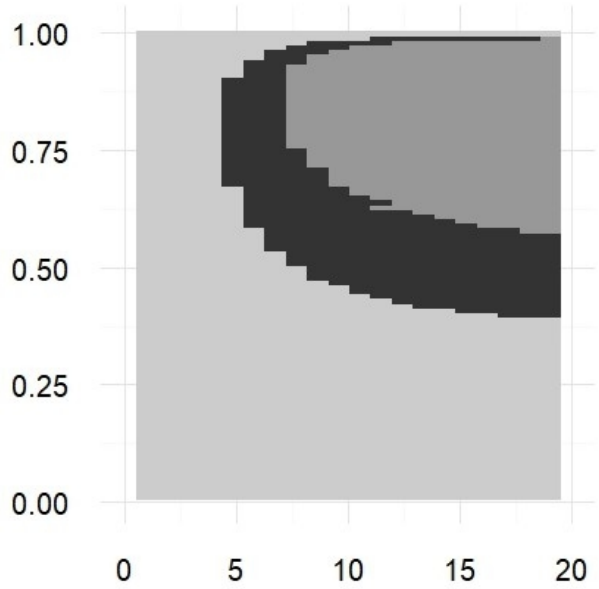


***Figure S3.1**: Host/parasite coevolutionary dynamics for varying host seed bank persistence m (x axis) and germination fraction of the host population b_0 (yaxis). The age-specific recruitment pattern is increasing with age. Costs are fixed, $c_H=c_P=0.2$, $s=0.3$ and $d=0.002$. The dynamics is unstable (light grey), reaches a limit cycle (grey) or the internal equilibrium state (black).*

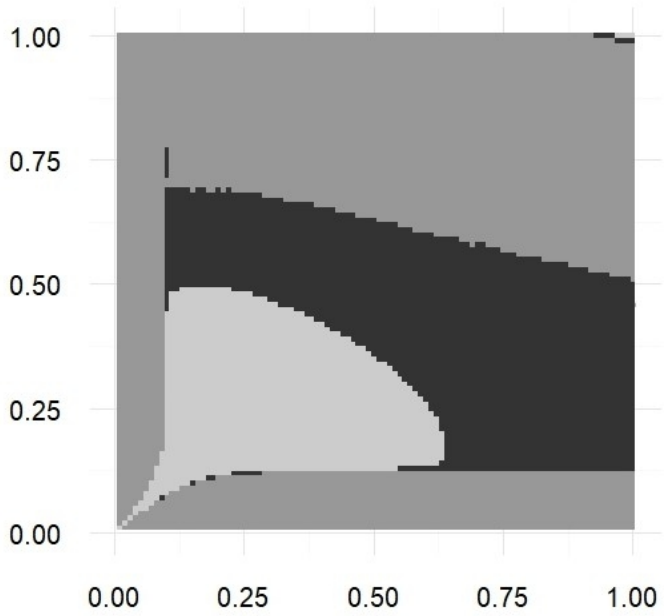


***Figure S3.2**: Host/parasite coevolutionary dynamics for varying susceptible germination fraction (x-axis) and resistant host germination fraction (y-axis). The seed bank persistence m is fixed to 15 years. The age-specific recruitment pattern is increasing with age. Costs are fixed, $c_H=c_P=0.2$, $s=0.3$ and $d=0.002$. The dynamics is unstable (light grey), reaches a limit cycle (grey) or the internal equilibrium state (black).*

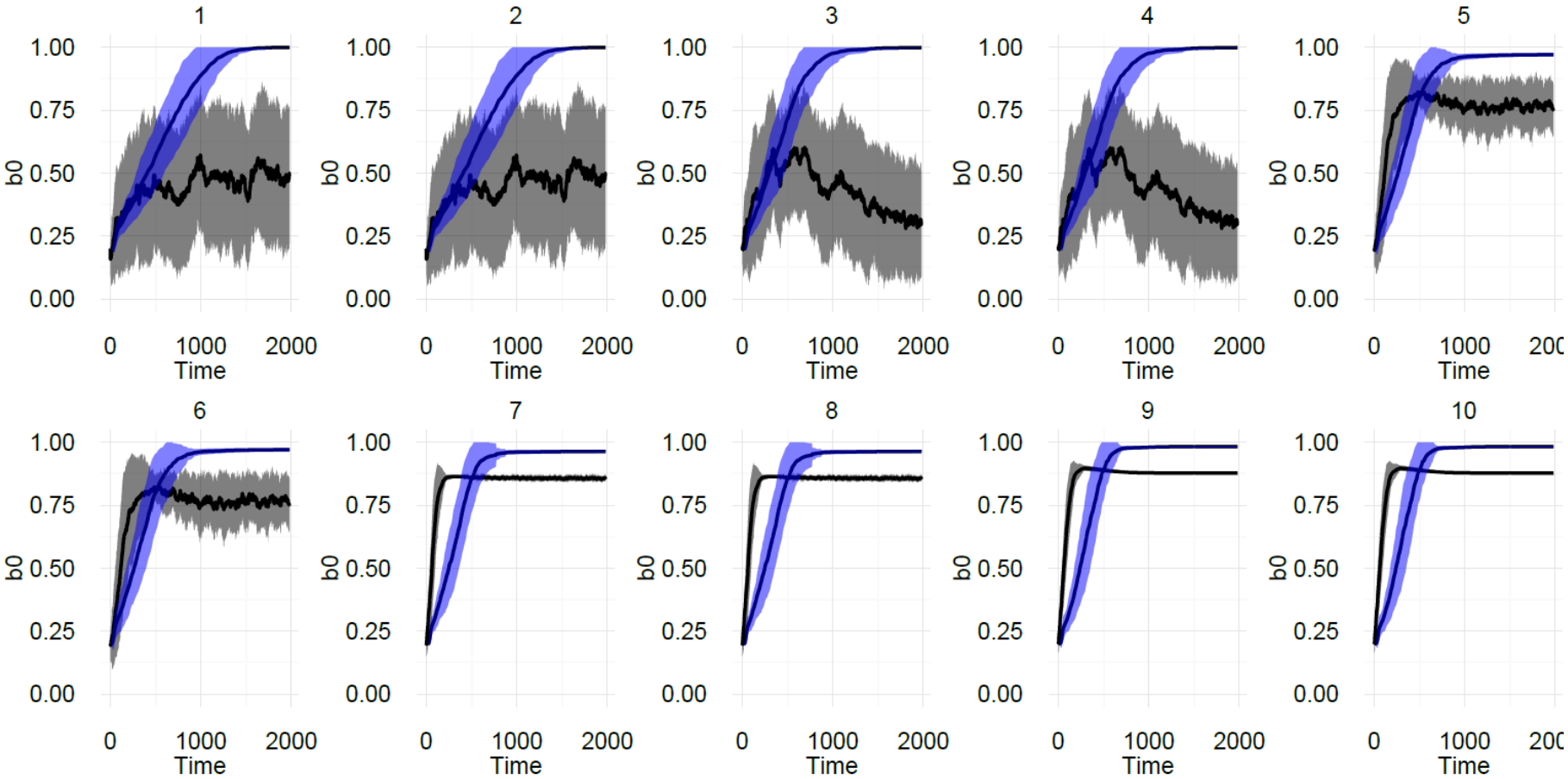


Figure S3.3: *Evolution of the mean germination fraction (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for a* ***fixed seed bank persistence varying from 1 year to 10 years, seed recruitment is constant with age****. Costs are fixed,* $c_H=c_P=0.2$*,* $s=0.3$*,* $d=0.002$*. The initial resident value is* $b_0=0.2$*.*

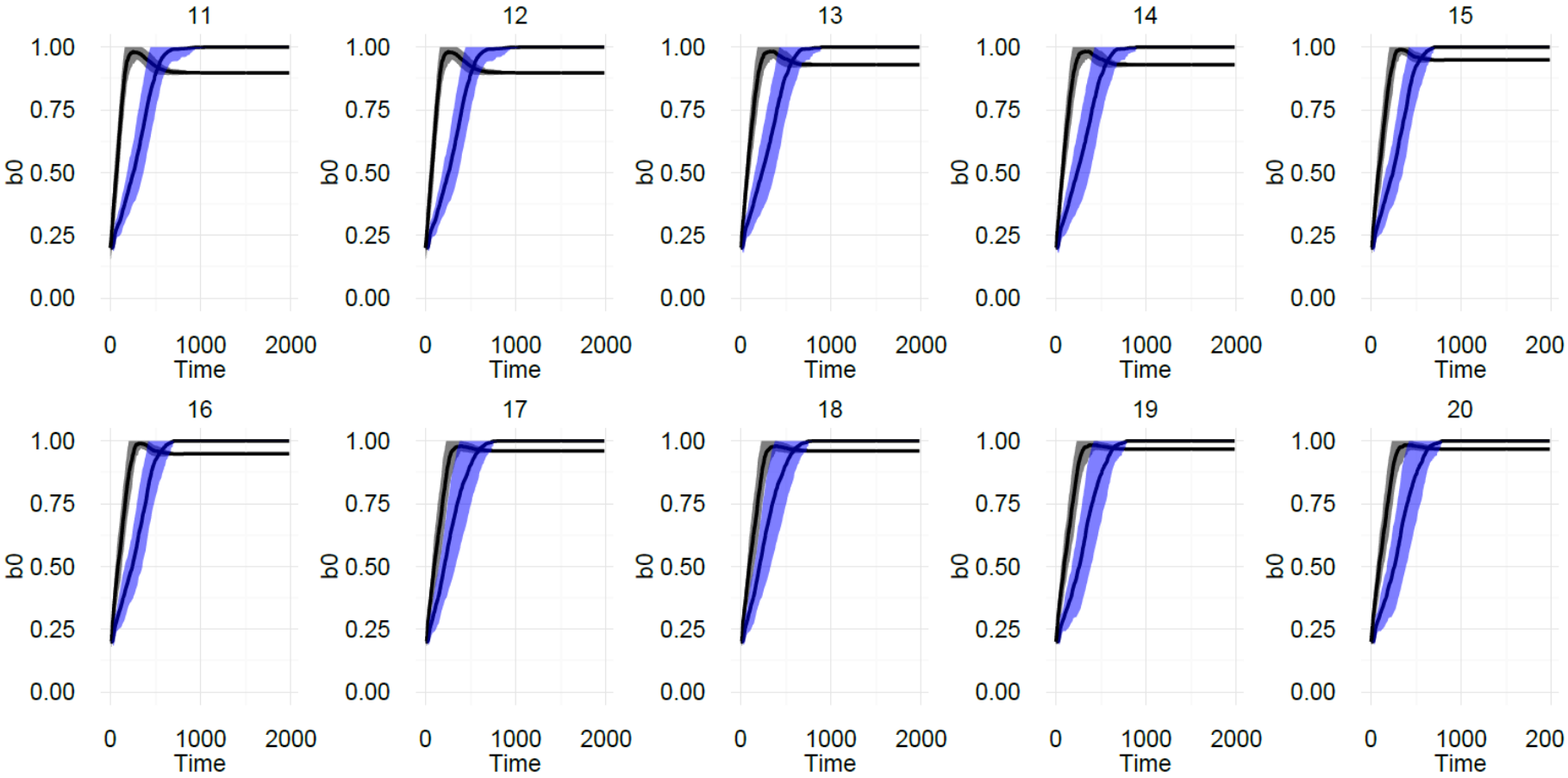


***Figure S3.4**: Evolution of the mean germination fraction (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for a fixed **seed bank persistence varying from 11 year to 20 years, seed recruitment is constant with age**. Costs are fixed, $c_H=c_P=0.2$, $s=0.3$, $d=0.002$. The initial resident value is $b_0=0.2$.*

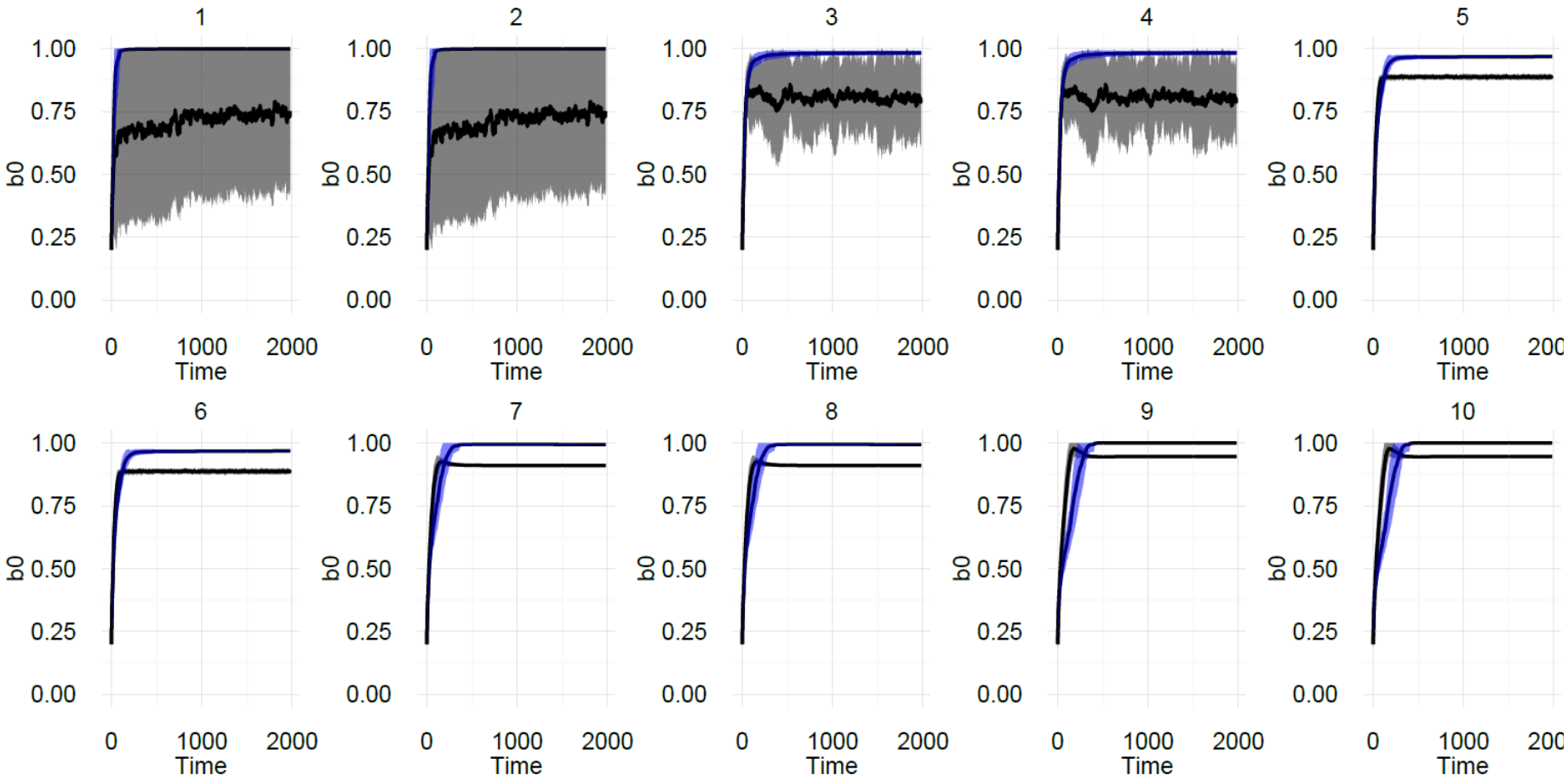


***Figure S3.5**: Evolution of the mean germination fraction (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for a fixed **seed bank persistence varying from 1 year to 10 years, seed recruitment is increasing with age**. Costs are fixed, $c_H=c_P=0.2$, $s=0.3$, $d=0.002$. The initial resident value is $b_0=0.2$.*

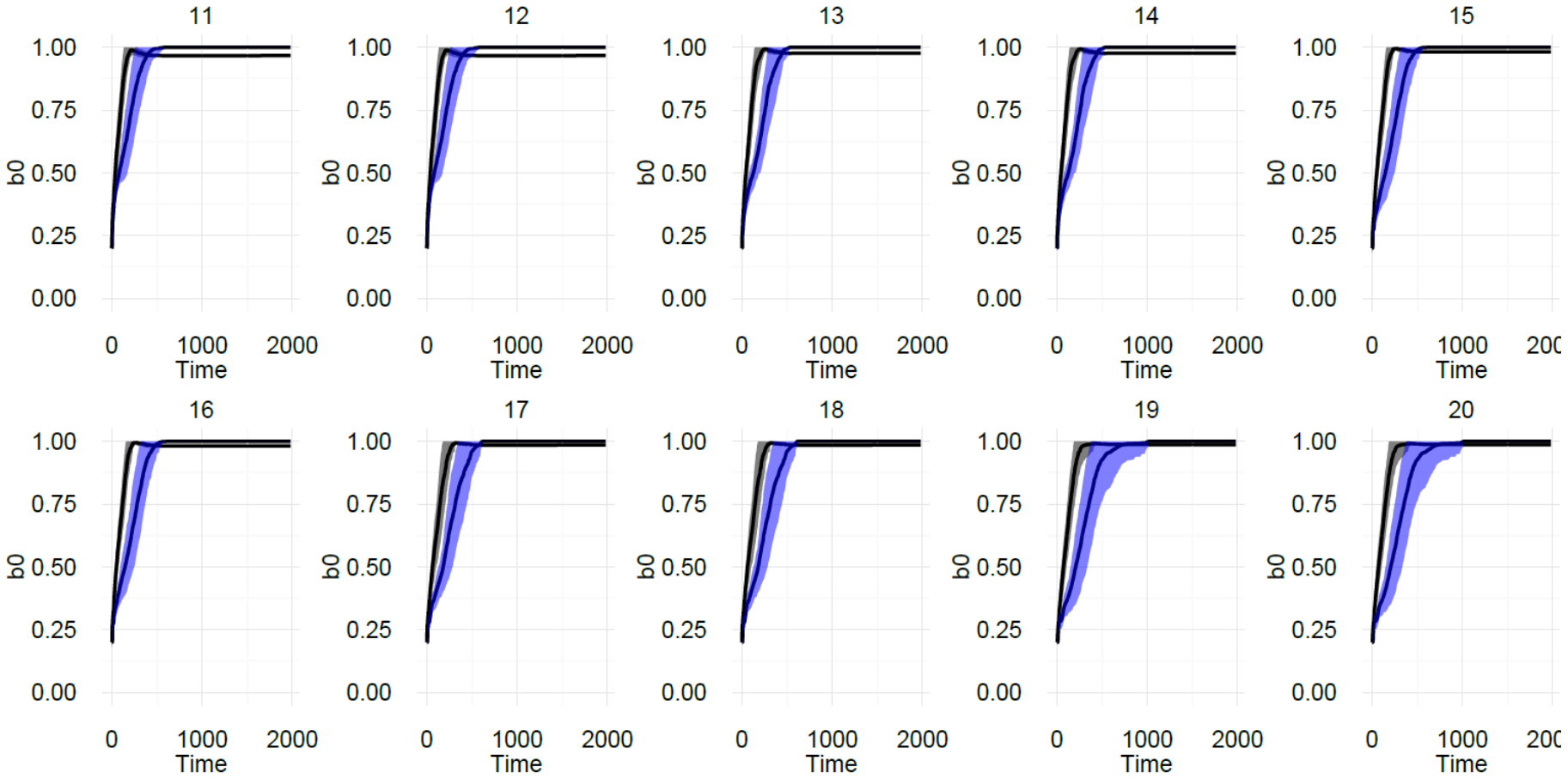


Figure S3.6: *Evolution of the mean germination fraction (+- sd) of the susceptible (blue) and resistant (black) host under multiple competition (GFG model, linked loci), for a fixed* ***seed bank persistence varying from 11 year to 20 years, seed recruitment is increasing with age****. Costs are fixed,* $c_H=c_P=0.2$*,* $s=0.3$*,* $d=0.002$*. The initial resident value is* $b_0=0.2$*.*

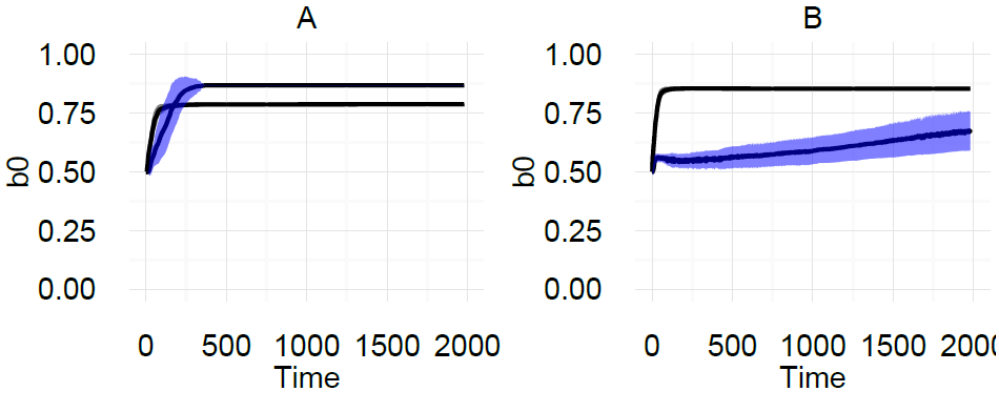


Figure S3.7*: Evolution of the host population mean germination fraction* $\bar{b}_0$ *(+-sd) under multiple competition considering A) constant B) increasing recruitment of seed with age (MA model, unlinked loci), for short term persistence m=5 (black) and long term persistence m=15 (blue) over 50 repetitions. Costs are fixed to s=0.3, and d=0.002. The initial resident value is* b_0*=0.5.*

Density-dependent regulation and environmental stochasticity

4.1 Introduction

Environmental stochasticity, in the sense of unpredictable abiotic variations is central to the hypothesis of bet-hedging. Density-dependent processes (*i.e.* population growth regulation) also play a major role in the evolution of diversified bet-hedging strategies, amplifying the impact of environmental stochasticity (Ellner 1985). The evolution of dormancy strategies in response to both sources of variations has been well studied (Templeton & Levin 1979; Charlesworth 1980; MacDonald & Watkinson 1981; Bulmer 1984; Ellner 1985a, b, 1987; Tuljapurkar 1989). But this is not the case concerning seed banking strategies, indeed no theoretical model assume both a limited seed persistence in the soil seed bank and an age-dependent seed germination.

The aim of this chapter is to study the evolution of seed banking strategies in response to (i) coevolution and density-dependency and (ii) coevolution, density-dependency and environmental stochasticity. We therefore extend the previous host/parasite model of coevolution by including both sources of variation. Introducing population regulation and thus finite population size in the model is an arduous task. That is why as a first step we assume that only the host population is regulated, while the parasite population remains infinite. Exactly as the previous model (described in chapters 2 and 3), all hosts receive parasites at a given generation *g*. Consequently, the success of infection does not depend on the density of the host population but only on the frequency of each host type (either resistant or susceptible). Likewise host infection only depends on the frequency of each parasite type (infective or non infective). Regarding environmental stochasticity, assuming its influence on the infinite parasite population is totally irrelevant. Thus density dependency and environmental stochasticity represent two

additional sources of variability for the host population only, allowing us to evaluate their joint influence on the evolution of seed banking strategies.

Extending the model to a finite parasite population size represent a bigger challenge as it requires further assumption about the transmission of the disease (*i.e.* the relation between the density of hosts and the transmission rate) which itself depends on the life history cycle of the parasite (*e.g.* endoparasites, ectoparasites, vector-borne). The model and results presented in this section are thus preliminary only, but provide a valuable source of reflection for potential extensions of the general model.

4.2 Materials and methods

4.2.1 Density-dependency

The coevolutionary models analysed in the previous chapters describe hosts and parasites population with an unlimited amount of resources in their environment, thus assuming an infinite growth. We now introduce density dependent regulation in the host population.

We assume a logistic growth of type $r(1-\frac{N_{Host}}{K})$ of the host population, for which the population growth rate decreases linearly as the population size N_{Host} increases (**Fig. 1A**). The parameter K defines the carrying capacity (*i.e.* the maximum amount of individuals that the environment can sustain) and the parameter r defines the maximum rate of growth per individual of the population. To avoid any confusion with the hosts genotypes named R and r, we name it δ in the model. For population size N_{Host} smaller than K, the population grows almost exponentially. As N_{Host} increases, the population growth rates start to decline; and at K itself the population size neither increases nor decreases. The two host types belong to the same population and compete against each other for the same resources, thus the growth of a host of type 1 and of a host of type 2 follows the following logistic function:

$$\delta(1-\frac{N_{H1}+N_{H2}}{K})$$

In terms of the life history of the annual plant, we now assume that density-dependency regulates the germination of seeds at a given generation g. Then all successfully germinated seeds, which are adult plants receive parasites and can become infected (Equations 4.1). The parasite population is not regulated and remains as an infinite population size.

The following recurrence equations describe the host population:

$$R_{g+1}=R_g\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_R,g)} b_i(1-d)^i R_{g-i}+\sum_{i=0}^{min(m_r,g)} b_i(1-d)^i r_{g-i}}{K}\right]\right)(1-c_H)(1-s(1-A_g))$$

$$r_{g+1}=r_g\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_r,g)} b_i(1-d)^i r_{g-i}+\sum_{i=0}^{min(m_R,g)} b_i(1-d)^i R_{g-i}}{K}\right]\right)(1-s) \qquad \text{(Equations 4.1)}$$

To simplify the equations we can introduce the terms DR and Dr, corresponding to the regulated host population size prior infection for resistant and susceptible hosts respectively,

$$DR=R_g\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_R,g)} b_i(1-d)^i R_{g-i}+\sum_{i=0}^{min(m_r,g)} b_i(1-d)^i r_{g-i}}{K}\right]\right)(1-c_H)$$

$$Dr=r_g\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_r,g)} b_i(1-d)^i r_{g-i}+\sum_{i=0}^{min(m_R,g)} b_i(1-d)^i R_{g-i}}{K}\right]\right)$$

the parasite population is then simply described with:

$$A_{g+1}=A_g\left(\frac{Dr}{Dr+DR}\right)/\overline{W_P}$$

$$a_{g+1}=a_g\left(\frac{Dr}{Dr+DR}+\frac{DR}{Dr+DR}\right)/\overline{W_P}$$

So far the model always assumed a deterministic abiotic environment. As a final extension of the model, we now introduce environmental stochasticity. Since only the host population is density regulated, the impact of environmental stochasticity is direct on hosts survival, but only secondary on parasites.

4.2.2 Environmental stochasticity

The environment can either be unfavourable with probability P_b or favourable with probability $(1-P_b)$. At each generation g a random number is sampled from a uniform distribution $U(0,1)$, and if lower than P_b the conditions are defined as unfavourable.

Bet-hedging models usually assume a complete reproductive failure under unfavourable conditions. However, this current model concerns annual plants with a defined seed bank persistence of 5 years or 15 years. In this context, such drastic influence of environmental stochasticity invariably leads to the extinction of the host population. We instead reduce the seed production under unfavourable conditions, either by a fixed factor of 10, or by a random factor sampled in a uniform distribution $U(0,1)$.

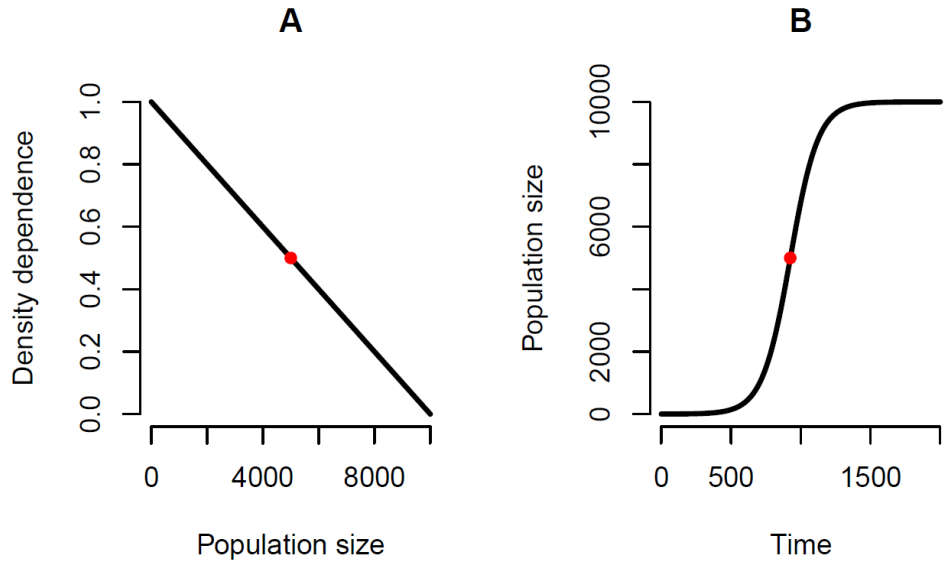


***Figure 4.1**: Logistic growth for K=10000 A) The density-dependent regulation increases linearly with the population size K= B) Sigmoid shape r=0.01. The red dot indicates the population size at which the growth rate is divided by two.*

4.2.3 The seed banking strategy

A quantitative locus determines the rate b_i at which seeds produced *i* generations ago germinates at a given generation *g*. The seed bank length is defined by the fixed parameter *m*, in this section we investigate *m=5* and *m=15*. Although the seeds of all ages can germinate as $\sum_{i=0}^{m} b_i = 1,$, seeds die in the bank with probability *d* each generation. A fraction b_0 of newly produced seeds germinates the following generation *g* while a fraction $1\text{-}b_0$ enter the bank – the bank being empty for $b_0 = 1$.

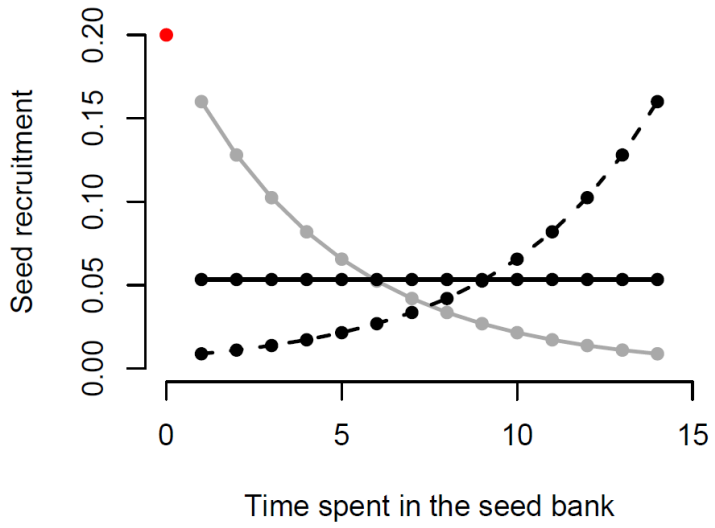


Figure 4.2*: Age-specific recruitment patterns. The recruitment of a dormant seed is constant over time (solid line, black), increasing with age (dotted line, black) or decreasing with age (solid line, grey). The red dot indicates the germination fraction* $b_{i=0}=0.2$.

The probability of a dormant seed to be recruited in the above ground population depends on the age of the seed. We investigate three shapes of the age-specific seed recruitment. Firstly the classic assumption, assuming that probability of a seed to germinate does not depend on its age $b_{i\neq 0} = b_0(1-b_0)^{(i-1)}$, so that the recruitment of the seed in the above ground population

geometrically decreases with age (**Fig. 4.2**, grey line). The second shape corresponds to a constant recruitment with age $b_{i\neq 0}=(1-b_0)/m$ (**Fig. 4.2**, solid line) and the third shape to an increasing recruitment with age $b_{i\neq 0}=b_0(1-b_0)^{(m-i)}$ (**Fig. 4.2**, dashed line).

4.2.4 Evolution of seed banking strategy: multiple mutant competition

We perform simulations of competition between multiple mutants, and adapt the method used in the previous chapter to finite population size.

After a burn in phase of 20,000 generations, a number *N* of mutants with different strategies (different values of b_0*) are introduced in a resident population (with initial value b_0). We use here *N*=5. Each mutant has a different seed banking strategy, b_0*, which is sampled in a Normal distribution with mean the resident value b_0 and a small standard deviation σ=0.05. The mutants are introduced with equal numbers assuming *N*=1. The resident and the mutants compete during a fixed number of generation *T*=1,000, and we then compute the geometric mean fitness over *T* for each strategy. The strategy with the smallest fitness amongst the *N+1* genotypes present *(N* b_0* mutants and the b_0 resident) is removed from the population, and a new mutant (with an effective of 1/*N=0.01*) is introduced. We make the assumption that hosts with the highest fitness have higher chances to produce mutants, and that the mutational step is small. Thus the new mutant strategy b_0* to be introduced is sampled in a Normal distribution with a mean equal to the population average strategy $\bar{b_0}$ and a small standard deviation σ=0.05. The average strategy $\bar{b_0}$ is defined as the sum of the remaining strategies (*N* values of b_0) times their geometric mean of frequency in the population.

Two cases are investigated, (i) independence and (ii) non-independence of germination and resistance loci (*e.g.* linkage equilibrium or disequilibrium). In other words, either resistant

and susceptible types have the same germination strategy which is then the strategy of the population ($b_0=b_{r0}=b_{R0}$), or these alleles can evolve each their own strategy ($b_{r0}\neq b_{R0}$). The method described above corresponds to the first case (linkage equilibrium). In the case of linked loci, we amend the above simulation protocol by removing and then adding at a given time point one resistant and one susceptible mutant. The mutant strategies are sampled around the average strategy of each type $\bar{b}_{R0}$ and $\bar{b}_{r0}$, respectively.

In these preliminary results we investigate the evolution of seed banking under coevolution for only one parameter combination: high costs c_H and c_P equal to 0.2 and a medium disease severity s equal to 0.3. For each set of parameters, 50 repetitions are performed, and record over $2\text{x}10^6$ generations the population mean strategy $\bar{b}_0$, or the resistant and susceptible mean strategies $\bar{b}_{R0}$ and $\bar{b}_{r0}$ to account for the variability of the mutation sampling procedure.

4.3 Results

4.3.1 Dynamic of the system

Without a seed bank

The dynamics of a non-seed banking host population size $b_0 = 1$ and the parasite frequency over time are shown in **Figure 4.3**. We observe that both size and frequency reaches stable oscillations but not an equilibrium size. For increasing intrinsic growth rates δ, higher host population sizes are reached (**Fig. 4.3, C**). We chose to fix δ to the value 0.5 in the remaining of the chapter.

The dynamics of the system is dependent on the coevolutionary costs c_P, c_H and s. For costs $c_H = c_P = 0.2$, if $s < c_H, c_P$ both susceptible and non-infective alleles fix in the population. However when $s > 0.4$, the strength of infection is so strong that it drives the host population to extinction.

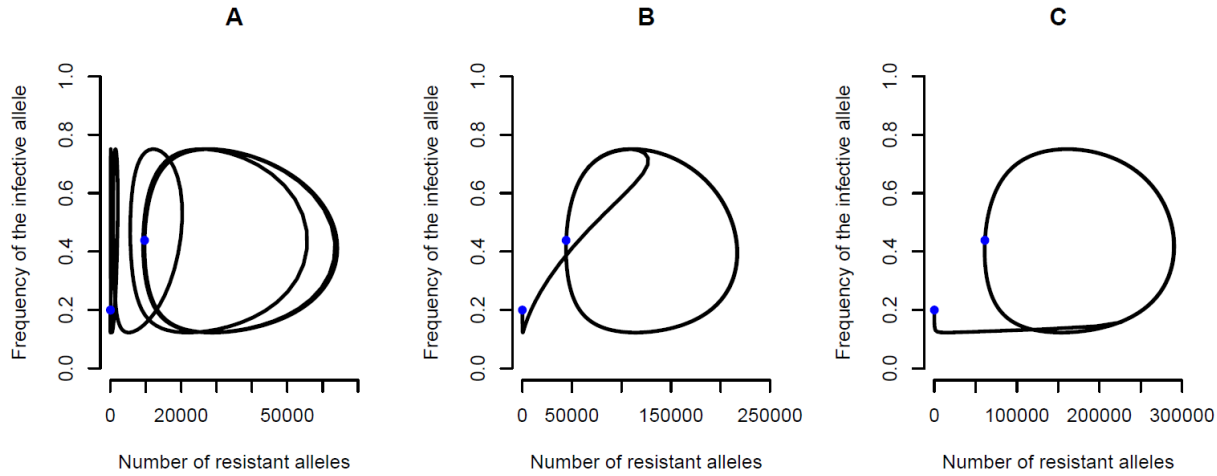


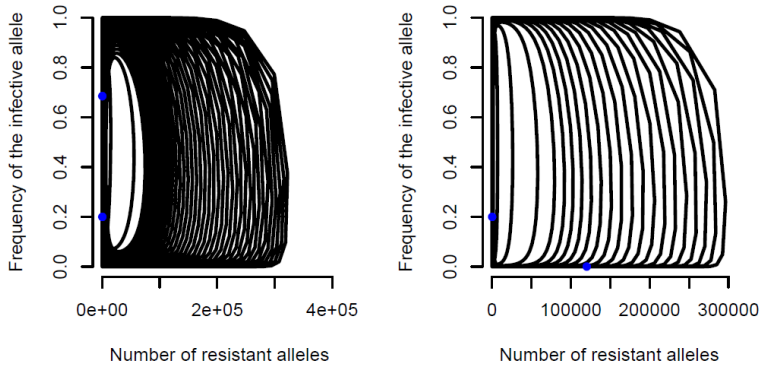
Figure 4.3*: Frequency of the infective allele against the number of resistant alleles under density dependent regulation of the host population (GFG model) for 10.000 generations. The host population does not have a seed bank $b_0 = 1$. The limit capacity is $K = 1{,}000{,}000$ and $\delta = 0.5$ (A), 1(B) and 2(C). The coevolutionary values are $c_p = c_h = 0.2$ and $s = 0.3$. The initial values are $R_0 = 10$ and $a_0 = 0.2$ are indicated with a blue dot. The second blue dots indicates the number of resistant host and the frequencies of the infective parasite after 10,000 generations.*

With a seed bank

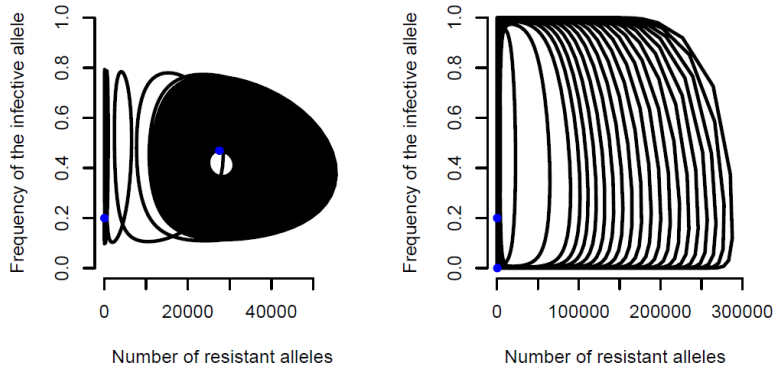
Introducing a seed bank “disturbs” the oscillations and the dynamic outcome is not straightforward (**Fig. 4.4-5**). As expected from the results of chapter 1 and 2, the germination fraction b_0, the seed bank persistence m and the considered age-specific seed recruitment all influence the coevolutionary dynamics.

Under a decreasing seed recruitment with age, we first note that the host population always show increasing oscillations of its size, whereas stable equilibrium sizes can be reached under constant and increasing seed recruitment. Decreasing the germination fraction b_0 leads to slower oscillations in time. Indeed higher fractions of newly produced seeds are entering the bank and thus germinate over multiple generation which in return slows down the growth of the population.

Decreasing recruitment with age



Constant recruitment with age



Increasing recruitment with age

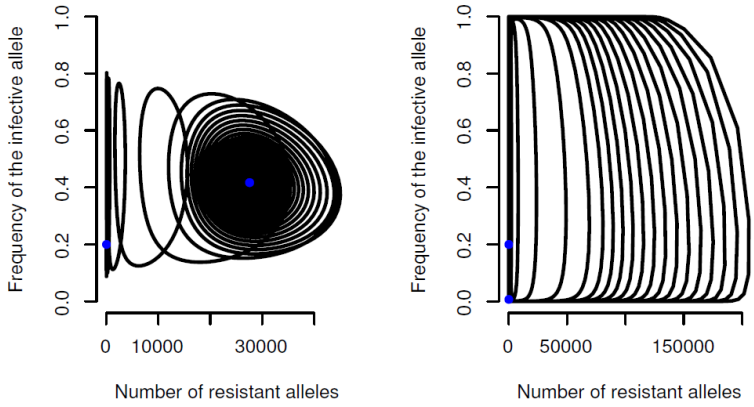
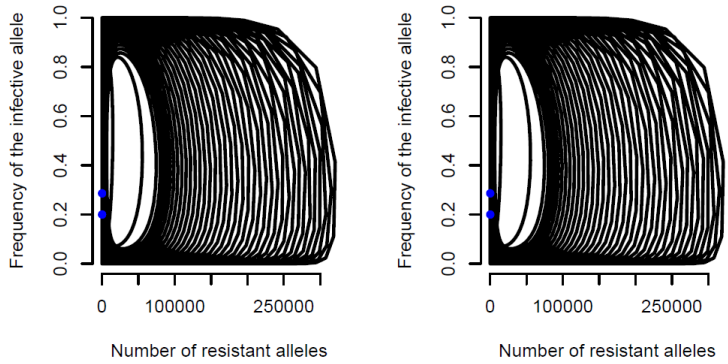
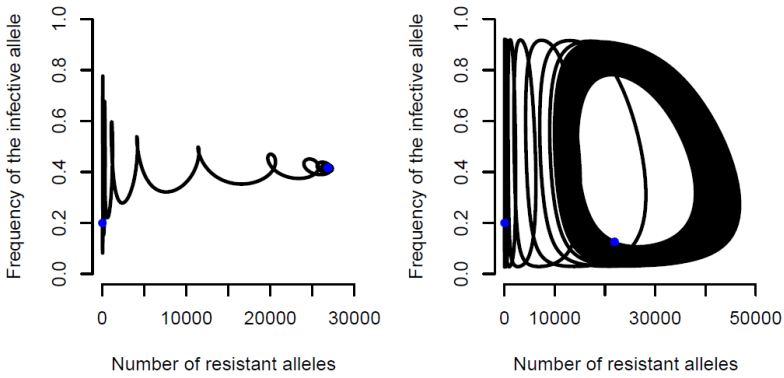


***Figure 4.4**: Frequency of the infective allele against the number of resistant alleles under density dependent regulation of the host population (GFG model) for 10,000 generations. The host population has a short term seed bank m =5, and b_0 =0.8 (left), 0.5 (middle), 0.3 (right). The limit capacity is K =1,000,000 and δ=0.5. The coevolutionary values are $c_p = c_h$ =0.2 and s =0.3. The initial values are R_0 =10 and a_0 =0.2 are indicated with a blue dot. The second blue dots indicates the number of resistant host and the frequencies of the infective parasite after 10,000 generations.*

Decreasing recruitment with age



Constant recruitment with age



Increasing recruitment with age

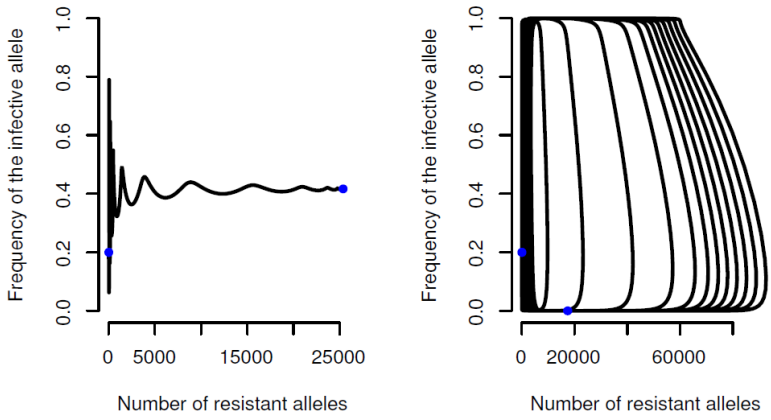


***Figure 4.5**: Frequency of the infective allele against the number of resistant alleles under density dependent regulation of the host population (GFG model) for 10,000 generations. The host population has a short term seed bank m =15, and b_0 =0.8 (left), 0.5 (middle), 0.3 (right). The limit capacity is K =1,000,000 and δ=0.5. The coevolutionary values are $c_p = c_h$ =0.2 and s =0.3. The initial values are R_0 =10 and a_0 =0.2 are indicated with a blue dot. The second blue dots indicates the number of resistant host and the frequencies of the infective parasite after 5,000 generations.*

4.3.2 Multiple mutant competition

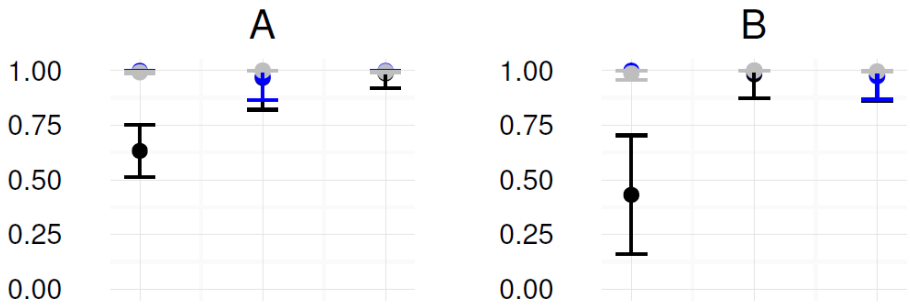


***Figure 4.6**: GFG model with density dependency regulation. Population mean germination fraction (grey), resistant mean germination fraction (black) and susceptible mean germination fraction (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations). The limit capacity is K =1,000,000 and δ=0.5. The GFG model has costs of resistance and infectivity fixed to $c_H = c_P = 0.2$ and $s = 0.3$. The recruitment of seeds is decreasing (left) constant (middle) or increasing with age (right) for seed bank persistence m fixed to 5 years (A) and 15 years (B). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{0R} = b_{0r} = 0.5$. The initial values are R_0 =10 and a_0 =0.2.*

Considering unlinked loci of coevolution and seed banking, we systematically observe the loss of the seed bank, as b_0 evolves toward the value 1. This loss is observed only for the susceptible host type when considering unlinked loci. The results concerning the resistant host are contrasted depending on the age structure assumed in the seed bank (**Fig. 4.6**).

When the recruitment of seeds decreases with the age of the seed, we observe the evolution of the germination fraction towards a mean value of b_{R0}=0.63 for a short term seed bank (m=5) and of b_{R0}=0.43 for a long term seed bank (m=15). When the seed bank assumes an age structure, with a constant or increasing recruitment of seeds with age, we observe the evolution of the germination fraction towards very high values between 0.97 and 0.99, however strictly below the value 1 (*i.e.* seed bank loss). The reason is that in such contexts the interaction between density dependency and the age structure can lead to stable population sizes. The resistant host, undergoing severe fluctuations inherent to the GFG interaction, evolves towards

germination fraction maximising its geometric fitness, which then correspond to high germination fractions up to the value 1. The evolution of the germination fraction in the susceptible does however feedback on the resistant strategy, as once the susceptible host seed bank is lost, the dynamics returns to an unstable state, this lead to a minimum investment in the seed bank (b_0=0.99) strictly above the value 1.

4.3.3 Density dependency and environmental stochasticity

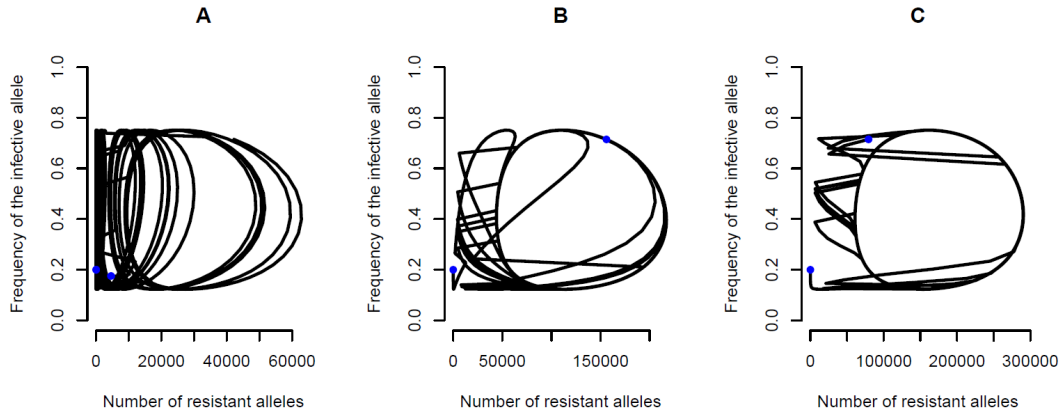


***Figure 4.7**: Frequency of the infective allele against the number of resistant alleles with density dependent regulation of the host population and under environmental stochasticity (GFG model) for 10.000 generations. The host population does not have a seed bank b_0 =1. The limit capacity is K =1,000,000 and δ=0.5 (A), 1(B) and 2(C). The coevolutionary values are $c_p = c_h$ =0.2 and s =0.3. The initial values are R_0 =10 and a_0 =0.2, and indicated with a blue dot. The second blue dots indicates the number of resistant host and the frequencies of the infective parasite after 10,000 generations.*

As illustrated above (**Fig. 4.7**), the introduction of environmental stochasticity generates perturbations of high amplitudes of the population size. Consequently, the influence of environmental stochasticity on the evolution of the seed banking strategy is drastic. It is indeed the first context where the population (unlinked loci) and both susceptible and resistant host type evolve a clear seed banking strategy (linked loci) (**Fig. 4.8-9**).

4.3.4 Multiple mutant competition

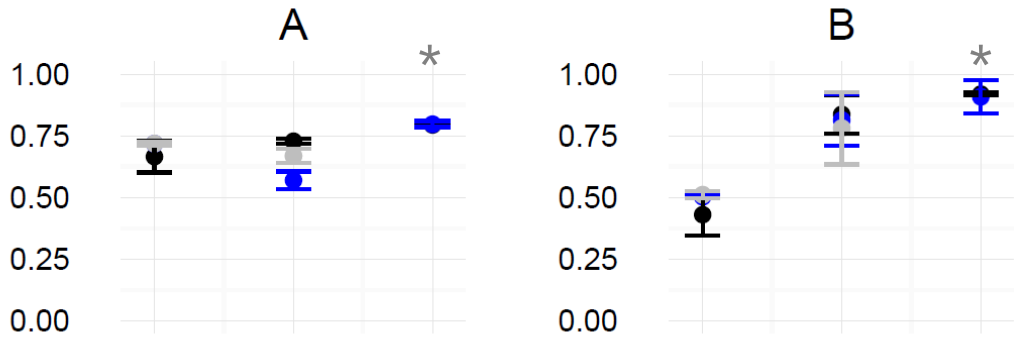


Figure 4.8*: GFG model with density dependency regulation and environmental stochasticity, under unfavourable conditions, the seeds production is reduced by a constant equal to 10. Population mean germination fraction (grey), resistant mean germination fraction b_{R0} (black) and susceptible mean germination fraction b_{r0} (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations). The limit capacity is K =1,000,000 and δ=0.5. The GFG model has costs of resistance and infectivity fixed to $c_H = c_P = 0.2$ and $s = 0.3$. The recruitment of seeds is independent (left) constant (middle) or increasing with age (right) for seed bank persistence m fixed to 5 years (A) and 15 years (B). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{R0} = b_{r0} = 0.5$. The initial values are R_0 =10 and a_0 =0.2.* Simulations missing assuming unliked loci.*

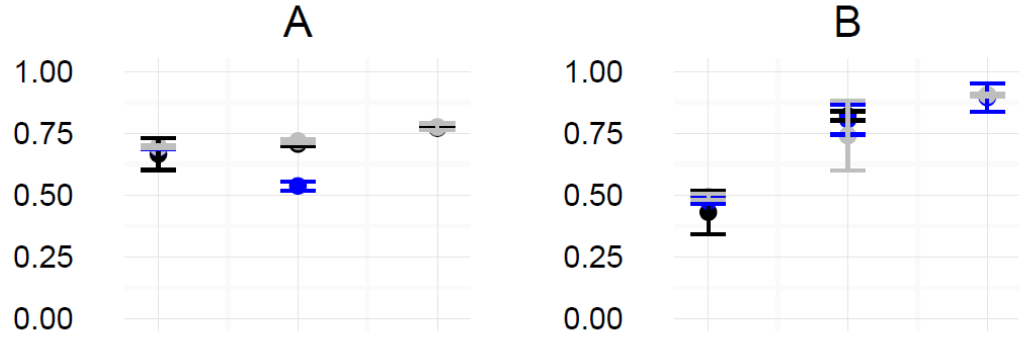


Figure 4.9*: GFG model with density dependency regulation and environmental stochasticity, under unfavourable conditions, the seeds production is reduced by random constant. Population mean germination fraction (grey), resistant mean germination fraction b_{R0} (black) and susceptible mean germination fraction b_{r0} (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations). The limit capacity is K =1,000,000 and δ=0.5. The GFG model has costs of resistance and infectivity fixed to $c_H = c_P = 0.2$ and $s = 0.3$. The recruitment of seeds is independent (left) constant (middle) or increasing with age (right) for seed bank persistence m fixed to 5 years (A) and 15 years (B). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{R0} = b_{r0} = 0.5$. The initial values are R_0 =10 and a_0 =0.2.*

The germination fractions evolving for susceptible and resistant belong to the same range of parameters, except in one specific context: short term seed bank with a constant recruitment of seeds with age, under which we observe stronger investment in the seed bank for the susceptible host than for the resistant host. It is in this context too, that the population evolves towards the optimum strategy of the resistant host when assuming unliked loci.

The interaction of the seed bank age-structure with density-regulation buffers against the effect of environmental stochasticity, as we observe the evolution of the germination fraction towards higher germinations fractions for both constant and increasing recruitment with age. Finally a fixed long term seed persistence strongly reduces the investment in the seed bank. The two types of environmental stochasticity assumed do not affect the results, a random effect on seed production seems to only reduce the variability between the repetitions.

4.3.5 Matching allele

We modify the model to include a Matching Allele recognition matrix (see supplement for equations) in which both hosts are infected by their specific parasite and have equivalent fitness fluctuations. The dynamics of the MA model without a seed bank reaches stable oscillations of the population size and these oscillations may reach a stable equilibrium size when the seed bank exhibits a constant or increasing age-specific seed recruitment.

Since hosts show equivalent fitness fluctuations, both genotypes systematically evolve the same seed banking strategy, the small variation observed due to the sampling of mutants is in fact negligible (**Fig. 4.10-11**). When seed recruitment decreases with age, hosts evolve a very high mean germination fraction, $b0_{H1}=b0_{H2}=0.99$ for short term persistent banks (**Fig. 4.9**). The

fraction decreases towards the value 0.82 for long term persistent banks. Under environmental stochasticity, the fraction evolves towards 0.68 for short term banks and 0.45 for long term banks (**Fig. 4.11**).

When age-structured seed bank is assumed and under density-dependency only the germination fraction systematically evolves towards the value 0.99. Once environmental stochasticity is introduced, we observe that (i) the investment in the seed bank is always stronger in the context of constant seed recruitment, (ii) the investment is more important under short term persistent banks.

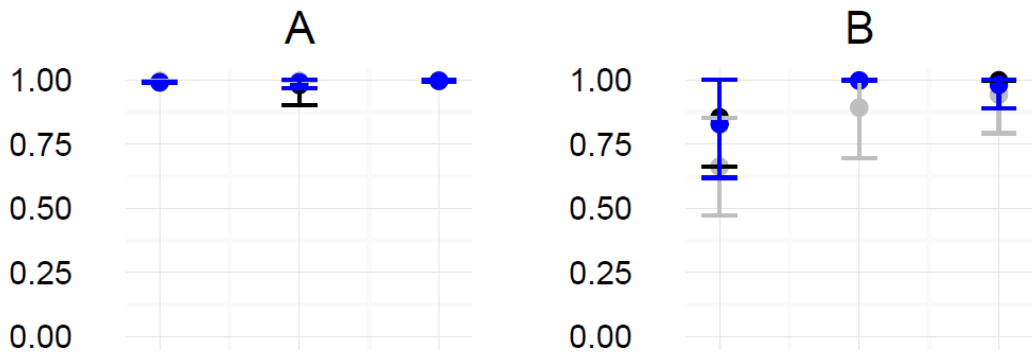


***Figure 4.10**: MA model with density dependency regulation. Population mean germination fraction (grey), resistant mean germination fraction b_{0R} (black) and susceptible mean germination fraction b_{0r} (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations). The limit capacity is K =1,000,000 and δ=0.5. The cost of infectivity is fixed to $s=0.3$. The recruitment of seeds is independent (left) constant (middle) or increasing with age (right) for seed bank persistence m fixed to 5 years (A) and 15 years (B). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{R0}=b_{r0}=0.5$. The initial values are R_0 =10 and a_0 =0.2.*

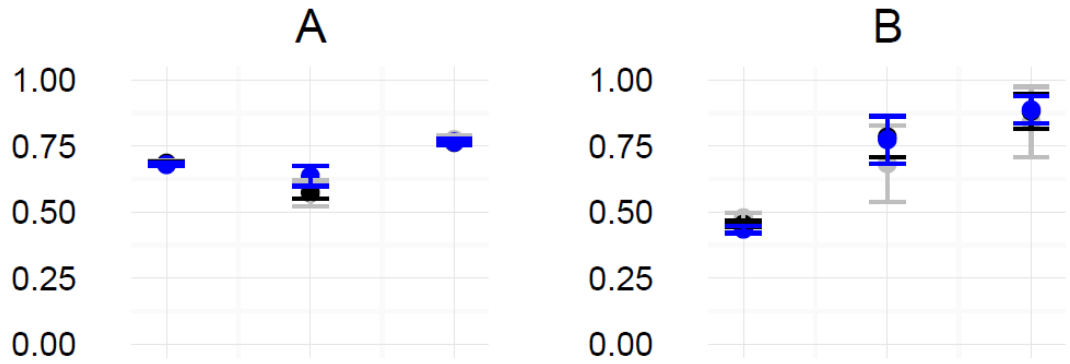


***Figure 4.11**: MA model with density dependency regulation and environmental stochasticity, under unfavourable conditions, the seeds production is reduced by random constant. Population mean germination fraction (grey), resistant mean germination fraction b_{0R} (black) and susceptible mean germination fraction b_{0r} (blue) as a result of the multiple mutant simulation process ($2x10^{16}$ generations). The limit capacity is K =1,000,000 and δ=0.5. The cost of infectivity is fixed to $s=0.3$. The recruitment of seeds is independent (left) constant (middle) or increasing with age (right) for seed bank persistence m fixed to 5 years (A) and 15 years (B). Error bars indicate variation over the 50 repetitions per parameter set. The initial germination fraction is $b_{R0}=b_{r0}=0.5$. The initial values are R_0 =10 and a_0 =0.2.*

4.4 Discussion

The results obtained show the complex interaction between the infection matrix considered (*i.e.* GFG or MA infection), density-dependency, environmental stochasticity and the parameters of the seed bank.

Decreasing seed recruitment with age

The simplest context investigated concerns the assumption of a decreasing seed recruitment with age Rees & Long (1993). For such a seed bank, the coevolutionary dynamics are only driven by negative indirect frequency dependent selection (niFDS), meaning that the population sizes and allele frequencies of the hosts and parasites are always unstable, cycling away from the internal equilibrium of the system. In the GFG model the susceptible host is the most common type, with a higher geometric mean frequency than the resistant type. Secondly the resistant host shows increases of sizes of extremely high amplitudes (*e.g.* going from a hundred to 4.10^5 individuals in the course of a few generations) but these populations outbursts occur for extremely reduced periods, due to the combined effect of niFDS and density-dependency regulation. Density-dependency indeed amplifies the fluctuations which are due to unstable coevolutionary dynamics. In response to such selective pressures, the resistant host type evolves a seed bank (b_{R0}<1), while the seed bank is lost for the susceptible type (b_{r0}=1). Under genetic independence of the coevolutionary and germination loci, selection is driven by the susceptible host and the seed bank is accordingly lost (b_0=1).

Environmental stochasticity equally affects susceptible and resistant hosts by disturbing the coevolutionary dynamics. The loss of reproduction under unfavourable conditions generates a strong decrease of the total host population size, which lowers the geometric mean fitness of

both host types. In response, hosts evolve seed banking as a bet-hedging strategy (independently of the genetic linkage between the coevolutionary and the germination loci). Resistant host evolves almost identical mean germination fraction if environmental stochasticity is included in the model or not. This raises the question whether coevolution and environmental stochasticity have an equivalent influence on the evolution of seed banking. The resistant host evolve a slightly lower mean germination fraction than that of the susceptible host under GFG, which might be explained by the stronger pressures acting on resistant hosts. However the variance observed over the simulations repetitions shows an overlap between the seed banking strategies evolving for both resistant and susceptible hosts. These preliminary results are thus not sufficient to address this question, but investigating different values of the coevolutionary costs and varying the probability for bad conditions to occur might help to understand the relative influence of both selective pressures.

Under a MA model hosts undergo symmetric unstable dynamics. As such both host types strictly evolve the same seed banking strategy, independently of the genetic linkage. Considering density-dependency only, the seed bank is lost for short term persistent bank but is maintained for long term persistent banks, while the introduction of environmental stochasticity selects for seed banking in both contexts. The investment in the seed bank is higher for long term persistent bank, as the temporal window covered by the seed bank length is larger with regard to the period of the coevolutionary cycles. As these cycles are faster for MA, the investment in the bank is found to be higher than under a GFG interaction.

Constant and increasing seed recruitment with age

The introduction of an age-specific seed recruitment, meaning that the probability of a seed to germinate now depends on its age, greatly complexifies the outcome of the simulations.

Indeed age-specific recruitment may generate negative direct frequency dependent selection (ndFDS), leading to stable population host sizes and host and parasite allele frequencies in time. Here we observe that density-dependency influences the parameter space under which a stable state can be reached. For instance concerning constant seed recruitment with age, stability can be reached for both short term and long term persistent seed banks (in the GFG model). When only density regulation is assumed, we systematically observe the evolution of the highest germination fraction value b_0=0.99. This fraction remains strictly below 1, otherwise this would lead to the loss of the seed bank and the re-occurrence of unstable coevolutionary dynamics.

The introduction of environmental stochasticity highlights the complex influence of the seed bank age structure on the evolution of strategies. When bad conditions occur at a generation *g*, the reproduction of plants is strongly decreased. Let us define Y_g the reduced amount of seeds produced, the key element is the timing at which Y_g germinates. If the germination fraction b_0 is high, most of Y_g germinate at the following generation $g+1$, which will have a drastic influence on the population size above-ground as the population is barely maintained by the germination input. Then, depending on the age-specific seed recruitment, the remaining amount $Y_g(1-b_0)$ germinates differentially through the *m* years during which seeds can persist.

We investigated two scenarios where the probability of a seed to germinate is age-dependent (i) either a constant amount germinates during the following *m* generations or (ii) this amount increases each generation. In this latter case (ii), the seed bank structure allows for a better delay in time of the disturbance due to environmental stochasticity since the biggest fraction of $Y_g(1-b_0)$ will germinate at the generation $g+m$. The above ground population is thus maintained by the germination input from older seeds after the occurrence of bad conditions, and consequently the seed bank is almost replenished before the repercussion of environmental stochasticity reaches the above-ground population (that is the negative effect of stochasticity on

population persistence). Longer seed persistence *m* introduces a longer delay, therefore lower investment in the seed bank are found for long term persistent banks versus short term persistent banks.

In the context (i) the repercussion of environmental stochasticity is not delayed but averaged over the *m* years, which is less efficient to buffer against the reproductive loss. Therefore higher investment in the seed bank are found for a constant seed recruitment with age than for an increasing recruitment. We can further note that the investments evolving for short term persistent bank belong to the same range of strategies than when the seed bank does not allow time delay of the consequences of bad conditions (*i.e.* decreasing seed recruitment with age). More surprisingly considering a GFG infection matrix, a unique case occurs where the susceptible host evolves towards a higher investment in the seed bank than the resistant host. The explanation is triple, (i) the fitness asymmetry favours the susceptible host type in the GFG approach, leading to higher stable populations sizes of the susceptible host under density-dependent regulation, (ii) therefore the introduction of environmental stochasticity (which equally affects resistant and susceptible hosts) generates fluctuations of the susceptible host population size of higher amplitudes than for the resistant population size, and as (iii) the age-specific recruitment does not buffer efficiently the reproductive loss, susceptible host evolve towards lower germination fractions, leading to a reduction of both the size of the host population and the amplitude of the fluctuations.

The results obtained in this study are extremely complex, although density-dependency is modelled assuming the classic but simple logistic relation between the growth rate of the host population size. This function maintains the host population at an arbitrary limiting carrying capacity *K*. It is however unlikely that natural population reach such a definite equilibrium size. Several studies proposed different shape of density-dependency functions, for instance a

sigmoidal shape, allowing to simulate sizes fluctuations over time more accurately. Such functions can exhibit cyclic and chaotic behaviour which could potentially select for the evolution of seed banking strategies in the susceptible host type as does environmental stochasticity in our study.

Supplementary informations

Equations for the matching allele model:

In an MA approach, hosts and parasites do not carry costly resistance and infectivity traits, but parasites genotype must match the most common host genotype to infect them successfully (*e.g.* Gandon et al. 1996). We have two types of host H1 and H2 and two types of parasite P1 and P2; P1 matches the genotype of H2 and P2 the genotype of H1. The rest of the model assumptions are common to the GFG model described in the methods (***Density-dependency)***. The following difference equations describe changes in allele frequencies including a seed bank for the host.

The following recurrence equations describe the host population:

$$H_{1,g+1}=H_{1,g}\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_{H1},g)} b_i(1-d)^i H_{1,g-i}+\sum_{i=0}^{min(m_{H2},g)} b_i(1-d)^i H_{2,g-i}}{K}\right]\right)(1-sP_{2,g})$$

$$H_{2,g+1}=H_{2,g}\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_{H2},g)} b_i(1-d)^i H_{2,g-i}+\sum_{i=0}^{min(m_{H1},g)} b_i(1-d)^i H_{1,g-i}}{K}\right]\right)(1-sP_{1,g}) \quad \text{(Equations 4.2)}$$

To simplify the equations we can introduce the following terms DH_1 and DH_2, describing the regulated host population prior infection for host 1 and host 2,

$$DH_1=H_{1,g}\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_{H1},g)} b_i(1-d)^i H_{1,g-i}+\sum_{i=0}^{min(m_{H2},g)} b_i(1-d)^i H_{2,g-i}}{K}\right]\right)$$

$$DH_2=H_{2,g}\left(1+\delta\left[1-\frac{\sum_{i=0}^{min(m_{H2},g)} b_i(1-d)^i H_{2,g-i}+\sum_{i=0}^{min(m_{H1},g)} b_i(1-d)^i H_{1,g-i}}{K}\right]\right)$$

the parasite population is simply described with:

$$P_{1,g+1}=P_{1,g}\left(\frac{DH_2}{DH_2+DH_1}\right)/\overline{W_P}$$

$$P_{2,g+1}=P_{2,g}\left(\frac{DH_1}{DH_1+DH_2}\right)/\overline{W_P}$$

General discussion

5.1 Summary of the results and further questions

The global results of my thesis support the hypothesis that host seed banking can evolve as a bet-hedging strategy in response to host-parasite coevolutionary dynamics. Throughout this work, I tested how the genetic interaction assumed between hosts and parasites, namely the GFG and MA approaches, influences the evolution of the germination fraction b_0 for seed banks assuming (i) a fixed seed persistence m in the bank and (ii) an age-specific seed recruitment in the above ground population (either decreasing, constant, or increasing with the age of a seed). I additionally tested for (i) linkage equilibrium and (ii) linkage disequilibrium of the GFG and MA loci with the germination loci. Lastly I assumed two other sources of variation, density-dependent regulation of the host population and environmental stochasticity, which influence was contrasted to my results obtained under coevolution only. An overview of the results is given in **Tables 5.1** and **5.2**, for GFG and MA respectively. The tables show the population mean strategy $\bar{b}_0$ and the resistant and susceptible mean strategies $\bar{b}_{R0}$ and $\bar{b}_{r0}$ evolving in the host population as a result of the multiple mutant simulation process for each context investigated.

Under coevolution, the type of interaction considered (GFG or MA model) and the fixed age-specific seed recruitment are key elements driving the evolution of the germination fraction (**table 5.1-2**, first column). Under a GFG interaction, the two host types always show a distinct optimal strategy, whereas under a MA interaction, the two host types have the same optimum. This optimal strategy may not be reached under a GFG interaction, as the evolution of the resistant strategy is driven towards the optimal of the susceptible host when assuming linkage equilibrium and sometimes constrained to a sub-optimal strategy under linkage disequilibrium. Whereas under MA, the linkage assumed does not influence seed banking. Therefore under MA, coevolution generally selects for stronger investments in the seed bank.

Now the age-specific seed recruitment considered may generate negative direct frequency dependent selection (ndFDS) leading to stable polymorphism. Stability does not occur if the recruitment of seeds decreases with age, but stable states can be reached when seed recruitment is constant or increasing with age. Therefore, bet-hedging occurs in two distinct ways.

Firstly, when coevolutionary cycles are always unstable, storing high amounts of seeds in the bank is advantageous only if the persistence of the bank is large enough in comparison to the frequency of changes, revealing the major influence of the speed of the coevolutionary cycles. Secondly, at the equilibrium state, the geometric mean fitness of the host is maximised, thus the evolution of the seed banking strategy is driven towards values of the germination fraction leading to stable polymorphism. These values are generally high, corresponding to a small investment in the seed bank. However if the seed bank persistence is too short and does not generates ndFDS, then high investments in the bank will evolve. To summarise, the amplitude of the coevolutionary cycles is either decreased through a higher investment in the bank with increasing persistence m, or via the strict opposite through lower investment with increasing persistence m.

In this work I considered the evolution of the germination fraction only, but theoretically both the seed persistence and the age-specific seed recruitment could evolve. With that in mind, we can see the dichotomy described above from a different point of view. Indeed bet-hedging occur either via the evolution towards longer seed bank persistence combined with high storage of seeds, or, via the evolution of a different age-specific recruitment with a reduced storage of seeds. This brings forward questions about 1) the evolvability of both traits and 2) whether and which one of the two is more likely to evolve?

The evolution of a different age-specific recruitment, for instance from a decreasing relation with age (few old seeds recruited) to a constant relation with age (homogeneous number of seeds of all ages) is theoretically more likely than the evolution of longer seed persistence, as the geometric mean fitness of hosts is maximised when the coevolutionary dynamics are stable. Stability is generally reached independently of the seed persistence in the MA model, suggesting the evolution of the age-specific recruitment. Yet in the GFG model stability only occurs for long term persistent banks, thus under coevolution, the joint evolution of the two traits is expected. This discrepancy between MA and GFG vanishes once multiple sources of variations are considered in the model (density-dependent regulation of the host population and environmental stochasticity). Indeed, an increasing seed recruitment with age better buffer against drastic unpredictable fluctuations and would theoretically evolve rather than longer seed bank persistence.

Gene for gene infection

Age-specific recruitment	m	Linkage	Coevolution		Coevolution Density-dependency		Coevolution Density-dependency Stochasticity	
			$\bar{b}_{R0}$	$\bar{b}_{r0}$	$\bar{b}_{R0}$	$\bar{b}_{r0}$	$\bar{b}_{R0}$	$\bar{b}_{r0}$
Decreasing	5	L	**0.72** [0.62,0.82]	**1** [-]	**0.63** [0.51, 0.75]	**1** [0.99,1]	**0.66** [0.60,0.72]	**0.69** [0.68,0.69]
		U	**1** [-]		**0.99** [0.98,0.99]		**0.69** [0.68,0.69]	
	15	L	**0.53** [0.36,0.70]	**1** [-]	**0.43** [0.16,0.70]	**0.98** [0.87,1]	**0.43** [0.34,0.51]	**0.47** [0.46,0.48]
		U	**1** [-]		**0.96** [0.96,1]		**0.49** [0.47,0.50]	
Constant	5	L	**0.75** [0.65,0.85]	**0.97** [0.96,0.98]	**0.97** [0.82,1]	**0.97** [0.87,1]	**0.70** [0.69,0.71]	**0.54** [0.52,0.55]
		U	**0.94** [0.93,0.96]		**0.99** [–]		**0.67** [0.63,0.70]	
	15	L	**0.94** [-]	**1** [–]	**0.99** [0.99,1]	**0.99** [0.99, 1]	**0.82** [0.80, 0.83]	**0.80** [0.74,0.86]
		U	**0.99** [–]		**1** [–]		**0.74** [0.59,0.88]	
Increasing	5	L	**0.88** [0.87,0.89]	**0.97** [–]	**0.99** [0.91,1]	**1** [–]	**0.77** [0.76.0.78]	**0.77** [0.76,0.78]
		U	**0.93** [0.91,0.95]		**0.99** [–]		**0.77** [0.76.0.78]	
	15	L	**0.99** [–]	**1** [–]	**0.98** [0.96,1]	**1** [–]	**0.90** [0.90, 0.91]	**0.89** [0.84,0.95]
		U	**0.99** [–]		**0.99** [–]		**0.90** [0.89, 0.91]	

Table 5.1*: Overview of the results assuming a GFG interaction. The resistant and susceptible mean strategies* $\bar{b}_{R0}$ *and* $\bar{b}_{r0}$ *(Linkage, L) and the population mean strategy* $\bar{b}_0$ *(Linkage, U) evolving in hosts, as a result of the multiple mutant simulation process, are given for each context tested (age-specific recruitment X seed bank persistence m X source of variation). The results highlighted in blue correspond to the chapter 2, in grey to the chapter 3, and blank to the chapter 4.*

Matching allele

Age-specific recruitment	m	Linkage	Coevolution		Coevolution Density-dependency		Coevolution Density-dependency Stochasticity	
			$\bar{b}_{R0}$	$\bar{b}_{r0}$	$\bar{b}_{R0}$	$\bar{b}_{r0}$	$\bar{b}_{R0}$	$\bar{b}_{r0}$
Decreasing	5	L	**0.69** [0.68,0.70]	**0.69** [0.68,0.70]	**0.99** [0.98,0.99]	**0.99** [0.98,0.99]	**0.68** [0.67,0.69]	**0.68** [0.67,0.69]
		U	**0.69** [0.68,0.70]		**0.99** [–]		**0.68** [–]	
	15	L	**0.40** [0.40,0,41]	**0.40** [0.40,0.41]	**0.82** [0.61,1]	**0.82** [0.61,1]	**0.45** [0.44,0.47]	**0.43** [0.42,0.44]
		U	**0.40** [0.40,0,41]		**0.66** [0.47,0.85]		**0.48** [0.46,0.49]	
Constant	5	L	**0.79** [0.78,0.79]	**0.79** [0.78,0.79]	**0.98** [0.90, 1]	**0.99** [0.97,1]	**0.57** [0.55,0.6]	**0.63** [0.59,0.67]
		U	**0.79** [0.78, 0.79]		**0.99** [–]		**0.57** [0.52,0.61]	
	15	L	**0.87** [0.86,0.87]	**0.87** [0.86,0.87]	**0.99** [–]	**0.99** [–]	**0.78** [0.70, 0.86]	**0.77** [0.68, 0.86]
		U	**0.87** [–]		**0.89** [0.69,1]		**0.68** [0.53 0.82]	
Increasing	5	L	**0.85** [–]	**0.85** [–]	**0.99** [–]	**0.99** [–]	**0.76** [0.75, 0.77]	**0.76** [0.75, 0.77]
		U	**0.85** [–]		**0.99** [–]		**0.76** [0.75, 0.77]	
	15	L	**0.66** [0.33,0.98]	**0.69** [0.36,1]	**0.99** [–]	**0.99** [–]	**0.88** [0.81,0.95]	**0.89** [0.84, 0.94]
		U	**0.67** [0.59,0.75]		**0.94** [0.79,1]		**0.88** [0.81,0.94]	

***Table 5.2**: Overview of the results assuming a MA interaction. The resistant and susceptible mean strategies $\bar{b}_{R0}$ and $\bar{b}_{r0}$ (Linkage, L) and the population mean strategy $\bar{b}_0$ (Linkage, U) evolving in hosts, as a result of the multiple mutant simulation process, are given for each context tested (age-specific recruitment X seed bank persistence m X source of variation). The results highlighted in blue correspond to the chapter 2, in grey to the chapter 3, and blank to the chapter 4.*

5.2 Future perspectives

Epidemiology

In the host-parasite model of coevolution derived throughout the thesis, the most complex host life cycle assumes density-dependent survival of germinating seeds (at a given generation *g)*, and once the established seedlings grow into adult plants, all individuals in the population receive parasites and can become infected depending on the outcome of the genetic interaction. Hereby we ignore potential feedbacks between the varying size of the host population and the success of infection of the parasite. However epidemiological studies identified that disease transmission, which has for main parameter the transmission coefficient β, can be sensitive to changes in the host population density.

The importance of such feedbacks is crucial when investigating directly transmitted pathogens, meaning that pathogens are transmitted via the contact between infected and non-infected hosts. The number of potentially infectious contacts made per infected host and per unit of time defines the per capita contact rate. This rate may be independent or dependent on the density of infected hosts, and the choice of the relation must be chosen with care (Borremans *et al.* 2017).

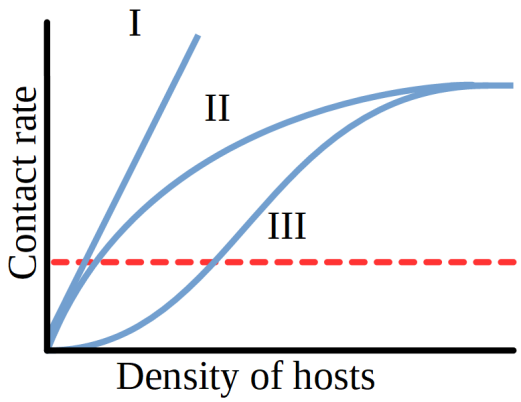


Figure 5.1*: Contact density function.*

The models with transmission being independent of the density of infected host, contexts concern mostly sexually transmitted pathogens, and the transmission is assumed of the following form: $\beta SI/N$, where *S* and *I* respectively correspond to the density of non infected and infected hosts, and *N* the total size of the population. If hosts are randomly mixed, *S* hosts make on average the same number of contacts with infected hosts, regardless of their density as shown in the figure below (**Fig. 5.1**, doted red line).

When disease transmission depends on the density of hosts, the transmission is of the form βSI, the number of random contacts of *S* and *I* hosts being proportional to the density of infected hosts *I*. Density-dependent disease transmission also depends on the shape of the contact density function. Transmission is often assumed directly proportional to density (**Fig. 5.1**, blue line I), a shape analogous to Holling type I functional response – defining the relation between the rate of prey consumption by a predator and the density of preys in the context of predator-prey models.

Transmission can also be directly proportional to the density for low densities, but reach a maximum rate of contact for high densities (**Fig. 5.1**, blue line II). A relation which is then analogous to Holling type II functional response (McCallum *et al.* 2001). Some studies advance that the neighbourhood of non-infected host has more influence on the disease transmission than the total density of the hosts population, explaining the occurrence of patchy infection in space. This means that an infected host will be more likely to be surrounded with other infected hosts, while non-infected hosts are more likely to be surrounded with other non-infected hosts. In this context, the transmission rate is low and almost constant at low density of infected hosts *I*, then increases for increasing density of *I*, until reaching a plateau of maximum contact. This relation is then analogous to a Holling type III functional response (**Fig. 5.1**, blue line III).

As mentioned earlier, models describing frequent-dependent and density-dependent disease transmission are restricted to the context of directly transmitted pathogens. This appears limiting to the purpose of extending our general model of any system of host-parasite coevolution. Indeed directly transmitted pathogens represent only a part of plant-parasite interactions, such as few virus and bacteria pathogens, for example tobacco mosaic virus and *Ralstonia solanacearum* bacteria (responsible for the potato brown rot disease). However many pathogens are vector-borne or disseminated by wind and rain, and the life histories of the parasite is overlooked in epidemiological models, as models are compartmentalized between classes of hosts: Susceptible (healthy) and eventually Exposed (latently infected), Infected and Recovered (*i.e.* SI-SIS-SIR-SEIR models).

Rabajante *et al*. (2016) introduced the concept of functional responses in host-parasite interactions, defined by the rate of infection and host utilization by parasites. This is once again analogous to functional responses in predator-prey models, but in a stricter sense. The three functional responses proposed by Rabajante *et al.* are defined as follow (**Fig. 5.2**):

Type I – Linear parasitic utilization of hosts.

Type II – Parasitic utilization of hosts following a hyperbolic curve, with linear utilization at low densities but parasites satiation at high densities.

Type III – Parasitic utilization of hosts following a sigmoidal curve, with a low rate of utilization at low densities, then linear at intermediate densities until parasites satiation at high densities.

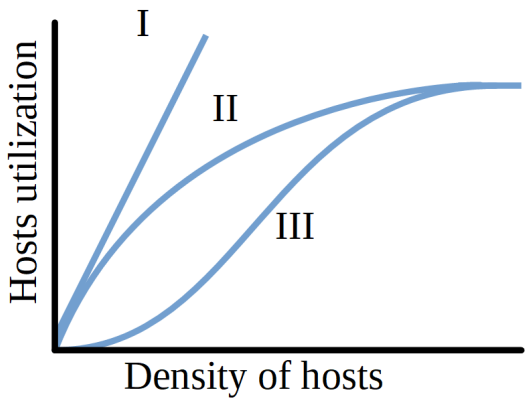


***Figure 5.2**: Types of functional responses in host-parasite interactions.*

The model developed by Rabajante *et al.* shows two very interesting features. Firstly it introduces the parasitism efficiency matrix defining the relation between host and parasite, for instance that host *i* is the main host of parasite *j*. This allow studies of gene-for-gene and matching-allele interactions and all intermediate interactions in-between (Agrawal & Lively 2002). Secondly it is a resource-consummer model, following the density of both hosts and parasites populations over time. As it is very common to observe temporal variation of the presence of pathogens in relation to variations in the abiotic environment (Jarosz & Davelos 1995), this type of model could investigate disease seasonality as well or even the potential for temporal bet-hedging strategies to evolve in parasites.

Integrating an evolving seed banking strategy, with its two key elements (i) the age-dependent probability to germinate and (ii) the limited seed persistence in time, remains an issue considering that models discussed above assume continuous time. In this regard, it is worth exploring discrete time models of host-parasitoid interaction, such as the classic model of Nicholson & Bailey (1935) and the extensions of this model including diapause in either one or both hosts and parasitoids (Ringel *et al.* 1998; Corley & Capurro 2000; Corley *et al.* 2004). The functional responses involved in host-parasitoid models, linking the success of parasitoid attacks

with the density of hosts are of special interest, as they depend on the searching efficiency of parasitoids and the handling time of hosts. The searching efficiency varies according to the probability of encountering a suitable host and the handling time is affected by super-parasitism (Godfray 1994), which has parallel to the context of fungi spores disseminated by wind that might land on already infected leaves/plants.

An other lead to explore are models combining continuous and discrete dynamics (*e.g.* Hamelin *et al.* 2011) in an epidemiology framework. Epidemiological models implicitly assume that parasites evolve much faster than hosts. Thus under this framework host and parasite life cycles can be implemented discretely between years, while the interaction resulting in host infection can be implemented assuming continuous time within years. Seed banking in both host and parasites can be considered, together with polycyclic epidemics that are common to crop pathogens and fungal or bacteria plant diseases. Additionally, parasites can drive the host population to extinction, which would be prevented by the host seed bank. Such model coud test for the evolution of seed banking as an adaptation to extremely virulent parasites.

Testing the hypothesis of bet-hedging in natural systems

Understanding the role and the composition of soil seed banks is of particular importance in natural systems as seed banks facilitate recovery of indigenous vegetation after invasions and habitat deterioration. The density of viable seeds in the soil seed bank and its composition in terms of species can be recovered from soil samples from various landscape, for example in arid and semi-arid ecosystems (Caballero *et al.* 2003; Fourie 2008), or managed grasslands (Suter & Lüscher 2012). These studies showed that the soil seed bank is mostly composed of annual species and can be extremely large with thousands of seeds per m^2. The emergence of collected

seeds can be followed over time in laboratory conditions to assess there viability and test there susceptibility to various light, temperature or also stratification treatments. However as soil samples are collected at one point in time only, they do not represent appropriately all the characteristics of the seed bank, and notably the persistence of the bank. Conclusions regarding the age structure and emergence patterns of a seed bank can be drawn only from soil samples collected repetitively over in time at the same location.

Few methods exist to estimate the age of the seeds composing soil seed banks, shortly reviewed in (Saatkamp *et al.* 2009) and most consist in monitoring for several years the emergence of seeds from experimental seed banks in the field as in (Kalisz 1991). However Moriuchi *et al.* (2000) proposed a method using tandem accelerator mass spectrometry (TAMS). Applying this method on samples from the natural population of *Pectocarya recurvata*, they found that the seed number declined exponentially with age, with a mean of two years. Based on the knowledge that *Pectocarya recurvata* has a high germination fraction they concluded that the age-structure was adequate to buffer the population from typical fluctuations encounter in this area.

Knowing the difficulties of estimating the characteristics of a soil seed bank it appears ambitious to target a specific plant-pathogen interaction to test the hypothesis of the host-parasite general model. Yet, there is one promising system, the wild plant *Plantago lanceolata* and its specialist powdery mildew pathogen *Podosphaera plantaginis*, which are intensively studied in the Åland islands of sowthwestern Finland. Indeed *P. lanceolata* and *P. plantaginis* interaction is characteristic of a gene-for-gene (Thompson & Burdon 1992; Laine 2007). There is a large amount of variation in resistance and infectivity between host and parasite populations (Laine 2004) and rapid ongoing coevolution between these two species has been shown, host resistance evolving in response to pathogen pressure at both spatial and temporal scales (Laine 2005, 2006;

Laine & Hanski 2006). Secondly *P. lanceolata* reproduces clonaly and form long term persistent banks (Bos 1992), admitted to explain the relative stability of metapopulations in time (Tack & Laine 2014) thus suggesting bet-hedging. Only one study investigated the age-specific seed recruitment of *P. lanceolata* and deduced a decreasing recruitment with age over the course of 5 years (Rees & Long 1993). Therefore field studies of *P. lanceolata* and *P. plantaginis* interaction could test for the hypothesis of seed banking evolving in response to coevolution and the results of this work suggest that 1) variation of the germination fraction of *P. lanceolata* should account for the temporal variation of the pathogens pressure, and 2) that lower germination fraction should be observed in population subject to higher parasite pressure.

Regarding animals, *Daphnia sp.* commonly named water flea are planktonic crustaceans with a particularly interesting life cycle. When conditions are optimal, adult females produce diploid eggs via parthenogenesis, these eggs become larvae directly within the female brood chamber before being released in the water column. Biotic factors such as overcrowding and scarce resources triggers the production of haploid resting eggs, named ephippia via sexual reproduction (Ebert 2005). Once released, these dormant ephippia may float in the water or sink to the sediment layer at the bottom of the water column, forming an egg banks: an assemblage of viable dormant eggs. Ephippia are long-lived and may survive in sediments for decades and even centuries (Cáceres 1998; Radzikowski 2013; Frisch *et al.* 2014). These banks are extremely large, exceeding tens thousands of eggs per square meter (Herzig 1985; Carvalho & Wolf 1989; Brendonck & De Meester 2003; Cáceres & Tessier 2004), but only a small fraction of these viable eggs hatch each growing season. For instance Radzikowski et al. estimated that only a few dozen of eggs hatch per square meter per year (2016). Sexual reproduction appears to be a plastic response to a/biotic changes in the environment, on the other hand the hatching of eggs from different generations allow for risk dispersal in time and bet-hedging theory is suggested to

explain low-hatching of dormant eggs from the top layer of sediment (Cohen 1966; Brendonck & De Meester 2003; Evans & Dennehy 2005). The remaining eggs buried slightly deeper in the sediment are trapped, as sediments create a physical barrier isolating neonates from the water column (Gleason *et al.* 2003) and low concentration of oxygen in the deeper layer of sediments may inhibit hatching (Kasahara *et al.* 1975; Uye *et al.* 1979; Lutz *et al.* 1992). Dating of sediments allow to recover the age structure of egg banks from core samples (Hairston 1996; Brendonck & De Meester 2003) and showed how large the amount of viable resting eggs is in the deep layers of sediment. To contribute to the active population, these trapped eggs require sediment mixing, caused by storms, gas bubbles and the burying of fishes and invertebrate in the sediment.

Daphnia populations are strongly affected by parasitism, and studies of the interaction of *Daphnia magna* with *Pasteuria ramosa* are particularly interesting. Indeed *D. magna* randomly receive *P. ramosa* spores while feeding, but the successful attachment and thus infection is genetically determined, a resistant host genotypes preventing the spore attachment. The D.*magna*-P.*ramosa* host-parasite system then follows a matching allele model driven by negative frequency-dependent selection (Decaestecker *et al.* 2007; Luijckx *et al.* 2013; Bento *et al.* 2017). One field study in a natural population of *D. magna* showed higher frequencies of male and resting eggs just before an annual *P. ramosa* epidemic (Duncan *et al.* 2006) suggesting that some genotypes avoid the annual epidemic by diapausing. Finally some clones of *D. magna* showed an increased investment in male production in response to the presence of hosts infected with *P. ramosa* (Duncan *et al.* 2009). Experimental studies of *D. magna* and *P. ramosa* interaction could test for the evolution of egg banks in response to parasitism, and the prediction of higher investment in the bank in population subjected to higher parasite pressure.

The egg banks of *Daphnia magna* are also influenced by the frequency of environmental disturbance and the density of the population. Indeed populations living in pounds that are susceptible to drought produce higher amounts of resting eggs than population from large lakes (Ebert 2005), and correlation between low hatching (at the top layers of the sediments) and increasing density of the population have been found (Hobaek & Larsson 1990; Kleiven *et al.* 1992). As mentioned earlier, large amounts of eggs are available in the deeper layer of the sediment but recruited only if a physical disturbance occurs. This specific structure of the egg-bank, with potential for older eggs to be recruited in the population in case of a major disturbance in the sediment, supports the last prediction of this work: the evolution of an egg structure allowing for a fast recovery of the adult population and thus a fast replenishment of the egg-bank in population facing multiple sources of variations.

Concluding remark

Beyond the influence of parasitism on the evolution of bet-hedging in hosts, this work emphasis the need for experimental studies regarding the evolvability of the age-specific recruitment of seeds or eggs. Lastly, empirical study of the evolution of seed banking is also complicated by the evolution of spatial bet-hedging as an alternative strategy to seed banking. More theoretical and empirical studies are needed to study bet-hedging across several populations of hosts and parasites to reveal the underlying evolutionary mechanisms.

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