Stochastic models for the ecology and population genetics of introduced species

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ERKLÄRUNG

Diese Dissertation wurde im Sinne von §12 der Promotionsordnung von Prof. Dr. Dirk Metzler betreut. Ich erkläre hiermit, dass die Dissertation nicht einer anderen Prüfungskommission vorgelegt worden ist und dass ich mich nicht anderweitig einer Doktorprüfung unterzogen habe.

EIDESSTATTLICHE VERSICHERUNG

Ich versichere ferner hiermit an Eides statt, dass die vorgelegte Dissertation von mir selbstständig und ohne unerlaubte Hilfe angefertigt worden ist.

Martinsried, den 26.11.2013 _____

Meike Wittmann

DECLARATION OF CO-AUTHOR CONTRIBUTIONS

The study in Chapter 1 was designed by Dirk Metzler, Wilfried Gabriel, Martin Hutzenthaler, and myself. With input from Dirk Metzler and Wilfried Gabriel, I developed the model, derived and interpreted all results and the diffusion approximation. Regarding the convergence results presented in the supplementary material, Dirk Metzler and Martin Hutzenthaler gave me an introduction to the relevant mathematical techniques. Martin Hutzenthaler pointed out key theorems from the literature and outlined important steps in the proofs, in particular for Lemmata 3 and 4 and Theorem 6. I worked out and formulated all the proofs in detail, with occasional assistance from Dirk Metzler and Martin Hutzenthaler. Martin Hutzenthaler checked my derivations and suggested ways to shorten their presentation. I wrote the entire manuscript, including the supplementary material, with feedback from Dirk Metzler, Wilfried Gabriel, and Martin Hutzenthaler.

The study in Chapter 2 was designed by Jonathan Jeschke and myself. With input from Jonathan Jeschke and Dirk Metzler, I performed all the analyses and interpreted the results. I wrote the paper and supplementary material, incorporating feedback from Jonathan Jeschke, Dirk Metzler, and Wilfried Gabriel.

The projects in Chapters 3 and 4 were designed by Dirk Metzler and myself. I developed the simulation program and performed all analyses, regularly discussing ideas with Dirk Metzler and Wilfried Gabriel. With input from Dirk Metzler and Wilfried Gabriel, I interpreted the results and wrote the manuscripts and appendices.

Meike Wittmann	Prof. Dr. Dirk Metzler

Table of Contents

When you have eliminated the impossible, what ever remains, however improbable, must be the truth. — Sherlock Holmes

Summary

The long-term success of an introduced population depends on the ecological conditions in its new environment, but is also influenced by stochasticity. This is particularly clear in the first stage of an invasion when the population is still small and either goes extinct quickly or establishes a self-sustaining population. Once established, some populations grow and spread spatially, with potential impacts on native communities and ecosystems. The role of stochasticity during these later invasion stages remains unclear. Furthermore, little is known about the population genetic and evolutionary consequences of stochastic invasion trajectories. With this dissertation, I would like to contribute to a stochastic eco-genetic theory of the entire invasion process—from the first introduction up to potential impacts. The overarching questions in this dissertation are:

a) How does a population's movement through the invasion process depend on ecological factors influencing its average growth rate?

b) How does it depend on factors influencing the stochastic variability in the population dynamics?

c) How much genetic diversity do introduced populations harbor on average upon reaching a certain point in the invasion process?

d) To what extent can the population-genetic consequences of invasion trajectories feed back onto the population dynamics?

Together with my advisors and coauthors, I have conducted four studies, each addressing two or more of these questions for specific ecological scenarios. We employ several types of stochastic models: Markov chains, Markov processes, their diffusion approximations, and coalescent-like genealogy simulations.

In Chapter 1 (Wittmann *et al.*, 2013a, appeared in *Theoretical Population Biology*), we focus on a factor influencing the introduced population's average growth rate: the intensity of competition with an ecologically similar native species. Our results indicate that

the expected time until the introduced species drives the native competitor to extinction is smallest for intermediate competition intensity. This phenomenon results from the opposing effects of competition intensity at different points of the invasion process: On the one hand, intense competition renders the establishment of the introduced population more difficult; on the other hand, it facilitates the later exclusion of the native species. In Chapter 1, we also investigate to what extent the native species' extinction is accelerated if a reduction in population size entails a reduction in genetic diversity and thus a reduced ability to adapt to a changing environment. We find this eco-genetic feedback to be particularly strong at small competition intensities.

In Chapter 2 (Wittmann *et al.*, 2013b, in press at *Oikos*), we compare introduction regimes with the same average number of individuals introduced per time unit, but with a different temporal distribution. Relative to regimes with many small introduction events, regimes with few large introduction events generate more variability in population-size trajectories. We show that this variability helps introduced populations to overcome difficult stages in the invasion process (those with a negative average growth rate), but is disadvantageous during easy stages (those with a positive average growth rate). In the light of our results, we can reinterpret three published data sets on invasion success under different introduction regimes.

In Chapters 3 and 4 (Wittmann *et al.*, 2013c,d), we examine levels of genetic diversity in populations that have successfully overcome a strong demographic Allee effect. In this ecological scenario, the average population growth rate is negative below a certain critical population size and positive above, such that the first stage in the invasion process is difficult and the second one easy. In Chapter 3, we assume Poisson-distributed offspring numbers. We show that compared to successful populations without an Allee effect, successful Allee-effect populations are expected to harbor either more or less genetic diversity, depending on the magnitude of typical founder population sizes relative to the critical population size. Part of the explanation is that, counter-intuitively, successful Allee-effect populations escape particularly fast from the range of small population sizes where genetic drift is strongest. In Chapter 3, we also identify conditions under which the critical population size can be estimated from genetic data.

In Chapter 4, we consider a range of offspring-number models leading to either more or less variability in population dynamics than the Poisson model. For a fixed founder population size, we observe that the Allee effect has a negative influence on genetic diversity for small amounts of variability, but a positive influence for large amounts of variability. We show that the differences between our various offspring-number models are so substantial

that they cannot be resolved by rescaling the parameters of the Poisson model.

Taken together, these results offer some general conclusions with respect to the four main questions raised above.

a) How fast an introduced population completes the invasion process is mainly determined by the presence and severity of difficult stages. Therefore, an ecological change promotes invasion success if it lessens such difficult stages.

b) From the perspective of the introduced population, variability is advantageous during difficult but not during easy stages of the invasion process.

c) Because the strength of genetic drift depends on population size, a key to understanding the population genetic consequences of invasion trajectories is to consider how much time the population of interest spends in different population-size ranges.

d) Feedbacks between a reduction in population size and a loss of genetic diversity are strongest in ecological scenarios where the population of interest spends considerable time at small population sizes.

Some of the most striking results in this dissertation cannot be understood from a deterministic point of view, but only when considering stochasticity. Thus, stochasticity does not just add "noise" to some average outcome, but can qualitatively change the behavior of biological systems.

Zusammenfassung

Der langfristige Erfolg einer eingeführten Population hängt von den ökologischen Bedingungen in ihrer neuen Umgebung ab, aber auch vom Zufall. Besonders offensichtlich ist die wichtige Rolle des Zufalls für kleine Populationen im Anfangsstadium einer Invasion. In diesem Stadium entscheidet sich, ob die eingeführte Population nach kurzer Zeit ausstirbt oder sich dauerhaft etablieren kann. Manche etablierten Populationen wachsen dann weiter und breiten sich räumlich aus, zum Teil mit schwerwiegenden Folgen für einheimische Gemeinschaften und Ökosysteme. Bislang ist nicht klar, welche Rolle der Zufall in diesen späteren Invasionsstadien spielt und welche populationsgenetischen und evolutionären Auswirkungen vom Zufall geprägte Invasionsverläufe haben. Mit dieser Dissertation möchte ich beitragen zu einer stochastischen öko-genetischen Theorie des gesamten Invasionsprozesses – von der Einführung bis hin zu möglichen Auswirkungen. Meine übergreifenden Fragen sind:

a) Welche Rolle für den Invasionsverlauf spielen ökologische Faktoren, die die durchschnittliche Wachstumsrate der eingeführten Population beeinflussen?

b) Und welche Rolle spielen Faktoren, die die stochastische Variabilität der Populationsdynamik beeinflussen?

c) Wie viel genetische Diversität weisen eingeführte Populationen im Durchschnitt auf, wenn sie einen bestimmten Punkt im Invasionsprozess erreichen?

d) Inwiefern können die populationsgenetischen Auswirkungen von Invasionsverläufen wiederum die Populationsdynamik beeinflussen und so zu einer Rückkopplung führen?

Zusammen mit meinen Betreuern und Koautoren habe ich vier Studien durchgeführt, die sich für bestimmte ökologische Szenarien jeweils mit mindestens zwei dieser Fragen befassen. Dazu kommen im Verlauf der Dissertation verschiedene Typen von stochastischen Modellen zum Einsatz: Markov-Ketten, Markov- und Diffusionsprozesse sowie Coalescent-artige Genealogie-Simulationen.

In Kapitel 1 (Wittmann *et al.*, 2013a, erschienen in *Theoretical Population Biology*) konzentrieren wir uns auf einen Faktor, der die durchschnittliche Wachstumsrate der Population beeinflusst: die Stärke der Konkurrenz mit einer ökologisch ähnlichen einheimischen Art. Unsere Ergebnisse deuten darauf hin, dass die erwartete Zeit bis zum Aussterben des einheimischen Konkurrenten für mittlere Konkurrenzstärken am kleinsten ist. Das können wir dadurch erklären, dass die Konkurrenzstärke gegensätzliche Auswirkungen in verschiedenen Stadien des Invasionsprozesses hat: Einerseits erschwert eine hohe Konkurrenzstärke die Etablierung der eingeführten Art, andererseits führt eine hohe Konkurrenzstärke aber auch dazu, dass die einheimische Art schnell verdrängt werden kann. Zusätzlich untersuchen wir in Kapitel 1, wie stark eine öko-genetische Rückkopplung das Aussterben der einheimischen Population beschleunigen würde. Dazu berücksichtigen wir, dass ein Rückgang der einheimischen Populationsgröße zu einem Verlust an genetischer Diversität führt, und das wiederum zu schlechterer Anpassung an veränderte Umweltbedingungen und darum weiterem Schrumpfen der Population. Unsere Ergebnisse legen nahe, dass diese öko-genetische Rückkopplung dann besonders stark ist, wenn die Konkurrenz zwischen einheimischer und eingeführter Art eher schwach ist.

In Kapitel 2 (Wittmann *et al.*, 2013b, im Druck bei *Oikos*) untersuchen wir für feste durchschnittliche Einführungsraten (Individuen pro Zeiteinheit), welche Rolle die zeitliche Verteilung der Individuen spielt. Besonders wichtig ist hierbei die Beziehung zwischen zeitlicher Verteilung und der Variabilität in der Größenentwicklung der Population. Wir zeigen, dass Fälle mit wenigen großen Einführungsereignissen zu mehr Variabilität führen als Fälle mit vielen kleinen Einführungsereignissen. Diese Variabilität hilft den eingeführten Populationen dabei, schwierige Stadien im Invasionsprozess (also solche mit einer negativen durchschnittlichen Wachstumsrate) zu bewältigen, ist aber anderseits in einfachen Stadien mit positiver durchschnittlicher Wachstumsrate von Nachteil. Im Lichte unserer Ergebnisse können wir aus der Literatur bekannte Daten zu Invasionsprozessen neu interpretieren.

In den Kapiteln 3 und 4 (Wittmann *et al.*, 2013c,d) untersuchen wir die genetische Diversität von Populationen, die einen starken demografischen Allee-Effekt erfolgreich überwunden haben. Laut Definition ist dabei die durchschnittliche Wachstumsrate bei Populationsgrößen unterhalb einer gewissen kritischen Größe negativ und in größeren Populationen positiv, so dass das erste Stadium des Invasionsprozesses schwierig ist und das zweite einfach. In Kapitel 3 zeigen wir unter der Annahme Poisson-verteilter Nachkommenzahlen, dass erfolgreiche Allee-Effekt-Populationen je nach Startgröße entweder eine höhere oder eine niedrigere durchschnittliche genetische Diversität aufweisen als erfolgreiche Popu-

lationen ohne Allee-Effekt. Das kommt zum Teil daher, dass erfolgreiche Allee-Effekt-Populationen besonders schnell das schwierige erste Stadium des Invasionsprozesses verlassen, wo genetische Drift am stärksten ist. Außerdem untersuchen wir in Kapitel 3, unter welchen Bedingungen sich die kritische Populationsgröße aus genetischen Daten schätzen lässt.

In Kapitel 4 betrachten wir eine Reihe von Modellen für die Anzahl an Nachkommen von Individuen oder Paaren in der Population. Manche dieser Modelle führen zu mehr stochastischer Variabilität in der Populationsdynamik, andere zu weniger Variabilität als das in Kapitel 3 betrachtete Poisson-Modell. Für feste Startgröße beobachten wir, dass der Allee-Effekt bei kleiner Variabilität einen negativen Einfluss auf die genetische Diversität hat und bei großer Variabilität einen positiven Einfluss. Wir zeigen weiterhin, dass die Unterschiede zwischen unseren Nachkommenzahl-Modellen so substanziell sind, dass sie sich nicht durch eine Umskalierung der Parameter des Poisson-Modells erklären lassen.

Zusammen genommen erlauben uns diese Ergebnisse einige allgemeine Schlussfolgerungen bezüglich der vier oben aufgeführten übergreifenden Fragen.

a) Wie schnell eine eingeführte Population den Invasionsprozess durchläuft, hängt hauptsächlich davon ab, ob es schwierige Stadien gibt, und wie schwierig diese sind. Deshalb begünstigt eine ökologische Veränderung den Invasionserfolg dann, wenn sie schwierige Stadien im Invasionsprozess mindert.

b) Aus der Perspektive der eingeführten Population ist Variabilität in schwierigen Stadien des Invasionsprozesses von Vorteil, aber in einfachen Stadien von Nachteil.

c) Da die Stärke der genetischen Drift von der Populationsgröße abhängt, können wir die populationsgenetischen Auswirkungen von Invasionsverläufen verstehen, indem wir analysieren, wie viel Zeit die betrachtete Population in verschiedenen Populationsgrößenbereichen verbringt.

d) Rückkopplungen zwischen einem Rückgang der Populationsgröße und einem Verlust genetischer Diversität sind am stärksten, wenn die Population viel Zeit im Bereich kleiner Populationsgrößen verbringt.

Einige der wesentlichsten Ergebnisse dieser Dissertation können aus einer deterministischen Perspektive nicht verstanden werden, sondern sind ein direktes Produkt von Stochastizität. Dies macht deutlich, dass Stochastizität nicht einfach einem gewissen Durchschnittsergebnis etwas Rauschen hinzufügt, sondern das Verhalten biologischer Systeme qualitativ verändern kann.

General Introduction

Introduced populations and the invasion process

Imagine a few hundred individuals of a plankton species being transported to a distant port in the ballast water of a cargo ship, or a few seeds of some plant species attached to the hiking boots of a traveler going back to her home country, or any other group of individuals of a certain species that is just being introduced, either accidentally or on purpose, to a location where the species has been previously absent. Now imagine you could fast-forward and see the same location 100 years later. What will have happened to the introduced population? Will it be extinct now? Will it still be small, hardly noticeable, and without any consequence for native communities? Or will it be a common, widespread species? Has there been evolutionary change in the population? Will native communities and ecosystems have changed as a result of its invasion?

These are some of the key questions of invasion biology, a branch of biology that is concerned with the ecology—and sometimes evolution—of populations outside their native range. To better understand the fate of introduced populations, invasion biologists have developed a helpful abstraction: the invasion process (see Glossary). This concept stands for the spectrum of possible states of an invasion, delimited on the one extreme by the point where the focal species is absent and, on the other extreme, by the state in which the species is large, widespread, and possibly has large impacts on native communities, ecosystems, or even on human health and economy. Usually, invasion biologists think of this spectrum as subdivided in some way. For example, Williamson & Fitter (1996) distinguish between four stages: at any point in time an introduced population can be either imported, introduced, established, or a pest species. Richardson *et al.* (2000), on the other hand, have focused on the barriers that can prevent the transition of introduced populations through the process. After their arrival, for example, populations first face the environmental barrier: abiotic and biotic conditions that might be unsuitable and prevent the survival of the introduced individuals. Later, they have to overcome a reproduction barrier and a dispersal barrier. A

population's position in the invasion process is then defined by how many barriers it has passed already. Recently, Blackburn *et al.* (2011) have combined both perspectives, the one centered on stages and the one centered on barriers, into a unified picture of the invasion process.

Differentiating between several stages in the invasion process is helpful in several ways. First, the stages in the invasion process can be used as a basis for terminology, for example to agree on what it means for a species to be "naturalized" or "invasive" (Richardson *et al.*, 2000; Colautti & MacIsaac, 2004). Although this sounds trivial, there has been much debate about such issues and many authors think that confusion about terminology is seriously impeding the progress of invasion biology as a scientific discipline (Davis & Thompson, 2000; Richardson *et al.*, 2000; Heger *et al.*, 2013). Second, the influence of various biotic and abiotic factors seems to depend on the current position of the introduced population in the invasion process. For example, migratory behavior in birds appears to be disadvantageous at the establishment stage, but advantageous for later spread of the population (Kolar & Lodge, 2001). Thus, lumping together these stages would yield ambiguous results: One study might find a positive effect, a second study no effect, and yet another study a negative effect of such a factor on invasion success. Furthermore, it does not seem to be the case that those species that have a high likelihood of establishing or spreading at new locations are also the ones that cause large impacts (Ricciardi & Cohen, 2007). Thus, depending on the research question, we must carefully consider which stage of the invasion process needs to be investigated. Finally, a subdivision of the invasion process can also guide the choice of management actions (Lodge *et al.*, 2006). For example, complete eradication may be feasible only for populations in the early stages of the invasion process. If a population is in a late stage of the invasion process, that is large and widely spread, it may be more reasonable to just control and slow its spread.

Overview of this dissertation

The aim of my dissertation was to develop and analyze mathematical models that help us to understand how ecological, evolutionary-genetic, and stochastic effects interact as the introduced population moves through the invasion process. Together with my coauthors, I have conducted four studies within this general context. They correspond to the four chapters of this dissertation. The main factors and processes of interest in each chapter are summarized in Fig. I.1. In Chapter 1 (Wittmann *et al.*, 2013a), we investigate how the intensity of competition with a native species influences the introduced population's move-

ment through the invasion process. We further quantify a feedback between the growth of the introduced population and loss of genetic diversity in the native species. In Chapter 2 (Wittmann *et al.*, 2013b), we study how the temporal distribution of introduced individuals, one source of variability, affects invasion dynamics. We compare the results among a variety of invasion processes, either with interspecific competition, as in Chapter 1, or with a demographic Allee effect, i.e. an average per-capita growth rate that increases with population size in small populations (Stephens *et al.*, 1999). Chapter 3 (Wittmann *et al.*, 2013c) is concerned with the population genetic consequences of population-size trajectories under a strong demographic Allee effect, i.e. a demographic Allee effect in which the average per-capita growth rate is negative for small population sizes (Taylor & Hastings, 2005). Chapter 4 (Wittmann *et al.*, 2013d) builds on Chapter 3 and examines how the population genetic consequences of the Allee effect are influenced by variation in offspring number among individuals in the population. In the remainder of this General Introduction, I will first outline our approach to quantifying the invasion process, specifically under interspecific competition or an Allee effect. I will then explain why we need stochastic models to understand invasion dynamics and review current knowledge on how stochasticity interacts with competition and an Allee effect. Finally, I will discuss population genetic and evolutionary consequences of invasion trajectories.

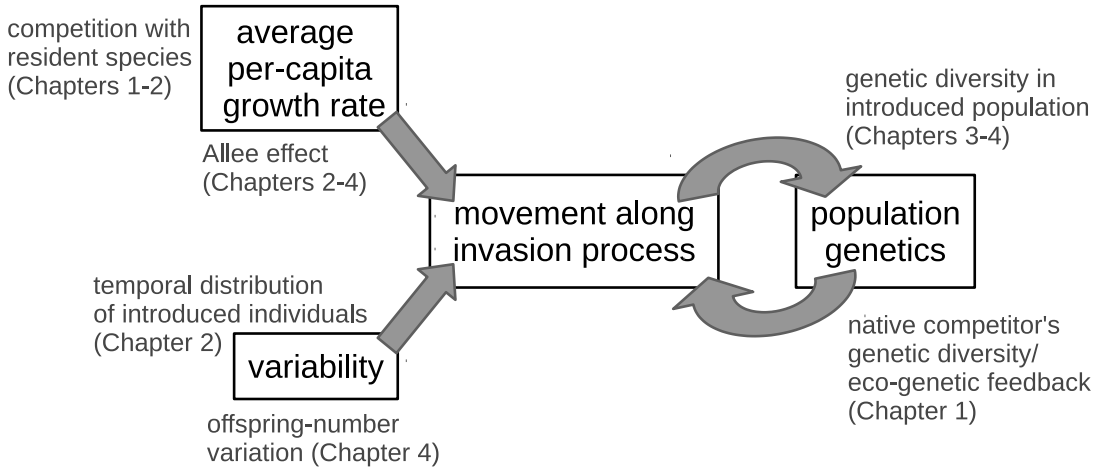

Figure I.1: Overview of the various factors and processes that are investigated in the four chapters of this dissertation.

Quantifying the invasion process

In this dissertation, we focus on single, homogeneous introduced populations and use the current population size to locate them in the invasion process. We will characterize the invasion process by specifying the average per-capita growth rate of the introduced population as a function of its current population size (Fig. I.2). For the understanding of many results in this dissertation, it will be helpful to distinguish between "easy" and "difficult" stages in the invasion process. An easy stage is characterized by a positive average per-capita growth rate, whereas a difficult stage has a negative average per-capita growth rate, and thus can be considered analogous to one of Richardson *et al.*'s (2000) barriers.

Of course, a one-dimensional characterization of the invasion process is a simplification. Other quantities varying along with the size of the introduced population may also influence invasion success, for example the population sizes of competitors, predators, and prey, or the genetic structure of the introduced population. At various points in this dissertation, we will explore ways to incorporate such an additional dimension into our models. In models with a spatial component, one would also need to include a measure of range size to quantitatively determine the introduced population's invasion status. The various classification schemes discussed above are often based on complex criteria, for example whether a population is already self-sustained or not. However, it can be challenging to apply these criteria to the data at hand. Since local abundance and range size information is more commonly available, these measures have also been used elsewhere as a proxy for position in the invasion process (see e.g. Murphy *et al.*, 2006).

The magnitude of the average per-capita growth rate and its dependence on population size is shaped by various factors: the number of individuals arriving per time unit (propagule pressure, see Glossary), the abiotic conditions in the new environment, the life-history of the introduced species itself, and interactions with resident species. In this dissertation, we will focus on the role of interspecific competition and the Allee effect in shaping the invasion process.

Interspecific competition can give rise to very different invasion processes depending on the intensity of competition between introduced and resident species. If interspecific competition is weaker than intraspecific competition, the rare species has an advantage. In this case, the first stage of the invasion process is easy and the second stage is difficult (Fig. I.2 A). By contrast, if interspecific competition is stronger than intraspecific competition, the more common species has an advantage. Accordingly, the first stage in the invasion process is difficult and the second stage is easy (Fig. I.2 B). Thus, an increase in the in-

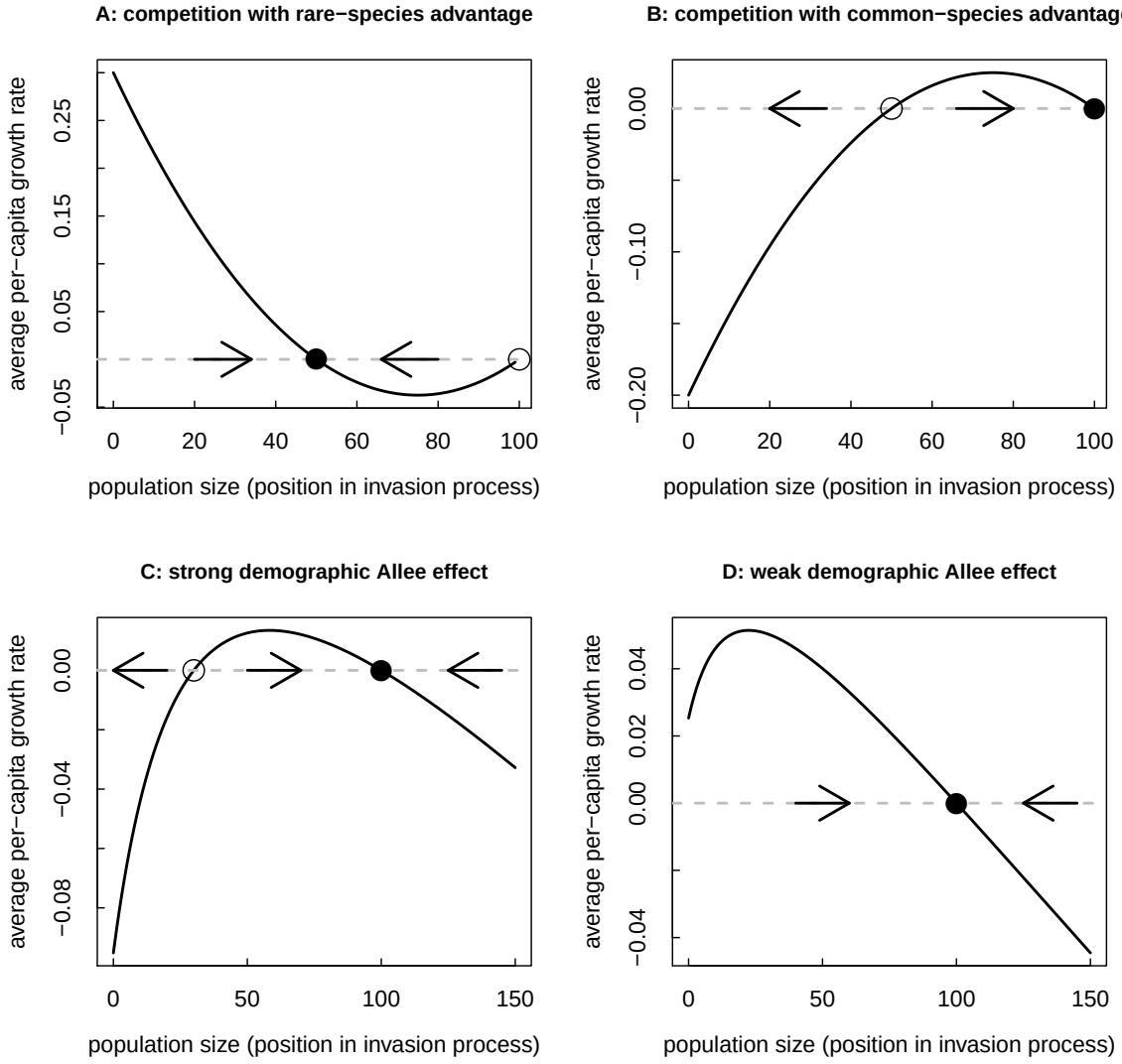

Figure I.2: Four example invasion processes. Arrows indicate the average movement of populations through the invasion process: During easy stages, i.e. those with a positive average per-capita growth rate, arrows point to the right, indicating population growth. During difficult stages, i.e. those with a negative average per-capita growth rate, populations tend to decline and thus arrows point to the left. Solid dots indicate points that would be stable equilibria in a deterministic system; open dots indicate unstable equilibria. The dashed gray line corresponds to an average per-capita growth rate of zero.

tensity of competition has opposing effects on the success of the introduced population at different invasion stages: it impedes its establishment, but facilitates its growth once it is very common and makes it more likely that the introduced species will become dominant in the community and exclude the resident competitor. Given these opposing effects at different invasion stages, it is not surprising that the influence of interspecific competition on invasion success has been rather controversial (Moulton & Pimm, 1983; Moulton, 1985; Simberloff & Boecklen, 1991; Moulton, 1993; Duncan, 1997; Davis, 2003). One of the main contributions of Chapter 1 (Wittmann *et al.*, 2013a) is to clarify this double-edged role of interspecific competition in the invasion process and to quantify the resulting overall effect. Specifically, we investigate how competition intensity influences the expected time until the introduced population reaches the end of the invasion process, which we define as the point where it drives the resident species to extinction.

Figs. I.2 C and D display invasion processes generated by a demographic Allee effect, a phenomenon featured in Chapters 2–4. If the per-capita growth rate is negative at small population sizes, the Allee effect is called strong (Taylor & Hastings, 2005) and the first stage of the invasion process is difficult (Fig. I.2 C). If, on the other hand, the average per-capita growth rate is reduced but still positive at small population sizes, the Allee effect is called weak (Taylor & Hastings, 2005) and the invasion process starts with an easy stage (Fig. I.2 D).

A stochastic theory of the invasion process

So far, our perspective on the invasion process has been entirely deterministic. We have described it in terms of average per-capita growth rates, and stable or unstable equilibria (Fig. I.2). In this section, I will point out three ways in which stochasticity shapes the progress of the introduced population, and thereby argue for a stochastic theory of the invasion process.

Why do we need a stochastic theory of the invasion process?

First and most obviously, introduced populations are almost always very small. In a population that consists of few individuals, it can happen by chance that all individuals die before leaving any offspring or that they produce offspring of only one sex. This type of stochasticity is called demographic stochasticity and can cause the extinction of small populations even in scenarios where a deterministic model would predict their growth (as for example in Fig. I.2 A and D). Since the randomness at the level of individuals increasingly averages

out as the population becomes larger, the importance of demographic stochasticity quickly diminishes with increasing population size. Notably, the expected time until a population goes extinct due to demographic stochasticity increases exponentially with population size (Lande, 1993).

Second, in scenarios where the first stage of the invasion process is difficult (as for example in Fig. I.2 B and C), deterministic models would always predict the extinction of small introduced populations. Successful establishment under such scenarios is certainly rather unlikely. Many species, however, are regularly transported around the world by humans, either intentionally as is the case for garden plants, pets, and game species, or unintentionally in the case of many invertebrates that are traveling as stow-away in ballast water or other means of cargo transport. If a given species arrives to a certain location not just once, but again and again, even unlikely events can occur eventually. For example, the European red deer *Cervus elaphus* needed at least 32 introduction attempts to New Zealand, before it finally established and spread (Simberloff, 2009). Since a single successful population among many failed ones can be responsible for large impacts, knowing that the average introduced population will go extinct is not sufficient. We need stochastic models to understand the factors that cause populations to deviate from this expectation and the properties of such exceptional populations.

Third, stochasticity can still be important when a population leaves the range of small population sizes (Black & McKane, 2012). For many introduced species, so-called lag times have been observed: an introduced species proceeds to a certain stage in the invasion process and then remains there for a long time (Kowarik, 1995; Crooks, 2005; Simberloff, 2009). A plausible explanation for this phenomenon is that the population fluctuates for a long time around a point that would be a locally stable equilibrium in a deterministic model (e.g. the locally stable point indicated by a solid dot at intermediate population size in Fig. I.2 A) and that eventually one of these fluctuations is large enough to let the population overcome the subsequent difficult stage and continue its progress through the invasion process. Since a deterministic model would predict that the introduced population remains at the stable point forever, we need stochastic models to understand and quantify the movement of populations through difficult stages in the invasion process.

Modeling stochasticity

To define a meaningful stochastic model, it is not sufficient to take a deterministic model and add some "noise" to it; we have to be precise about the form of the variability that enters

the model. Apart from demographic stochasticity, there are two other forms of stochasticity that are important for introduced populations. Environmentally driven fluctuations in demographic parameters, such as birth rates or death rates, are called environmental stochasticity. In contrast to demographic stochasticity, environmental stochasticity does not lose importance in large populations because all individuals in a population are jointly affected by the environment. Another important source of stochasticity is the introduction process itself, i.e. temporal fluctuations in the number of individuals introduced.

All models in this dissertation include demographic stochasticity. It arises directly from the way we model birth and death events in the various projects. We additionally included environmental stochasticity in the eco-genetic model in Chapter 1 (Wittmann *et al.*, 2013a), and in Appendix 4 of Chapter 2 (Wittmann *et al.*, 2013b). In two cases, we focus in particular on how variability affects invasion success: In Chapter 2 (Wittmann *et al.*, 2013b), we consider variability deriving from the introduction process. We compare introduction regimes with the same average number of individuals introduced per time unit, but with a different temporal distribution. Introduction regimes with few large introduction events (large propagule size, small propagule frequency, see Glossary) are associated with more variability than regimes with many small introductions (small propagule size, large propagule frequency). In Chapter 4 (Wittmann *et al.*, 2013d), we investigate the role of offspring-number variation, a component of demographic stochasticity.

There is a variety of modeling approaches in invasion biology (see Box 1 for an overview), a small proportion of which include some form of stochasticity. Most stochastic models in invasion biology are concerned with the establishment probability of introduced populations under demographic and environmental stochasticity. Using branching processes, birth-death processes, or diffusion processes (see Glossary for definitions), formulas for the probability that an introduced population reaches a certain high population size before going extinct have been derived (Richter-Dyn & Goel, 1972; Haccou & Iwasa, 1996; Haccou & Vatutin, 2003). In contrast to branching processes, birth-death processes and diffusion processes can include different kinds of density dependence, although this usually makes it difficult or even impossible to find analytical solutions for quantities of interest. With the help of numerical methods, however, such models have already yielded important insights into the role of stochasticity in scenarios with Allee effect or interspecific competition.

Box 1: Mathematical models in invasion biology

From a methodological standpoint, models in invasion biology reflect the whole diversity of mathematical modeling approaches in ecology. They range from simple statistical models (e.g. Veltman *et al.*, 1996), over classical population models based on differential or difference equations (e.g. Crawley, 1986), to detailed individual-based simulation models (e.g. Byers & Goldwasser, 2001; Higgins *et al.*, 2001). Models either focus on a single alien species (e.g. Drake & Lodge, 2006), or take into account interactions with one or more resident species (Duncan & Forsyth, 2006), or even the whole network of interactions in the community (e.g. Romanuk *et al.*, 2009). Some models have taken into account life history and stage structure in alien populations (e.g. Thomson, 2005; Wittmann *et al.*, 2011). In the main text, we will discuss stochastic models in more detail. Here, I will just highlight two particularly popular modeling approaches in invasion biology:

Models for the spatial spread of invasions (see Hastings, 1996; Hastings *et al.*, 2005, for an overview) often take the form of partial differential equations that include a term for the local population dynamics and a term for diffusive dispersal. Under quite general assumptions, such models give rise to a traveling wave of population expansion in which the population density profile at the colonization edge stays constant in time and simply moves in space with a constant velocity. The results of such models fit quite well to data on some real-world expansions, for example of the muskrat (Skellam, 1951).

Ecological niche modeling = bioclimatic/ species distribution modeling (Peterson & Vieglais, 2001; Peterson, 2003; Jeschke & Strayer, 2008). The idea here is to use environmental information (e.g. on temperature, rainfall etc.) and presence-absence data from the current geographical range of a species to build a statistical model for its ecological niche, i.e. for the abiotic conditions under which the species is able to persist. This statistical model can then be projected onto geographical areas where the species has not arrived yet to predict its potential future range (Peterson, 2003). Major limitations of bioclimatic models are that they do not account for biotic interactions, or phenotypic changes over the course of an invasion, and that the absence of the species from a location within its current range is always ascribed to abiotic factors and not to a failure to disperse to the location or chance extinction (Jeschke & Strayer, 2008).

Models in invasion serve a variety of purposes, for example

- risk assessment (e.g. Drake, 2004)
- predicting the future range of species (e.g. Peterson & Vieglais, 2001)
- predicting or understanding impact on particular native species (e.g. Byers & Goldwasser, 2001)
- evaluating management options (e.g. Shea & Possingham, 2000)
- improving our understanding of basic ecological and evolutionary processes

Stochasticity and the Allee effect

Dennis (2002) incorporated a strong demographic Allee effect (see Fig. I.2 C) into stochastic population models, in this case diffusion processes. He derived formulas (involving integrals over shifted Gamma distributions) for the probability that a population reaches some upper threshold size b before reaching some lower threshold size a. Plots of this first-passage probability over a range of initial population sizes between a and b have an inflection point at the critical population size (Fig. I.3). For extremely low levels of stochasticity, this function looks almost like the deterministic step function: populations below the critical population size decline and never reach b, whereas populations above the critical size grow and reach b with probability 1. With increasing stochasticity, the curve becomes more shallow: the first-passage probability for population sizes below the critical size increases while the first-passage probability for population sizes above the critical size decreases. Furthermore, in the case of continuous immigration, an increase in stochasticity has been shown to decrease the expected time a small population needs to overcome the Allee effect and reach some high population size (Potapov & Rajakaruna, 2013). In summary, variability promotes the success of introduced populations below the critical population size, but is detrimental for populations above the critical size because it increases the risk that these populations will fall below the threshold and go extinct.

Freckleton *et al.* (2006) provide a case study for the interplay between the Allee effect and stochasticity. On Indo-Pacific coral reefs, the invasive crown-of-thorns starfish *Acanthaster planci* exhibits a predator-induced Allee effect: In small populations, individual starfish have a higher probability to be eaten by a predatory fish. In a stable, deterministic system, this effect could prevent the invasion of the starfish. However, commercial fishing leads to variability in the abundance of predatory fishes, i.e. environmental stochasticity. It appears that a temporary reduction in predation pressure can allow crown-of-thorns starfish populations to escape from the Allee effect.

Stochasticity and interspecific competition

While the importance of stochasticity in Allee-effect scenarios is broadly recognized, the role of interspecific competition with resident species is more often considered from a deterministic perspective (e.g. Case, 1990, 1995; Miller *et al.*, 2009; Wittmann *et al.*, 2013e). In one of the few studies incorporating stochasticity, Duncan & Forsyth (2006) simulated a two-species birth-death process to compute the establishment probability of an introduced species facing competition from a resident species. Their results can be interpreted as fol-

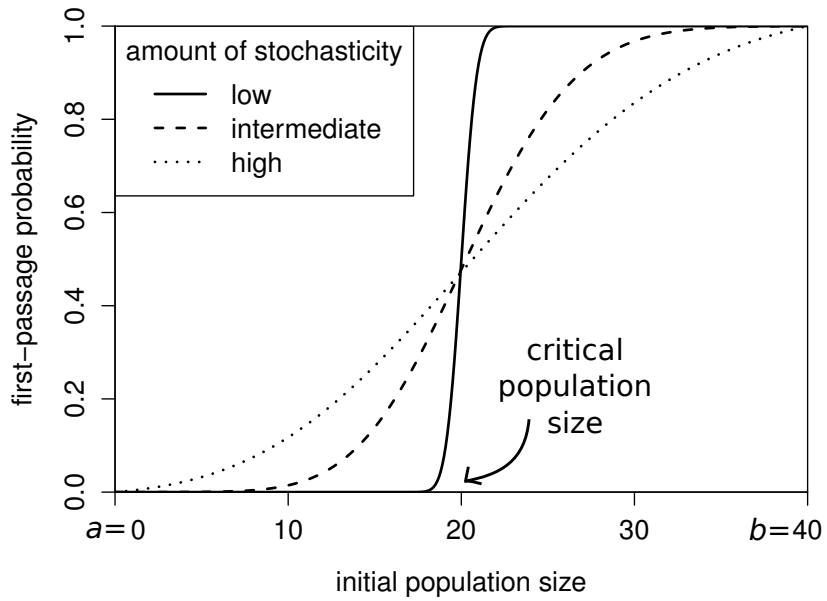

Figure I.3: First-passage probabilities (probability to reach population size b before a) with a strong demographic Allee effect and various amounts of stochasticity. The critical population size is 20. Curves are based on equation (34) in Dennis (2002).

lows: in cases where interspecific competition is weaker than intraspecific competition (as in Fig. I.2 A), the establishment probability of the introduced population is high and depends relatively little on the initial population size of the competitor. On the other hand, if interspecific competition is stronger than intraspecific competition (as in Fig. I.2 B), establishment probability strongly decreases with increasing initial population size of the competitor. Consequently, if environmental stochasticity leads to a temporal reduction in the competitor's population size, demographic stochasticity or stochasticity in the introduction process may allow the introduced population to establish.

This effect is illustrated by another of Freckleton *et al.*'s (2006) case studies: In Australian pastures, the introduced grass *Vulpia bromoides* is strongly competing with a native grass, *Lolium rigidum*. Fitting simple competition models to data from field experiments revealed that interspecific competition is stronger than intraspecific competition in this system, such that the coexistence of the two species is unstable (like in Fig. I.2 B). Thus, in a deterministic world, *Vulpia* would not be able to invade pastures dominated by *Lolium*. Every few years, however, severe droughts reduce the densities of all species in the system to low levels. This form of environmental stochasticity appears to facilitate the invasion of *Vulpia* because the introduced population more rapidly recovers after a drought than the native population.

Both in the case of an Allee effect and in the case of competition, previous research

has focused on the first stage of the invasion process. However, as I have argued above, stochasticity can also be important at later stages of the invasion process. Currently, there is no stochastic theory for a population's journey through the entire invasion process. Furthermore, there has been little research on the population genetic and evolutionary consequences of invasion trajectories.

Invasion trajectories and their genetic consequences

The average population dynamics and the associated variability not only influence whether and when a population reaches a certain point in the invasion process, but also the path it takes to get there. These population-size trajectories tell us how much time the population has spent at intermediate population sizes before first reaching a certain population size. Such information plays a large role for levels of genetic diversity in introduced populations because the average proportion of genetic variation that is lost through genetic drift per generation is inversely proportional to population size. Since it takes a long time until heterozygosity is restored by new mutations, introduced populations carry the genetic traces of their early history even after they have already reached high population sizes (Fauvergue *et al.*, 2012).

To understand patterns of neutral genetic variation and their dependence on demographic events like founding events, population growth or decline etc., population geneticists often use coalescent theory (see Wakeley, 2009, for an introduction). The coalescent is a continuous-time stochastic model for sample genealogies in large populations. The central idea is to trace the sampled lineages backwards in time and to fuse (coalesce) two ancestral lineages when their descendants have a common ancestor at that point in the past. Since the rate at which two lineages coalesce is inversely proportional to population size, sample genealogies from small populations tend to have shorter branches than those from large populations, and thus in small populations there is a reduced opportunity for mutations to generate variation that will be visible in the sample.

Coalescent theory is an elegant and powerful framework in population genetics, but when it comes to patterns of genetic diversity in small introduced populations we have to apply it with caution. For example, it is possible in small populations that three or more lineages coalesce, or that multiple coalescent events occur in a single generation. Such multiple and simultaneous mergers become very improbable as the population sizes increases and therefore do not occur in the standard coalescent. For this dissertation, however, I developed a simulation program that is specifically designed for the simulation of genealogies

and genetic data in small introduced populations. I simulate the stochastic demography forward in time, and then trace the sampled lineages backward in time generation by generation using a modified version of the coalescent that takes into account the particularities of small populations. In Chapters 3 and 4 (Wittmann *et al.*, 2013c,d), we use this simulation program to study levels of genetic diversity in introduced populations that have successfully overcome a strong demographic Allee effect.

The genetic consequences of population-size trajectories are of interest because 1) they provide an opportunity for parameter inference and 2) they have implications for the evolutionary potential of the introduced species and interacting native species, with possible feedback on their population dynamics. We will explore these aspects in turn in the next two sections.

Opportunities for parameter estimation from genetic data

If the movement of the introduced population through the invasion process leaves traces in genetic data (e.g. in microsatellite, SNP, or DNA sequence data), it might be possible to employ these patterns to infer parameters of the invasion process, such as growth rates and founder population sizes. Since the inference of demographic parameters is of interest not only in invasion biology, statistical population geneticists have developed a multitude of methods to estimate such parameters from various types of genetic data. One important class are full-data likelihood methods that employ Markov Chain Monte Carlo algorithms to approximate the probability of obtaining the complete observed data set, given the parameters, i.e. the likelihood of the parameters (see Glossary). Prominent examples for this approach are implemented in the programs GENETREE (see e.g. Griffiths & Tavaré, 1997), IM (Hey & Nielsen, 2004), and LAMARC (Kuhner, 2006). Other methods reduce the complexity of the data set by first condensing it to a set of summary statistics, for example Tajima's D (Tajima, 1989), indices for population differentiation such as F_{ST} (Wright, 1949) or Jost's D (Jost, 2008), or the entries of the (joint) site frequency spectrum ((J)SFS) of samples from one or two populations (see Glossary for definitions). The subsequent estimation procedure is then based for example on diffusion theory, as is the case in $\partial a \partial i$ (Gutenkunst *et al.*, 2009), or a composite-likelihood approach assuming that sites are effectively independent of each other, as in Jaatha (Naduvilezhath *et al.*, 2011; Mathew *et al.*, 2013a,b).

Despite this host of available methods, it remains challenging to find one that suits the needs of recently introduced populations. In a pessimistic perspectives article on the topic,

Fitzpatrick *et al.* (2012) argue that the utility of genetic data for parameter inference in invasion biology is limited by the short time scale on which the events of interest usually happen and by the fact that introduced populations often violate assumptions of classical population genetic analyses. It is true that especially the full-likelihood methods are restricted to specific models, since the evaluation of the likelihood function is quite difficult in general. For example, the model underlying LAMARC (Kuhner, 2006) assumes that the population structure has been stable for a long time, an assumption that is certainly not fulfilled for most introduced populations. Furthermore, most methods rely at their core on coalescent simulations, which only provide an accurate approximation for sample genealogies if populations are reasonably large. As we have seen in the previous section, the genealogies of small populations can exhibit features that are not observed in large populations.

One approach that is flexible enough to accommodate all particularities of small introduced populations is Approximate Bayesian Computation (ABC), a method allowing for the estimation of parameters from arbitrarily complex models, as long as they can be simulated in a reasonable amount of time (see Beaumont, 2010; Csilléry *et al.*, 2010, for an introduction). This method is based on a comparison of summary statistics between the observed data set and a large number of simulated data sets whose parameter values have been drawn from some prior distribution. In the simplest version of ABC, Pritchard *et al.*'s (1999) rejection algorithm, simulation runs with summary statistics that are too far away from the observed summary statistics are rejected. More specifically, if $S_1^*, \ldots, S_n^*$ are the observed summary statistics, a simulation run with summary statistics $S_1, \ldots, S_n$ is accepted if $|S_i - S_i^*|/S_i^* < \delta$ for all i in $\{1, \ldots, n\}$, where $\delta > 0$ is the rejection threshold. The parameter combinations of the accepted simulation runs are then taken as an approximate sample from the posterior distribution.

In principle, ABC poses no restrictions on the type and number of summary statistics, and thus it is even possible to combine genetic summary statistics with ecological information such as measurements of population sizes. In practice, however, the curse of dimensionality can strike: If δ is kept fixed, the proportion of simulations that fall within the acceptance region decreases with an increasing number of summary statistics. Consequently, the estimation of the posterior distribution is based on a smaller sample size and therefore more variable. If, on the other hand, the proportion of simulations to be accepted is kept fixed, the distance between the summary statistics of the accepted simulations and those of the observed data set tends to increase with an increasing number of summary statistics, leading to a bias in the estimation of the posterior distribution. One strategy to overcoming this "bias-variance tradeoff" is to use local linear or nonlinear regression to re-

duce the bias that results from accepting simulations whose summary statistics are at some distance from the true ones (Beaumont *et al.*, 2002; Blum & François, 2010). Another strategy is to find or construct a limited number of summary statistics that contain the most relevant information about the parameters of interest. Several statistical methods have been devised for this purpose (Boulesteix & Strimmer, 2007; Joyce & Marjoram, 2008; Nunes & Balding, 2010), but also biological insight can help to decide what aspects of the data will be most useful.

In the case of historically introduced populations, we generally do not expect many new mutations to have happened since the introduction event, except maybe for organisms with very short generation times. Thus, we cannot rely on such new mutations for demographic inference in most introduced organisms. However, there is other important genetic changes that do occur in small introduced populations: the genetic variation imported from the source region is subjected to genetic drift. As discussed above, the strength of genetic drift and hence how much of the variation is lost depends on the population-size trajectory. Therefore, we might be able to infer at least the founder population size, i.e. the strength of the bottleneck associated with the introduction event, and some measure of population growth. That genetic data can contribute to the inference of founder population sizes, even if ecological information is also available, has been demonstrated by Estoup *et al.* (2010). Keeping in mind the limitations of population genetic inference in introduced populations, we explore in Chapter 3 (Wittmann *et al.*, 2013c) under what conditions we could employ the population genetic patterns generated by a strong demographic Allee effect to infer the critical population size. We base our estimation on the site-frequency spectrum of samples from the introduced population, use partial least squares (Mevik & Wehrens, 2007) to reduce the number of summary statistics, and finally use ABC with linear regression adjustment (Beaumont *et al.*, 2002) to approximate the posterior distribution of the critical population size.

Evolutionary potential of introduced populations and eco-genetic feedbacks

A second motivation for studying the population genetic consequences of invasion trajectories is that levels of genetic diversity can influence the evolutionary potential of an introduced population: its ability to adapt to the abiotic and biotic conditions in the new environment and to persist in the face of environmental change. To demonstrate this connection between population size and evolutionary potential, Frankham *et al.* (1999) subjected ex-

perimental populations of *Drosophila melanogaster* first to a strong bottleneck and then to increasing salt concentrations. All populations evolved an increased salt tolerance, but, compared to populations that had not experienced a bottleneck, the bottlenecked populations went extinct at smaller salt concentrations.

Such direct evidence for a reduction in evolutionary potential after a reduction in population size is rare, however, and conclusions are often based on levels of neutral genetic diversity. A meta-analysis by Reed & Frankham (2001) has revealed that levels of genetic diversity at neutral markers correlate only weakly with the amount of additive genetic variance in quantitative traits (see Glossary) which is the basis of most evolutionary adaptation. This was also the case for allozyme heterozygosity (a measure of neutral genetic variation) and genetic variation in bristle number (a quantitative trait) in experimentally bottlenecked *Drosophila melanogaster* populations (Gilligan *et al.*, 2005). Nevertheless, both measures of diversity had a similar relationship with bottleneck size. Thus, the authors conclude that uncertainty in the estimates of genetic variation is the most likely explanation for the weakness of the correlation between them. In any case, we should be aware that levels of genetic diversity at neutral markers are not a perfect indicator for evolutionary potential.

Another complication is that there can even be an increase in additive genetic variance immediately after a bottleneck, as has been observed for example in experimentally bottlenecked houseflies (Bryant *et al.*, 1986). This observation can be explained either by a conversion of epistatic genetic variance into additive genetic variance (Goodnight, 1988), or by the presence of rare recessive alleles (Willis & Orr, 1993). These effects, however, are expected to be short-lived (Frankham, 2005) and overall strong bottlenecks as they occur in many species introductions should lead to a reduction in long-term evolutionary potential.

If a reduction in genetic diversity prevents a population from responding appropriately to environmental change, its population size may further decline, leading to additional loss of genetic diversity, and so forth until the population goes extinct. This mechanism—we will call it an eco-genetic feedback—is an example for an extinction vortex (Gilpin & Soulé, 1986), a synergistic interaction between different processes that can drive small populations to extinction: demographic and environmental stochasticity, the Allee effect, habitat fragmentation, loss of genetic variation, mutation accumulation, and inbreeding depression. Extinction vortices may affect small introduced populations, but of course also interacting resident populations, for example if an introduced population drives a resident competitor or prey species to small population sizes. In Chapter 1 (Wittmann *et al.*, 2013a), we quantify such an eco-genetic feedback in the impacts of an introduced population on a resident competitor in a variable environment.

General aims of this dissertation

In summary, the general aim of this dissertation is to develop and analyze stochastic models to further our understanding of the ecology and population genetics of an introduced population as it moves through the invasion process. More specifically, we investigate

a) the role of ecological factors that shape the average per-capita growth rate and its dependence on population size,

b) the role of ecological factors that influence the variability in population-size trajectories,

c) how the movement through the invasion process shapes levels of genetic diversity within the introduced population and possibly interacting resident populations, and

d) the eco-genetic feedbacks that may occur when the genetic consequences of population-size trajectories in turn influence the population dynamics.

In the General Discussion, we will come back to these four questions to synthesize and discuss the findings of the four chapters.

Chapter 1

Ecological and genetic effects of introduced species on their native competitors

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Ecological and genetic effects of introduced species on their native competitors

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ABSTRACT

Species introductions to new habitats can cause a decline in the population size of competing native species and consequently also in their genetic diversity. We are interested in why these adverse effects are weak in some cases whereas in others the native species declines to the point of extinction. While the introduction rate and the growth rate of the introduced species in the new environment clearly have a positive relationship with invasion success and impact, the influence of competition is poorly understood. Here, we investigate how the intensity of interspecific competition influences the persistence time of a native species in the face of repeated and ongoing introductions of the nonnative species. We analyze two stochastic models: a model for the population dynamics of both species and a model that additionally includes the population genetics of the native species at a locus involved in its adaptation to a changing environment. Counterintuitively, both models predict that the persistence time of the native species is lowest for an intermediate intensity of competition. This phenomenon results from the opposing effects of competition at different stages of the invasion process: With increasing competition intensity more introduction events are needed until a new species can establish, but increasing competition also speeds up the exclusion of the native species by an established nonnative competitor. By comparing the ecological and the eco-genetic model, we detect and quantify a synergistic feedback between ecological and genetic effects.

1. Introduction

When a new species is introduced to a location where it did not occur before, it begins to interact with the resident species, for example as a predator, mutualist, or competitor. These interactions are critical in determining the fate of the introduced species and whether and how the community changes in response to the introduction. In this study, we investigate the effects of introduced species on the native species with which they are competing for resources, such as food or territories. Often there is considerable variation between geographical locations in the impacts of an introduced species on a particular native competitor, e.g. in container-dwelling mosquitoes in Florida (Juliano, 1998) or for fish introductions to California (Herbold and Moyle, 1986). Thus the question is: Why does the native species suffer from severe impacts or even goes extinct in some places, but not in others?

While it is evident that the probability for an introduced species to become a high-impact invader at a certain location increases with introduction rate (also known as propagule pressure, one of the most important factors in invasion biology, Duncan, 1997, Drake et al., 2005, Lockwood et al., 2005) and with the species' growth rate in the new environment (Rejmánek and Richardson, 1996; Wu et al., 2005), the role of competition has remained controversial (Herbold and Moyle, 1986; Duncan, 1997; Davis, 2003). Some empirical studies suggest that differences in the intensity of interspecific competition can explain differences in impacts, e.g. in the competitive interaction between native bumble bees and introduced honey bees in California, where the overlap in the flowers visited by honey bees and bumble bees was used as a proxy for the intensity of interspecific competition (Thomson, 2006). However, it has been difficult to disentangle the effect of competition from that of other variables and no consensus has emerged yet. Here we contribute a first theoretical building block towards an understanding of how competition intensity influences introduced species impacts.

Considerable impacts of an introduced species on a native species or even extinction of the native species can only occur if the introduced species completes two major stages of the invasion process: The establishment stage starts with the introduction of one or more founding individuals. This new population can then either go extinct, in which case it has to await the next introduction event, or increase in size until it is of the same order of magnitude as the competing native population. From there, the introduced species can proceed to a second stage in which it becomes dominant and

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may eventually exclude the native species from the community. Thus far, knowledge on the effect of competition is limited to single stages of the invasion process.

At the establishment stage, the intensity of competition with native species appears to have a negative effect on the success of introduced species. With phylogenetic relatedness as a proxy for the intensity of competition, this intuitive idea goes back to Darwin (1859, Chapter 4). Darwin's naturalization hypothesis, as it is formulated nowadays, states that introduced species should be less successful at locations where closely related species are already present (Duncan and Williams, 2002). Surprisingly, the opposite pattern is sometimes observed in historical data, for example for plants introduced to Hawaii and New Zealand (Daehler, 2001; Duncan and Williams, 2002). A possible explanation is that closely related species not only compete with each other but also share characteristics that may confer a high intrinsic ability to survive and grow at the new location (Duncan and Williams, 2002).

In the only experimental study on Darwin's naturalization hypothesis that we know of, however, the establishment success of an invader in a microcosm bacterial community increased with the average phylogenetic distance to the recipient community (Jiang et al., 2010). In this case, it has also been confirmed that more closely related species had a higher overlap in resource use and were therefore competing more intensely. Additional support for the negative effect of competition at the establishment stage comes from historical bird introductions to Hawaii: Moulton and Pimm (1983) and Moulton (1993) observed a negative correlation between establishment success and the number of bird species already present, an observation that fits well with the predictions of a model for the assembly of a competitive Lotka–Volterra community (Gamarra et al., 2005).

At the second stage of the invasion process, when the nonnative species has already established, the impacts of the introduced species on the native competitors seem to increase with increasing intensity of competition. For example, among pairs of congeneric bird species introduced to Hawaii, pairs in which both species persisted had a significantly higher relative difference in beak length than pairs of species in which one or both species went extinct (Moulton, 1985). Contrarily, Ricciardi and Atkinson (2004) and Strauss et al. (2006b) found that species less related to the native community had higher impacts. However, their measures of impact summarized effects on various aspects of the native community, not only those on competitors, such that their results do not fully apply to our problem.

At this second stage, a number of evolutionary and genetic effects can contribute to the impacts of the introduced species on the native species. If the two species are closely related, there is the potential for hybridization and introgression, which can lead to new opportunities for evolution but also to extinction of rare native populations (Rhymer and Simberloff, 1996). Introduced and native competitors also impose new selection regimes on each other which can lead to shifts in life histories and resource use that reduce the intensity of competition and thus facilitate coexistence (Crowder, 1984), or even a coevolutionary arms race in the exploitation of a limiting resource (Leger and Espeland, 2010). These evolutionary effects are strongly contingent on the species involved.

There are also genetic effects, however, that are a direct consequence of the reduction in native population size due to competition and should therefore be present in most cases: smaller populations are subject to inbreeding depression, they can maintain a lower amount of genetic diversity, and accumulate deleterious mutations more rapidly (Frankham, 1995; Lande, 1995; Frankham and Kingsolver, 2004). Additionally, fluctuations in population size or habitat fragmentation can change the genetic configuration of the native species. So far, there are only a few empirical studies that examine such genetic effects of introduced species (e.g. Krueger and May, 1991; Kim et al., 2003). In this study, we focus on the reduction in a native population's genetic diversity caused by a population decline after the invasion of a competitor. A reduction in genetic diversity can lower the native species' ability to respond to changes in the environment (Strauss et al., 2006a) and thus lead to a reduced growth rate (Lande and Shannon, 1996). This, in turn, leads to a further decline in population size, thus closing the feedback loop. Such a synergistic feedback between ecological and genetic effects can accelerate population extinction (Robert, 2011). We will call this the eco-genetic effect of the introduced species and quantify how its strength depends on the intensity of competition with the native species.

In summary, a high intensity of competition between introduced and native species appears to have contrary effects at the different stages of the invasion process: it makes establishment of the introduced species more difficult but also increases the extinction risk imposed by an already established nonnative species on the native species (see also MacDougall et al., 2009). This raises the question: What is the overall effect of competition intensity integrated across the entire invasion pathway? In this study, we consider a scenario in which an unlimited series of introduction events would sooner or later lead to the extinction of the native species. Our goal is to quantify for how long the native species can persist depending on the intensity of competition with the introduced species.

Stochastic models based on birth, death, and migration events at the individual level have increased our understanding of a wide range of processes in community ecology (Black and McKane, 2012), for example diversity patterns in dispersal-limited communities (Alonso et al., 2006). Stochasticity in the fates of individuals is particularly important for the dynamics of small introduced populations. Thus we use a stochastic modeling approach and compute the expectation and the variance of the time to the extinction of the native species, its persistence time. Very long persistence times in our model can be interpreted as signs for indefinite coexistence, since in these cases we would expect events like evolutionary divergence of niches to occur before the extinction of the native species (see Strauss et al., 2006a, and references therein).

We also study how the relationship between competition and persistence time is modulated by the rate at which nonnative individuals are introduced and the nonnative species' intrinsic ability to grow and reproduce in the new environment, i.e. we address questions such as: Is the effect of competition different for species that are introduced at a high vs. a low rate or does it depend on whether the introduced species or the native species has a higher fecundity. First, we consider these questions for a purely ecological model, which can be analyzed using theory on birth and death processes and a corresponding diffusion approximation. Then we transform this model into an eco-genetic model by adding a genetic dimension that allows us to quantify the feedback of reduced genetic diversity and adaptability on extinction risk in a variable environment and its dependence on competition strength. Lastly, we address a question of interest for invasive species management: How low does the introduction rate of a certain species need to be such that the native species is expected to persist for a certain threshold time?

2. Modeling

2.1. The ecological model

We represent the population dynamics of the native and the introduced species as a continuous-time stochastic model similar to the Moran model in population genetics (Moran, 1958). Consider a community consisting of a fixed number K of individuals, each of which belongs either to the native or to the introduced species.

The rate at which individuals die is proportional to the extent of competition experienced from conspecifics and members of the other species (as in Neuhauser and Pacala, 1999). The strength of interspecific competition relative to intraspecific competition is described by the non-negative competition coefficient α. Small values of α represent weak interspecific competition and high values intense interspecific competition. In principle, this parameter can be estimated from data on the overlap in resource use between the two species (May, 1975). The individual whose offspring replaces the dead individual is selected by randomly drawing one individual from the whole community, including the individual that just died. In this draw, native individuals have weight 1 and members of the introduced species weight w. Thus w can be understood as the fecundity of the introduced species relative to that of the native species in the sense that it is proportional to the per capita number of offspring in a large offspring pool from which the new individual is drawn.

Note that for the sake of simplicity we assume that the competition coefficient α is the same for both species. Thus we are using this parameter to describe the symmetric component of the competitive interaction. Nevertheless, an asymmetric situation can be generated by setting the fecundity parameter w to a value different from 1. For $w > 1$ the introduced species has a fecundity advantage, for $w < 1$ a disadvantage. Initially, all K individuals belong to the native species. From time 0 onwards, single individuals of a nonnative species are introduced at rate γ (on average γ times per time unit) and start competing with the native species. To bring the community back from $K+1$ to K individuals after an introduction event, one individual is drawn to die with weights proportional to the competition experienced.

The population dynamics of the native species can be formulated as a Markov process $N = (N(t))_{t\geq 0}$ with state space $\{0, 1, 2, \ldots, K\}$ which describes the number of native individuals currently in the community. Since in this model transitions are only possible between neighboring states, it belongs to the class of birth and death processes (Karlin and Taylor, 1975, pp. 131–150). The rate λ_n at which the number of native individuals increases by one is

$$\lambda_n = \underbrace{\frac{c(K-n,n)\cdot(K-n)}{K}}_{\substack{\text{rate at which}\\ \text{members of the introduced}\\ \text{species die}}} \cdot \underbrace{\frac{n}{(K-n)\cdot w + n}}_{\substack{\text{probability that a}\\ \text{native individual}\\ \text{gives birth}}} \tag{1}$$

for $n \in \{0, \ldots, K-1\}$, where $c(x, y) = x + \alpha y$ is the competition experienced by an individual when the population size of the species it belongs to is x and the size of the competing species is y. The constant of proportionality for the death rate is chosen to be $1/K$, such that in the absence of the introduced species native individuals die at rate 1. Then there are on average K death events per time unit and one time unit can be considered as one generation. For $n \in \{1, \ldots, K\}$, the rate μ_n at which the number of native individuals decreases by one if there are currently n native individuals is

$$\mu_n = \underbrace{\frac{c(n,K-n)\cdot n}{K}}_{\substack{\text{rate at which}\\ \text{native individuals die}}} \cdot \underbrace{\frac{(K-n)\cdot w}{(K-n)\cdot w + n}}_{\substack{\text{probability that an}\\ \text{introduced individual}\\ \text{gives birth}}} + \underbrace{\gamma}_{\substack{\text{introduction}\\ \text{rate}}} \cdot \underbrace{\frac{c(n,K-n+1)\cdot n}{c(n,K-n+1)\cdot n + c(K-n+1,n)\cdot(K-n+1)}}_{\substack{\text{probability that a native}\\ \text{individual dies}}}. \tag{2}$$

The assumption of a fixed community size is a good approximation for pairs of ecologically similar species for which interspecific competition is as strong as intraspecific competition for the resource which is limiting population size. This can happen, for example, if there is a fixed number of territories or nesting places that can be occupied by one individual from either species. The competition for other important resources can be less intense ($\alpha < 1$) or there can be interspecific interference ($\alpha > 1$). The robustness of our model results against violations of the constant community size assumption is explored in the supplementary material.

In a community of finite size, coexistence of native and introduced species is not possible in the long run. We assume that our model encompasses the whole range of the native species, such that a reintroduction of native individuals from outside is not possible. The introduced species, in contrast, can fail to establish and go extinct after an introduction event, but will then be reintroduced at a later time. Therefore, the only absorbing state of the model is 0, the state at which the whole community consists of introduced individuals and the native species is extinct. Note that in the absence of immigration, the native species would be able to persist for an infinite amount of time, since by assumption the total number of individuals in the community is constant.

In the symmetric case ($w = 1$), the rare species has an advantage over the more common species for $\alpha < 1$ because then $c(n, K-n) = n + \alpha(K-n) < K - n + \alpha n = c(K-n, n)$ if $n < K/2$. This leads to fluctuations of the system around a point (which we will call the coexistence point) in which each species has population size $K/2$. For asymmetric competition ($w \neq 1$), whether coexistence is possible on an intermediate time scale, depends on the values of α and w. Also the position of the coexistence point depends on these parameters.

Let T_n be the random time to extinction of the native species in a realization of the process that starts with n native individuals. Let τ_n and σ_n^2 denote the expected value and the variance of T_n. We will use the ecological model to compute the expected value τ_K and the variance σ_K^2 of the time to the extinction of the native species when it is starting with population size K, i.e. in the state in which the nonnative species is still absent.

2.2. The eco-genetic model

Now we extend the ecological model by including a genetic component. To keep the model tractable, we chose the simplest possible genetic scenario: We assume that each native individual is haploid and possesses one bi-allelic locus which determines the individual's response to some environmental factor, for example whether or not the individual is resistant to a certain parasite. At any point in time, one of the two alleles is favored and its carriers have fecundity 1, whereas other native individuals have fecundity $1 - s$. Thus s is a measure for the strength of selection. With probability u an offspring mutates to the respective other allele and at rate ϵ the environment and with it the currently favored allele changes.

We assume that before introductions start, the number of native individuals that carry the favored allele has reached a stationary distribution, i.e. it is in mutation–selection equilibrium (see Appendix B for a derivation of this stationary distribution). To be able to compare the results of the eco-genetic model to those of the ecological model with the same introduced species fecundity parameter w, we multiplied w in the eco-genetic model by the average fecundity w^* (see (B.3)) of native individuals under the stationary distribution. Thus in both models, w can be interpreted as the fecundity of the introduced species relative to the average fecundity of native individuals.

This model can be represented as a Markov process, where the state with n native individuals, m of which carry the currently favored allele, is denoted by (n, m). From this state, we can reach the states $(n+1, m)$, $(n+1, m+1)$, $(n, m+1)$, $(n, m-1)$, $(n-1, m-1)$, and $(n-1, m)$ through birth–death events,

which possibly involve a mutation in the first four cases. The two latter states can also be reached through introduction events. The transition rates are defined analogously to those in the ecological model, with the death rate proportional to the competition experienced and the probability of giving birth proportional to fecundity. The main difference to the ecological model is that now the native population is divided into two allelic classes between which individuals can switch by mutation.

As an example (see Appendix B for all other transition rates), the transition rate from state (n, m) to state $(n, m+1)$ is

$$\frac{c(n, K-n)\cdot(n-m)}{K} \cdot \frac{m\cdot(1-u)+(1-s)(n-m)\cdot u}{m+(1-s)(n-m)+w^*\cdot w\cdot(K-n)}. \tag{3}$$

This is the rate at which one of the $n-m$ native individuals carrying the disfavored allele dies multiplied by the probability that it is replaced by a native individual with the favored allele. This new individual can either be the offspring of one of the m individuals with a favored allele that did not mutate (probability $1-u$) or an offspring of one of the $n-m$ parents with the disfavored allele that mutated (probability u). The transition from (n, m) to $(n, n-m)$ represents a change of the environment and happens at rate ϵ.

Our goal here is to compute the expected time to extinction of the native species. Here we start in the state (K, m^*), where the introduced species is absent and m^* is the average number of native individuals that carry the favored allele under the stationary distribution rounded to the next integer.

3. Results

3.1. Ecological effect

Measured from the start of introductions when the native population has still size K, the persistence time of the native species has expectation

$$\tau_K = \mathbf{E}[T_K] = \sum_{l=1}^{K}\sum_{i=l}^{K}\frac{1}{\mu_i}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j} \tag{4}$$

and its variance is

$$\sigma_K^2 = \mathbf{Var}(T_K) = \sum_{l=1}^{K}\sum_{i=l}^{K}\eta_i\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j}, \tag{5}$$

where

$$\eta_i = \begin{cases} \dfrac{1}{\mu_i(\mu_i+\lambda_i)}\left[1+\mu_i\lambda_i\left(\tau_{i+1}-\tau_{i-1}\right)^2\right] & \text{if } 1\le i\le K-1 \\ \dfrac{1}{\mu_K^2} & \text{if } i=K. \end{cases} \tag{6}$$

The results (4) and (5) are derived by noticing that the time to the extinction of the native species when starting in a state $n\in\{1,\dots,K\}$ has the same distribution (denoted $\stackrel{d}{=}$) as the sum of two independent random variables:

$$T_n \stackrel{d}{=} S_n + T_{N'} \stackrel{d}{=} S_n + \begin{cases} T_{n+1} & \text{with probability } \dfrac{\lambda_n}{\lambda_n+\mu_n} \\ T_{n-1} & \text{with probability } \dfrac{\mu_n}{\lambda_n+\mu_n} \end{cases} \tag{7}$$

for $n\in\{1,\dots,K-1\}$ and $T_K \stackrel{d}{=} S_K + T_{K-1}$. Here, S_n is a random variable for the time until the native population size first changes from state n to a new state N', which in our model is either $n-1$ or $n+1$. S_n is exponentially distributed with parameter $\lambda_n+\mu_n$ for $n\in\{1,\dots,K-1\}$ and with parameter μ_K for $n=K$. Taking the expectation on both sides in (7) and using $T_0=0$ we obtain the following recursion for τ_n (see Karlin and Taylor, 1975, pp. 145–150 for similar problems and solutions):

$$\tau_n = \begin{cases} 0 & \text{if } n=0 \\ \dfrac{1}{\lambda_n+\mu_n}+\dfrac{\lambda_n}{\lambda_n+\mu_n}\tau_{n+1} & \\ \quad+\dfrac{\mu_n}{\lambda_n+\mu_n}\tau_{n-1} & \text{if } 1\le n\le K-1 \\ \dfrac{1}{\mu_K}+\tau_{K-1} & \text{if } n=K, \end{cases} \tag{8}$$

which can be solved for τ_K.

Using the law of total variance

$$\begin{aligned}\mathbf{Var}(T_{N'}) &= \mathbf{E}\left[\mathbf{Var}(T_{N'}|N')\right]+\mathbf{Var}\left(\mathbf{E}[T_{N'}|N']\right)\\ &= \mathbf{E}[\sigma_{N'}^2]+\mathbf{E}[\tau_{N'}^2]-\mathbf{E}[\tau_{N'}]^2,\end{aligned} \tag{9}$$

we obtain a similar recursion for the variance σ_n^2

$$= \begin{cases} 0 & \text{if } n=0 \\ \dfrac{1}{(\lambda_n+\mu_n)^2}+\dfrac{\lambda_n}{\lambda_n+\mu_n}\left(\sigma_{n+1}^2+\tau_{n+1}^2\right) & \\ +\dfrac{\mu_n}{\lambda_n+\mu_n}\left(\sigma_{n-1}^2+\tau_{n-1}^2\right) & \\ -\left(\dfrac{\lambda_n}{\lambda_n+\mu_n}\tau_{n+1}+\dfrac{\mu_n}{\lambda_n+\mu_n}\tau_{n-1}\right)^2 & \text{if } 1\le n\le K-1 \\ \dfrac{1}{\mu_K^2}+\sigma_{K-1}^2 & \text{if } n=K, \end{cases} \tag{10}$$

which can be solved analogously to (8) once the τ_n are known (see Appendix C for details of these derivations).

The result given by (4) reveals that with increasing strength of competition, the expected time to extinction τ_K decreases until interspecific and intraspecific competition are of similar strength ($\alpha\approx 1$) (Fig. 1(A)). Here, τ_K reaches a minimum. If the strength of interspecific competition is further increased, τ_K grows again. The minimizing competition coefficient is below one for low introduction rates, and above one for large introduction rates (Fig. 1(B)). For low introduction rates ($\gamma<1$), the minimum moves towards lower competition coefficients, i.e. weaker competition, if fecundities are unequal, no matter whether the introduced species has a higher ($w>1$) or a lower fecundity parameter ($w<1$) than the native species (Fig. 2(A)). As expected, τ_K decreases with increasing introduction rate and increasing fecundity advantage of the introduced species.

These patterns in τ_K are paralleled by a corresponding pattern in the variance of the expected time to extinction σ_K^2. The variance increases with increasing τ_K and thus also exhibits a minimum. To compare the distribution of extinction times to an exponential distribution where the expected value equals the standard deviation, we computed the ratio between the standard deviation and the expected value of the persistence time in our model. This ratio is close to one for parameter combinations that lead to a high persistence time and below one for parameter combinations where the extinction of the native species is relatively fast (Fig. 3).

The numerical evaluation of (4) is practical only for small community sizes. For moderate to large community sizes we derived an approximation for the expected time to extinction that is easier to compute and gives more insight into the dependence of persistence time on the parameters:

$$\tau_K \approx K\cdot\int_0^1\frac{1}{(1-\xi)^\gamma}\int_\xi^1\frac{(1-\eta)^{\gamma-1}}{\eta}\cdot e^{\beta[\xi(1-\xi)-\eta(1-\eta)]}\cdot e^{\delta(\xi-\eta)}d\eta d\xi, \tag{11}$$

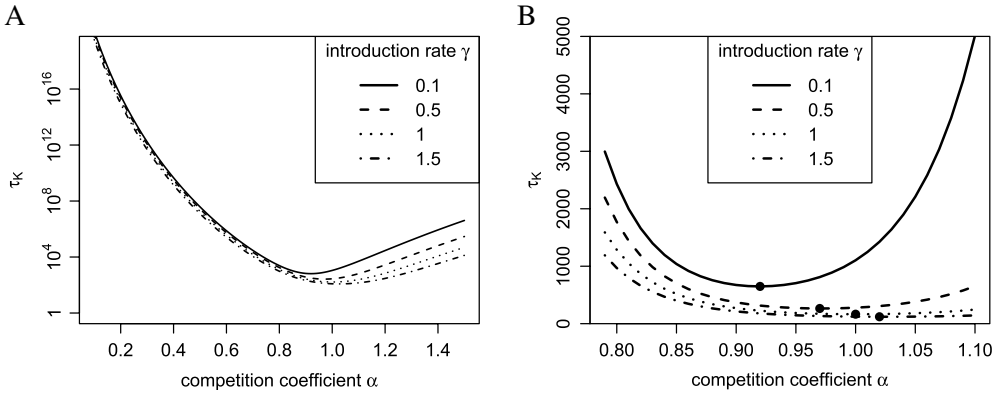

Fig. 1. The expected time to extinction τ_K for different values of the introduction rate γ as a function of the competition coefficient α. (B) magnifies the part of (A) around $\alpha = 1$. The minimum of each curve in B is indicated by a solid point ($K = 100$, $w = 1$).

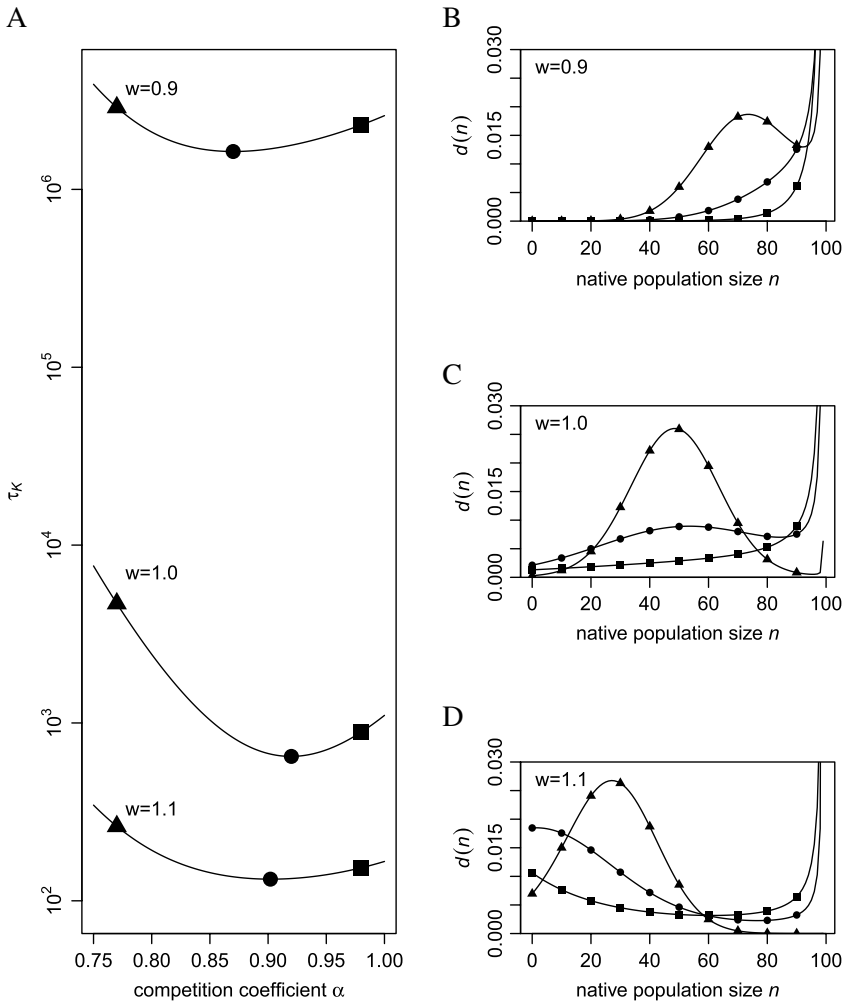

Fig. 2. The effect of changes in the introduced species fecundity parameter w on the expected time to the extinction of the native species and the position of the minimum (A) and on the quasi-stationary distribution ($d(1), \ldots, d(K)$) of the Markov process conditional on non-extinction of the native species (right column). The curves in the right column correspond to the competition coefficients marked with the respective symbol in (A) ($K = 100$, $\gamma = 0.1$).

where $\beta := (\alpha - 1) \cdot K$ is the rescaled advantage of being common and $\delta := (w - 1) \cdot K$ is the rescaled fecundity advantage of the introduced species.

The result (11) is based on a diffusion approximation of the birth and death process described by Eqs. (1) and (2). The process is rescaled such that the native population size is expressed as a fraction of the total community and time is sped up by a factor K:

$$X_K = (X_K(t))_{t \geq 0} = \left(\frac{N(K \cdot t)}{K} \right)_{t \geq 0}. \tag{12}$$

In the limit as K goes to infinity while β and δ are held constant, X_K converges in distribution to the diffusion process X with

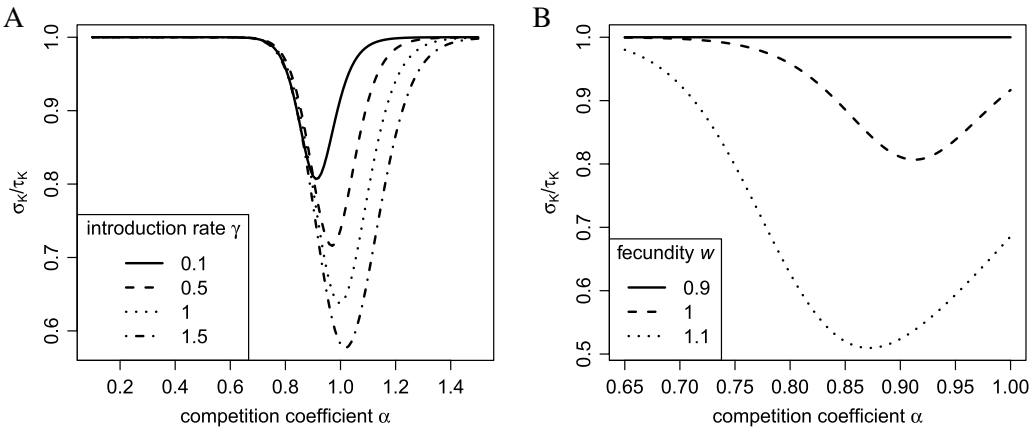

Fig. 3. The ratio of the standard deviation and the expected value of the time to extinction for (A) different introduction rates with $w = 1$ and (B) for different values of the introduced species fecundity with $\gamma = 0.1$ ($K = 100$).

infinitesimal generator (see Karlin and Taylor, 1981, p. 195, and the supplementary material for a derivation):

$$\begin{aligned} Lf(x) &:= \frac{d}{dt} E\left[f\left(X(t)\right)\middle|X(0) = x\right]\Big|_{t=0} \\ &= x(1-x)\frac{d^2}{dx^2}f(x) + \big(-\beta\,(1-2x)\,(1-x)\,x \\ &\quad - \gamma x - \delta x(1-x)\big)\frac{d}{dx}f(x). \end{aligned} \tag{13}$$

The expected time to extinction of the native species when its starting frequency is x is a solution $g(x)$ of the differential equation (Karlin and Taylor, 1981, p. 193)

$$Lg(x) = -1 \tag{14}$$

with boundary conditions $g(0) = 0$ and $|\lim_{x\nearrow 1} g'(x)| < \infty$, where $\lim_{x\nearrow 1} g'(x)$ denotes the left-sided limit at 1 (see supplementary material for details).

By numerically evaluating (11) in R (R Development Core Team, 2010) and using a golden section search algorithm (Heath, 2002) we computed $r(\gamma, \delta)$, the value of β that minimizes the right hand side of (11) for given values of γ and δ. After rescaling, we obtained for the competition coefficient $\hat{\alpha}$ which minimizes the expected time to the extinction of the native species.

$$\hat{\alpha} = 1 + \frac{r(\gamma, \delta)}{K}. \tag{15}$$

With increasing introduction rate γ, $r(\gamma, \delta)$ increases (Fig. 4(A)), becoming positive at $\gamma = 1$. The absolute value of $r(\gamma, \delta)$ increases with the differences in fecundity between the species (Fig. 4(B)).

3.2. Eco-genetic effect

As in (8), we recursively computed $\tau_{(n,m)}$, the expected time to the extinction of the native species when starting in state (n, m), by decomposing it according to what happens at the first jump. Doing this for all states gave rise to a system of $\frac{(K+1)(K+2)}{2} - 1$ linear equations, which we solved numerically in R for $\tau_{(K,m^*)}$, where m^* is the average initial number of native individuals with the favored allele.

Although we adjusted the fecundity of the introduced species to match the average fecundity of the native species, the expected time to extinction of the native species is lower under the eco-genetic model than under the ecological model described in Section 2.1 (Fig. 5). Over wide regions in parameter space, $\tau_{(K,m^*)}$ decreases with increasing selection strength s acting on the native species (Fig. 5(A)) and increases with increasing mutation probability u (Fig. 5(B)). Fig. 5(C) indicates that in the absence of environmental change, the expected time to extinction is similar to its counterpart in the ecological model. For a non-zero rate of change, the expected time to extinction is reduced, with a particularly strong reduction at small competition coefficients. In all cases, the minimizing competition coefficient (indicated by solid points in Fig. 5) is reduced if we take into account the eco-genetic feedback.

3.3. Critical introduction rate

We set a threshold persistence time τ_{crit} and determined the critical introduction rate γ_{crit}, such that $\tau_K > \tau_{\text{crit}}$ for all $\gamma < \gamma_{\text{crit}}$, using a bisection algorithm (Heath, 2002) for both the ecological and the eco-genetic model. To be able to compare the two models, we adjusted the fecundity parameter of the introduced species as above to match the average fecundity of the native species under mutation–selection equilibrium in a population of size K. As was the case with the expected time to extinction for fixed introduction rate, the critical introduction rate also reaches a minimum at an intermediate competition coefficient (Fig. 6). Not surprisingly, the critical introduction rate decreases with increasing fecundity advantage of the introduced species. Taking the eco-genetic effect into account, the critical introduction rate is lower than under the purely ecological model.

4. Simplifications and heuristics

To intuitively understand why competitors with intermediate interaction coefficients lead to the lowest persistence time of the native species, we simplify the state space to three possible states (Fig. 7(A)): the introduced species is absent and the whole community consists of the native species (N), both native and introduced species coexist ($N\&I$), or the native species is extinct and the whole community consists of the introduced species (I). If both species have the same fecundity ($w = 1$) and the introduction rate is small, native and introduced species exclude each other with approximately the same probability from the coexistence point and the rate $\phi(\alpha)$ at which this happens depends very little on γ. Let $\psi(\alpha, \gamma)$ be the rate at which the nonnative species establishes. Then, analogously to (8), we can formulate a recursion for the expected time to extinction of the native species:

$$\tau_N = \frac{1}{\psi(\alpha, \gamma)} + \tau_{N\&I} \tag{16}$$

and

$$\tau_{N\&I} = \frac{1}{2\phi(\alpha)} + \frac{1}{2}\tau_N. \tag{17}$$

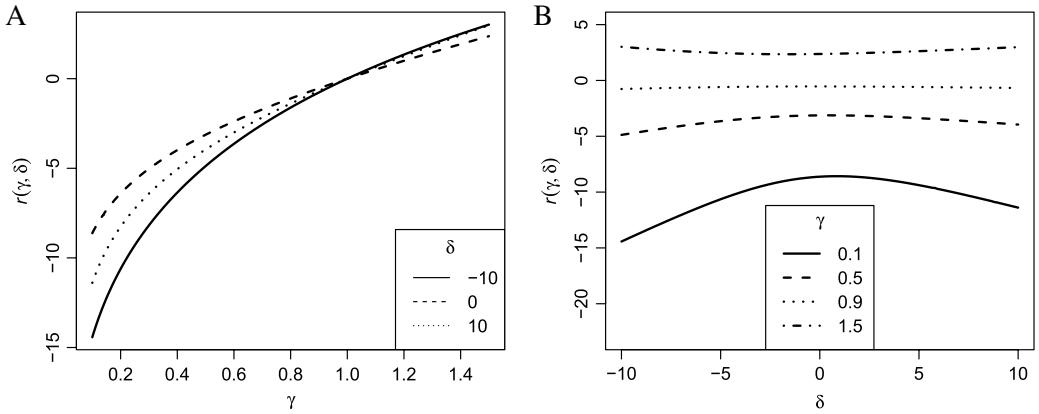

Fig. 4. The minimizing rescaled competition coefficient $r(\gamma, \delta)$ as a function of the introduction rate γ with fixed rescaled fecundity parameter δ (A) and as a function of δ with fixed γ (B).

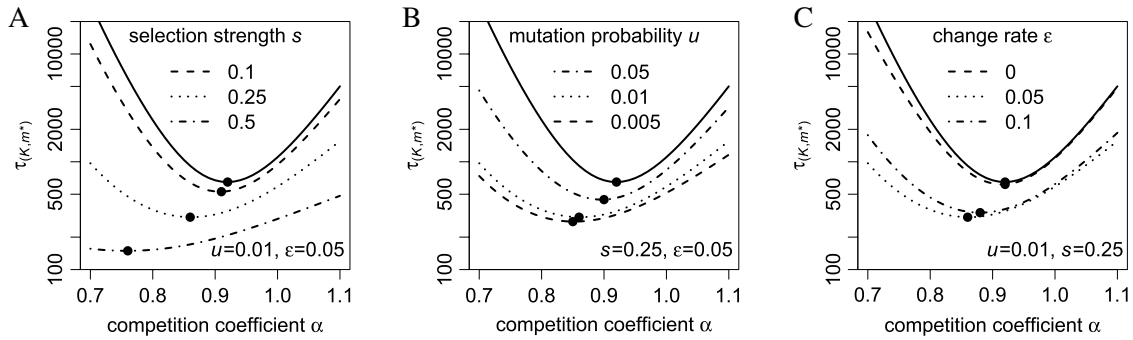

Fig. 5. The expected time to the extinction of the native species under the eco-genetic model as a function of the competition coefficient for different selection coefficients (A), different mutation probabilities (B), and different rates of environmental change (C). The solid line corresponds to the expected time under the ecological model (Eq. (4)). Minima are indicated by solid points ($K = 100$, $\gamma = 0.1$, $w = 1$).

As solution for the expected time to extinction of the native species when there is currently no introduced individuals we obtain

$$\tau_N = \frac{2}{\psi(\alpha, \gamma)} + \frac{1}{\phi(\alpha)}. \tag{18}$$

This is essentially twice the sum of the expected sojourn times in the states N and $N\&I$. What are these times? In the full model, $1/\psi(\alpha, \gamma)$ approximately corresponds to the expected time for the introduced species to reach population size $K/2$ starting from size 0. We will refer to this time as the establishment time of the introduced species. The term $1/(2\phi(\alpha))$ is the expected time for one of the two species to go extinct when they are currently coexisting with population size $K/2$ each. We will call this the exclusion time. Expressions for establishment time and exclusion time as functions of the model transition rates (1) and (2) were obtained by solving recursions similar to Eqs. (8) (see supplementary material).

The expected establishment time is an increasing function of the competition coefficient (dashed lines in Fig. 7(B) and (C)). The weaker the competition, the higher the advantage of an initially rare introduced population and the lower the expected time to reach $K/2$. The exclusion time (dotted lines) on the other hand is a decreasing function of the competition coefficient. The stronger the competition, the weaker the force is that drives the system back to the coexistence point, and the shorter the time is to the exclusion of one of the two species. Due to these two opposing effects the total time (solid lines) to the extinction of the native species, twice the sum of establishment time and exclusion time, can exhibit a minimum.

The higher the introduction rate, the smaller the influence of the competition coefficient is on establishment time, and the flatter

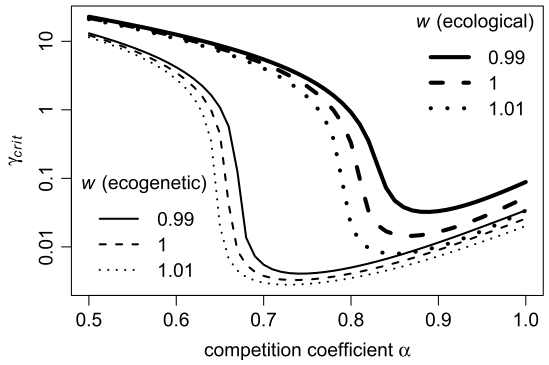

Fig. 6. The critical introduction rate (on a logarithmic scale) for different fecundity parameters w under the ecological and eco-genetic model as a function of the competition strength. In the eco-genetic case, w was multiplied by the average fecundity of native individuals under mutation–selection balance ($\tau_{\text{crit}} = 2000$, $\epsilon = 0.05$, $s = 0.25$, $u = 0.01$).

the curve of $1/(2\phi(\alpha))$ will be. This is the reason why the position of the minimum is shifted to higher values of α as the introduction rate γ increases (Fig. 7(C)). At $\gamma = 1$ the boundary where the introduced species is absent becomes an entrance boundary for the diffusion process; this means that the process can start at this boundary but can never return to it (Karlin and Taylor, 1981, p. 235). Thus, for higher introduction rates, establishment is no longer a limiting factor. To speed up the exclusion of the native species, the minimizing competition coefficient is above one in this region of parameter space.

In Figs. 2 and 4 we observed that in cases where the introduced and the native species differ in fecundity, the minimizing competition coefficient differs more from one than in the symmetric case.

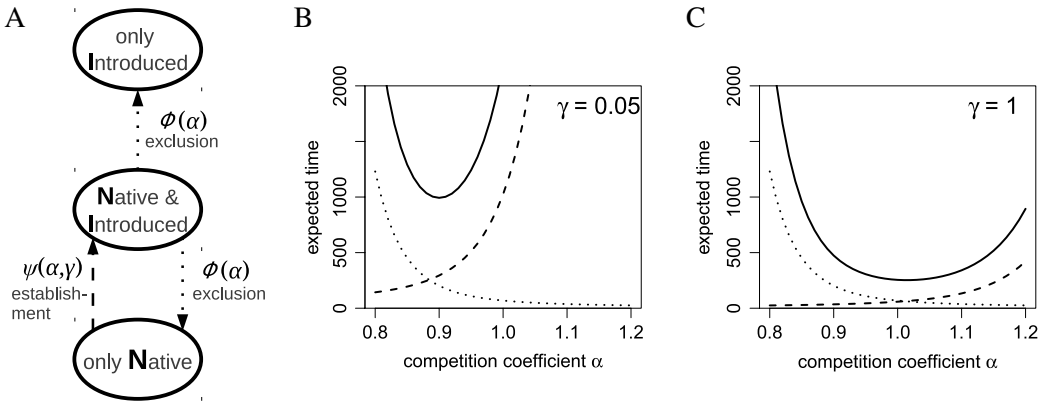

Fig. 7. A simple version of the model with only three states (A) illustrates the antagonistic effects of competition strength on establishment time (dashed lines) and exclusion time (dotted line) that lead to a minimum in the total time to native species extinction (solid line) which is at a smaller value of α in the case of a low introduction rate (B) and at a higher value for a high introduction rate (C). ($K = 100$, $w = 1$.)

An intuitive explanation for this phenomenon is that in asymmetric cases the dynamics are strongly shaped by the differences in fecundity and large changes in the competition coefficient are required to affect these dynamics, whereas in the symmetric case small changes in the competition coefficient can tip the balance.

To understand in which states the system spends most of its time, it is useful to examine the quasi-stationary distribution of the Markov process, the limiting distribution of population sizes of the native species given that it is not extinct yet. This is computed by eliminating the first row and first column, which belong to the absorbing state 0, from the rate matrix of the Markov process (see (A.1) in Box I). The left eigenvector of the remaining matrix associated with the eigenvalue with the largest real part is the quasi-stationary distribution (Darroch and Seneta, 1967). In the right column of Fig. 2 the quasi-stationary distribution is visualized for different parameter combinations. For competition coefficients on the right side of the minimum (indicated by squares), the introduced species is absent or has a low population size most of the time. For competition coefficients below the minimizing competition coefficient (triangles), the time the system spends around the coexistence point contributes most to the expected time to the extinction of the native species.

In regions of the parameter space where the quasi-stationary distribution is very stable, the ratio of the expected time to extinction and its standard deviation is almost one, suggesting that the time to extinction is approximately exponentially distributed. Moving towards parameter combinations that lead to a fast extinction of the native species, for example as the introduced species' fecundity is increasing (see Fig. 3(B)), the standard deviation is decreasing relative to the expectation, suggesting that the extinction of the native species becomes more deterministic.

The shift of the minimum in expected extinction time to smaller values of the competition coefficient in the model with genetic feedback can also be understood from the quasi-stationary distributions in Fig. 2. If competition is weak, the native population is most likely of intermediate size, and therefore a substantially lower amount of genetic diversity can be maintained within the population compared to a population that makes up the whole community. For high competition coefficients, the native population is most likely near its carrying capacity, if it is still present, which we condition on, and has therefore almost its full adaptability. Thus the additional extinction risk, or the reduction in expected time to extinction, would be larger for small competition coefficients than for large competition coefficients. This effect can be seen in Fig. 5, as the curves for the eco-genetic model diverge more from the curve belonging to the ecological model at lower than they do at higher competition coefficients.

5. Discussion

Our theoretical results indicate that the introduction of nonnative competitors raises the extinction risk of native species, both directly and indirectly, via a reduction in genetic diversity. The expected impact does not generally increase or decrease with competition intensity as one might expect, but there is an intermediate competition coefficient for which the expected time to extinction of the native species is minimized. This is the result of the opposing effects of competition strength on the establishment step of the invasion process and on the impact of an already established species. Introduced species that do not compete intensively with species from their new range can readily establish, but their ecological impacts are weak and it will take a long time for them to drive one of the native competitors to extinction. On the other hand, an introduced species that is competing very intensely with one of the native species has high potential ecological impacts once it has established. However, such a species may need a lot of introduction attempts before it can establish, because the native competitor can efficiently exclude it from the community.

Based on our results we expect competitors with intermediate interaction strength to also have the lowest critical introduction rate. Thus if one would set a management target to preserve an endangered native species for a certain time period, then the greatest introduction prevention efforts would be necessary for nonnative species that would have an intermediate intensity of competition with the native species.

With the help of our eco-genetic model, we quantified the feedback between ecological and genetic effects of the introduced species on the native competitor. This feedback is synergistic in the sense that ecological and genetic effects enhance each other: A reduction in population size causes a reduction in genetic diversity and this reduction in diversity can lead to further population decline in a changing environment. We found that this eco-genetic feedback is particularly strong for small intensities of competition between the introduced and the native species. This is because for high competition coefficients, the native species spends most of the time until its extinction in states with a high population size, whereas for intermediate and low competition coefficients the native species spends most of the time coexisting with the introduced species at an intermediate population size with a corresponding reduction in genetic diversity. Because the eco-genetic effect makes competitors with a relatively small competition intensity more dangerous for the native species, the minimizing competition coefficient is smaller compared to the ecological model. Similarly, also the critical introduction rate

is lower than in the purely ecological scenario. This highlights the importance of including eco-genetic feedbacks into risk assessment models. If we only bring the introduction rate down to the level required under the ecological model and there is an eco-genetic feedback, we will fail to keep the expected impacts below the prescribed threshold.

Eco-genetic effects are also a possible explanation for why there is relatively little evidence in the invasion biology literature for native species extinction due directly to an introduced competitor (Davis, 2003): Most endangered species are not threatened by a single stressor, but by combinations of them, for example habitat change and invasive species (Gurevitch and Padilla, 2004). The introduction of a competitor can weaken a native species' ability to respond to other stressors. There may have been many extinction events which were attributed to other factors and in which a significant contribution from an introduced competitor went unrecognized. Thus, when making predictions on native species population dynamics it can be important to consider the possibly synergistic interaction of species invasions with other drivers of global change (Didham et al., 2007).

Previous theoretical studies on the impacts of introduced species have built models designed to understand these impacts in specific systems (Byers and Goldwasser, 2001; Thomson, 2005). Here we contribute a building block towards the development of a theory that predicts impact from parameters of the introduced species, the native community and the introduction process. In this first stage of theory development our focus has been on simple models that are analytically tractable and give us insight into general phenomena. Of course, these models could be extended in many ways to incorporate more biological realism or to adjust them to specific biological systems, e.g. by including age or stage structure, which might in some cases influence the outcome of invasion and extinction dynamics (see e.g. Lande and Orzack, 1988).

Our finding, however, that the native species' persistence time is minimized at intermediate intensities of competition, is robust to a wide range of model modifications. To illustrate this robustness, we examined a model in which the assumption of a fixed community size was relaxed, a model in which competition affects fecundity instead of viability, and a model with an alternative formulation of the transition rates similar to the one used in neutral community theory (see e.g. Etienne and Alonso, 2007). All these models produced a minimum at intermediate competition intensities (see supplementary material for details of the analyses). Moreover, some of these modified models have the same diffusion approximation as the original model and thus behave very similarly, at least for large community sizes. Even a model in which we allow an immigration of native individuals from outside, exhibits a minimum in the expected time to the first extinction of the native species. However, possible measures of the long-term impact based on the stationary distribution of this process, like the proportion of time during which the native species is absent or the average native population size, have a monotonic relationship with competition intensity. This highlights that our results are most relevant for the short-term impacts of an introduced species on its native competitors.

In this study, we provide a model for the expected effect of an introduced species on one native competitor. Of course, native communities may consist of multiple competitors, as well as predators, mutualists, and parasites. To predict the impact of an introduced species on a whole community, our model could be combined with models for the other components of the community and interactions between them. Such detailed models have not been analyzed thus far. However, Fig. 2 in a study by Case (1990) shows that the probability that an introduced species can establish in a multi-species competitive community and replaces at least one native species is maximized for intermediate mean competition coefficients. Although Case (1990) does not address this point, this is one hint that our finding that intermediate levels of competition are most dangerous scales up to more complex communities.

In our model, single individuals of the nonnative species were introduced into the new habitat. What happens if multiple nonnative individuals are released at once? Drake et al. (2005) found that the product of introduction frequency and introduction size was a good predictor for the persistence of introduced *Daphnia* populations and that adding introduction frequency and size as single factors did not lead to significant improvements. However, if the nonnative population is subject to an Allee effect, i.e. positive density-dependence of population growth at low densities (Courchamp et al., 1999), the expected establishment success can strongly differ between a scenario with frequent introductions of one or a few individuals and one with rare introductions of many individuals (Drury et al., 2007). Disentangling the effects of propagule size and propagule frequency for such important scenarios is a promising field of future research.

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Appendix A. Rate matrix of the ecological model

The rate matrix of the ecological model is shown in Box I where the λ_i and μ_i are given by Eqs. (1) and (2).

Appendix B. Full specification of the eco-genetic model

Let (n, m) be the state with n native individuals, m of which carry the currently favored allele. Then the transition rates for $1 \le n \le K$ and $0 \le m \le n$ are

$$
(n,m) \to \begin{cases}
(n+1,m): & \dfrac{c(K-n,n)\cdot(K-n)}{K} \cdot \dfrac{(1-s)(n-m)(1-u)+mu}{\bar{w}_{n,m}} & \text{for } n < K \\
(n-1,m): & \dfrac{c(n,K-n)\cdot(n-m)}{K} \cdot \dfrac{w^*\cdot w\cdot(K-n)}{\bar{w}_{n,m}} + \gamma \cdot \dfrac{c(n,K-n+1)\cdot(n-m)}{c(n,K-n+1)\cdot n + c(K-n+1,n)\cdot(K-n+1)} & \text{for } n > m \\
(n,m+1): & \dfrac{c(n,K-n)\cdot(n-m)}{K} \cdot \dfrac{m\cdot(1-u)+(1-s)(n-m)u}{\bar{w}_{n,m}} & \text{for } n > m \\
(n,m-1): & \dfrac{c(n,K-n)\cdot m}{K} \cdot \dfrac{(1-s)(n-m)(1-u)+mu}{\bar{w}_{n,m}} & \text{for } m > 0 \\
(n-1,m-1): & \dfrac{c(n,K-n)\cdot m}{K} \cdot \dfrac{w^*\cdot w\cdot(K-n)}{\bar{w}_{n,m}} + \gamma \cdot \dfrac{c(n,K-n+1)\cdot m}{c(n,K-n+1)\cdot n + c(K-n+1,n)\cdot(K-n+1)} & \text{for } m > 0 \\
(n+1,m+1): & \dfrac{c(K-n,n)\cdot(K-n)}{K} \cdot \dfrac{m\cdot(1-u)+(1-s)(n-m)u}{\bar{w}_{n,m}} & \text{for } n < K \\
(n,n-m): & \epsilon, &
\end{cases}
\tag{B.1}
$$

$$\Lambda = \begin{pmatrix} -\lambda_0 & \lambda_0 & 0 & 0 & \cdots & 0 & 0 & 0 \\ \mu_1 & -(\lambda_1+\mu_1) & \lambda_1 & 0 & \cdots & 0 & 0 & 0 \\ 0 & \mu_2 & -(\lambda_2+\mu_2) & \lambda_2 & \cdots & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & \mu_{K-1} & -(\lambda_{K-1}+\mu_{K-1}) & \lambda_{K-1} \\ 0 & 0 & 0 & 0 & \cdots & 0 & \mu_K & -\mu_K \end{pmatrix}, \tag{A.1}$$

Box I.

where $\bar{w}_{n,m} = m + (1-s)(n-m) + w^* \cdot w \cdot (K-n)$ is the total fecundity in the community. To obtain w^*, the average fecundity of the native species under the eco-genetic model in the absence of the introduced species ($n = K$), we computed the stationary distribution $\mathbf{d} = (d(0), \ldots, d(K))$ of the birth and death process that describes the number of favored alleles in a native population of size K and has transition rates

$$m \to \begin{cases} m+1: & (K-m) \cdot \dfrac{m \cdot (1-u) + (1-s)(K-m)u}{m + (1-s)(K-m)} \\ & \text{for } m < K \\ m-1: & m \cdot \dfrac{mu + (1-s)(K-m)(1-u)}{m + (1-s)(K-m)} \\ & \text{for } m > 0 \\ K-m: & \epsilon \quad \text{for } 0 \le m \le K \end{cases} \tag{B.2}$$

and averaged

$$w^* = 1 - \frac{\sum_{m=0}^{K} d(m)(K-m) \cdot s}{K}. \tag{B.3}$$

Appendix C. Recursive solution for expectation and variance of the time to extinction in the ecological model

The middle equation in the recursion (8) can be rewritten as

$$\mu_n (\tau_n - \tau_{n-1}) = 1 + \lambda_n (\tau_{n+1} - \tau_n). \tag{C.1}$$

Define $z_n := \tau_n - \tau_{n-1}$, such that

$$z_n = \frac{\lambda_n}{\mu_n} z_{n+1} + \frac{1}{\mu_n}. \tag{C.2}$$

Solving this recursion for z with $z_K = \tau_K - \tau_{K-1} = \frac{1}{\mu_K}$ gives:

$$z_m = \sum_{i=m}^{K} \frac{1}{\mu_i} \prod_{j=m}^{i-1} \frac{\lambda_j}{\mu_j} \quad \text{and} \tag{C.3}$$

$$\tau_m = \sum_{l=1}^{m} z_l = \sum_{l=1}^{m} \sum_{i=l}^{K} \frac{1}{\mu_i} \prod_{j=l}^{i-1} \frac{\lambda_j}{\mu_j}. \tag{C.4}$$

With $m = K$ we obtain the expected persistence time (4).

Similarly, the middle equation in (10) can be written as

$$\xi_n = \eta_n + \frac{\lambda_n}{\mu_n} \xi_{n+1}, \tag{C.5}$$

where $\xi_n = \sigma_n^2 - \sigma_{n-1}^2$ and η_n is given by (6). As above, we can solve this recursion for ξ with $\xi_K = \eta_K$:

$$\xi_l = \sum_{i=l}^{K} \eta_i \prod_{j=l}^{i-1} \frac{\lambda_j}{\mu_j} \tag{C.6}$$

and finally obtain (5) by summing

$$\sigma_K^2 = \sum_{l=1}^{K} \xi_l. \tag{C.7}$$

Appendix D. Supplementary data

Supplementary material related to this article can be found online at http://dx.doi.org/10.1016/j.tpb.2012.11.003.

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Ecological and genetic effects of introduced species on their native competitors

Supplementary material

Diffusion approximation to the ecological model

We are seeking an approximation of equation (4) for large community sizes K. The strategy used here is to first rescale the Markov process defined by the transition rates (1) and (2) and approximate it by a diffusion process. Then we compute the expected time to native extinction in the diffusion process and finally prove that the exact recursive solution given by equation (4) converges to the expected time under the diffusion process as K goes to infinity.

Convergence in distribution $X_K \Rightarrow X$

Theorem 1. *As the community size K goes to infinity, the rescaled Markov process*

$$X_K = (X_K(t))_{t\geq 0} = \left(\frac{N(K\cdot t)}{K}\right)_{t\geq 0}. \tag{S.1}$$

with $\alpha = 1 + \frac{\beta}{K}$ and $w = 1 + \frac{\delta}{K}$, where β and δ are constants, converges in distribution to a diffusion process X with infinitesimal generator

$$Lf(x) = \frac{1}{2}b(x)\frac{d^2}{dx^2}f(x) + a(x)\frac{d}{dx}f(x)\,, \tag{S.2}$$

where

$$a(x) = -\beta(1-2x)(1-x)x - \delta x(1-x) - \gamma x \tag{S.3}$$

is the infinitesimal mean of the diffusion process and

$$b(x) = 2x(1-x) \tag{S.4}$$

is the infinitesimal variance.

To prove the theorem we need a few lemmata.

Lemma 2. *For $x \in \{0, \frac{1}{K}, \frac{2}{K}, \ldots, 1\}$ the generator of the Markov process X_K, defined as*

$$L_K f(x) := \frac{d}{dt}\mathbf{E}\big[f\big(X_K(t)\big)|X_K(0) = x\big]\big|_{t=0}\,, \tag{S.5}$$

where f is a bounded twice continuously differentiable function, converges to the generator of the diffusion process given by equation (S.2):

$$\lim_{K\to\infty} \max_{x\in\{\frac{0}{K},\frac{1}{K},\ldots,\frac{K}{K}\}} |L_K f(x) - Lf(x)| = 0\,. \tag{S.6}$$

Proof.

$$L_K f(x) = \left[f\left(x + \frac{1}{K}\right) - f(x)\right]\cdot K\cdot\lambda_{Kx} + \left[f\left(x - \frac{1}{K}\right) - f(x)\right]\cdot K\cdot\mu_{Kx} \tag{S.7}$$

$$= \left[f'(x) + \frac{1}{2}f''(x)\cdot\frac{1}{K} + \mathcal{O}\left(\frac{1}{K^2}\right)\right]\cdot\lambda_{Kx} + \left[-f'(x) + \frac{1}{2}f''(x)\cdot\frac{1}{K} + \mathcal{O}\left(\frac{1}{K^2}\right)\right]\cdot\mu_{Kx} \tag{S.8}$$

$$= f'(x)\cdot(\lambda_{Kx} - \mu_{Kx}) + \frac{1}{2}f''(x)\cdot\frac{\lambda_{Kx} + \mu_{Kx}}{K} + \mathcal{O}\left(\frac{1}{K^2}\right)\cdot(\lambda_{Kx} + \mu_{Kx})\,. \tag{S.9}$$

Substituting the scaled parameters into equations (1) and (2) and using $o_i(1)$ to denote terms which fulfill $\lim_{K\to\infty} o_i(1) = 0$ uniformly in $x \in [0,1]$, we obtain

$$\begin{aligned}\lambda_{Kx} - \mu_{Kx} =& K\cdot\frac{\left(1 + \frac{\beta}{K}x\right)(1-x)x - \left(1 + \frac{\beta}{K}(1-x)\right)x(1-x)\left(1 + \frac{\delta}{K}\right)}{1 + \frac{\delta}{K}(1-x)} \\ &- \gamma\cdot\frac{\left(1 + \frac{\beta}{K}(1-x)\right)x + o_1(1)}{1 + 2x(1-x)\frac{\beta}{K} + o_2(1)}\end{aligned} \tag{S.10}$$

$$= \frac{-\beta(1-2x)(1-x)x - \delta(1-x)x + o_3(1)}{1 + o_4(1)} - \gamma\cdot\frac{x + o_5(1)}{1 + o_6(1)} = \mathcal{O}(1)\,, \tag{S.11}$$

and

$$\lambda_{Kx} + \mu_{Kx} = K\cdot\frac{2(1-x)x + o_7(1)}{1 + o_4(1)} + \gamma\cdot\frac{x + o_5(1)}{1 + o_6(1)} = \mathcal{O}(K)\,. \tag{S.12}$$

Thus

$$L_K f(x) = x(1-x)\frac{d^2}{dx^2}f(x) + \big(-\beta(1-2x)(1-x)x - \gamma x - \delta x(1-x)\big)\frac{d}{dx}f(x) + o_8(1)\,. \tag{S.13}$$

All expressions are bounded uniformly in $\{\frac{0}{K}, \frac{1}{K}, \ldots, \frac{K}{K}\}$ and the error in equation (S.6) thus converges to zero uniformly in $\{\frac{0}{K}, \frac{1}{K}, \ldots, \frac{K}{K}\}$ as K goes to infinity. □

Lemma 3. *The sequence of Markov processes $(X_K)_{K\in\mathbb{N}}$ is tight.*

Proof. This is proven by using the compactness of the state space and the basic criterion for tightness and the Aldous condition from p. 34-35 in Joffe and Metivier (1986). □

Lemma 4. *The martingale problem for the generator L has at most one solution.*

Proof. The problem can be written as a stochastic differential equation with infinitesimal parameters extended beyond the interval $[0, 1]$ with $a(x) = a(0)$ and $b(x) = b(0)$ for $x < 0$ and $a(x) = a(1)$ and $b(x) = b(1)$ for $x > 1$ such that the process is now defined on $\mathbb{R}$ while leaving the behavior inside the interval $[0, 1]$ unchanged. Then we can apply the Yamada-Watanabe theorem (Theorem 26.10 in Klenke 2008) and conclude that the stochastic differential equation has a unique strong solution. This implies uniqueness in law (Theorem 26.18 in Klenke 2008), which in turn is equivalent to the statement that there exists at most one solution to the corresponding martingale problem (Rogers and Williams, 2000, p. 159). □

Proof of Theorem 1. The convergence in distribution follows from lemmata 2, 3, and 4 and Theorem 4.8.10 in Ethier and Kurtz (2005). □

Expected time to extinction under the diffusion process

Now we compute the expected time to the extinction of the native species under the diffusion process. Define

$$\sigma_l(X) := \inf\{t \geq 0 : X(t) \leq l\} \tag{S.14}$$

and let $\mathbf{E}_x$ denote the expectation for a process starting in x.

Theorem 5.

$$\mathbf{E}_x[\sigma_0(X)] = \int_0^x e^{\beta\xi(1-\xi)-\gamma\ln(1-\xi)+\delta\xi} \int_\xi^1 \frac{e^{-\beta\eta(1-\eta)+\gamma\ln(1-\eta)-\delta\eta}}{\eta(1-\eta)} d\eta\, d\xi\,. \tag{S.15}$$

for $x \in [0, 1]$.

Proof. An important tool for the analysis of diffusion processes is the scale function $S(x)$. The scale function transforms the state space such that the diffusion process becomes a martingale. Thus the scale function fulfills the differential equation $LS(x) = 0$ (Karlin and Taylor, 1981, p. 196). With $s(x) := \frac{d}{dx}S(x)$ the differential equation becomes

$$\frac{1}{2}b(x)\frac{d}{dx}s(x) + a(x)s(x) = 0\,. \tag{S.16}$$

Consequently,

$$s(x) = e^{\int_0^x -\frac{2a(y)}{b(y)}dy} = e^{\int_0^x -\left[-\beta(1-2y)-\frac{\gamma}{1-y}-\delta\right]dy} = e^{\beta x(1-x)-\gamma\ln(1-x)+\delta x}\,, \tag{S.17}$$

where the lower limit of the integral can be chosen arbitrarily and is here 0 for convenience (Karlin and Taylor, 1981, p. 194). With this we can write the infinitesimal generator as (Karlin and Taylor, 1981, p. 195):

$$Lf(x) = \frac{1}{2}s(x)b(x)\frac{d}{dx}\left[\frac{\frac{d}{dx}f(x)}{s(x)}\right]\,. \tag{S.18}$$

Let $g(x)$ be the solution of the differential equation (Karlin and Taylor, 1981, p. 193)

$$Lg(x) = -1 \tag{S.19}$$

with boundary conditions $g(0) = 0$ and $|\lim_{x \nearrow 1} g'(x)| < \infty$. Using the differential operator from (S.18), equation (S.19) is equivalent to

$$g(x) = \int_0^x e^{\beta\xi(1-\xi)-\gamma\ln(1-\xi)+\delta\xi} \left(C_1 - \int_{0.5}^{\xi} \frac{e^{-\beta\eta(1-\eta)+\gamma\ln(1-\eta)-\delta\eta}}{\eta(1-\eta)} d\eta \right) d\xi + C_2 \,. \tag{S.20}$$

The boundary conditions are fulfilled with $C_2 = 0$ and

$$C_1 = \int_{0.5}^{1} \frac{e^{-\beta\eta(1-\eta)+\gamma\ln(1-\eta)-\delta\eta}}{\eta(1-\eta)} d\eta \,. \tag{S.21}$$

With this

$$g(x) = \int_0^x e^{\beta\xi(1-\xi)-\gamma\ln(1-\xi)+\delta\xi} \int_{\xi}^{1} \frac{e^{-\beta\eta(1-\eta)+\gamma\ln(1-\eta)-\delta\eta}}{\eta(1-\eta)} d\eta \, d\xi \,. \tag{S.22}$$

Since $g(x) \leq g(1) < \infty$ for all $x \in [0,1]$, g is a bounded, twice continuously differentiable function. Then, since $(X(t))_{t\geq 0}$ solves the martingale problem for the generator L

$$g(X(t)) - \int_0^t Lg(X(s))ds = g(X(t)) + t \tag{S.23}$$

with $t \in [0,\infty)$ is a martingale. Application of the optional stopping theorem (see Ethier and Kurtz, 2005, p. 421, for a similar application) with $\tau > 0$ gives

$$\mathbf{E}_x \left[g\left(X(\sigma_0(X) \wedge \tau) \right) \right] + \mathbf{E}_x \left[\sigma_0(X) \wedge \tau \right] = g(x) \,. \tag{S.24}$$

Letting τ go to infinity, we obtain by the dominated and monotone convergence theorems

$$g(x) = \mathbf{E}_x \left[\sigma_0(X) \right] \,. \tag{S.25}$$

□

Convergence of extinction times

Because the first time to reach the boundary 0 is not a continuous functional of the process, we cannot conclude from the weak convergence of the rescaled birth and death process to the diffusion process that also the expected time to extinction converges to that under the diffusion process. Some more work is required to prove

Theorem 6.

$$\lim_{K\to\infty} \mathbf{E}_1[\sigma_0(X_K)] = \mathbf{E}_1[\sigma_0(X)] \,. \tag{S.26}$$

We start with proving the following lemmata:

Lemma 7. *For all* $j \in \{1, \ldots, K-1\}$

$$\frac{\mu_j}{\lambda_j} = \frac{1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)}{1 + \frac{\beta}{K}\frac{j}{K}} \cdot \left[1 + \frac{\delta}{K} + \frac{\gamma}{K-j} + \mathcal{O}\left(\frac{1}{K}\right) \right] \,. \tag{S.27}$$

Proof. Substituting the scaled parameters given in theorem 1 into equations (1) and (2), we find that

$$\lambda_j = \frac{\left(K + \frac{\beta}{K} \cdot j\right) \cdot (K - j) \cdot j}{K \cdot \left[K + \frac{\delta}{K} \cdot (K - j)\right]} \tag{S.28}$$

and

$$\begin{aligned} \mu_j =& \frac{\left[K + \frac{\beta}{K} \cdot (K - j)\right] \cdot j \cdot (K - j)\left(1 + \frac{\delta}{K}\right)}{K \cdot \left[K + \frac{\delta}{K} \cdot (K - j)\right]} \\ &+ \gamma \cdot \frac{\left[K + 1 + \frac{\beta}{K} \cdot (K - j + 1)\right] \cdot j}{\left[K + 1 + \frac{\beta}{K} \cdot (K - j + 1)\right] \cdot j + \left[K + 1 + \frac{\beta}{K} \cdot j\right] \cdot (K - j + 1)} . \end{aligned} \tag{S.29}$$

From equations S.28 and S.29 it follows that

$$\frac{\mu_j}{\lambda_j} = \frac{\left[1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)\right]\left(1 + \frac{\delta}{K}\right)}{1 + \frac{\beta}{K}\frac{j}{K}} + \frac{\gamma}{K - j} \frac{\left[1 + \frac{1}{K} + \frac{\beta}{K}\left(1 - \frac{j}{K} + \frac{1}{K}\right)\right]\left[1 + \frac{\delta}{K}\left(1 - \frac{j}{K}\right)\right]}{\left[\left(1 + \frac{1}{K}\right)^2 + 2\frac{\beta}{K}\left(1 - \frac{j}{K} + \frac{1}{K}\right)\frac{j}{K}\right]\left(1 + \frac{\beta}{K}\frac{j}{K}\right)} \tag{S.30}$$

$$= \frac{\left[1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)\right]\left(1 + \frac{\delta}{K}\right)}{1 + \frac{\beta}{K}\frac{j}{K}} + \frac{\gamma}{K - j} \frac{1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)}{1 + \frac{\beta}{K}\frac{j}{K}} \cdot \left(1 + \mathcal{O}\left(\frac{1}{K}\right)\right) . \tag{S.31}$$

After factorization one obtains (S.27). □

Lemma 8.

$$\prod_{j=l}^{i-1} \frac{\lambda_j}{\mu_j} < e^{2|\beta|+|\delta|} \cdot \frac{\left(1 - \frac{i}{K}\right)^\gamma}{\left(1 - \frac{l}{K}\right)^\gamma} \cdot e^{\mathcal{O}(1)} < e^{2|\beta|+|\delta|} \cdot e^{\mathcal{O}(1)} =: E \tag{S.32}$$

for all $l, i \in \{1, \ldots, K\}$ *with* $l \leq i$.

Proof. Using lemma 7 we can rewrite

$$\prod_{j=l}^{i-1} \frac{\lambda_j}{\mu_j}$$

as

$$\exp\left[-\sum_{j=l}^{i-1} \ln\left(\frac{1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)}{1 + \frac{\beta}{K}\frac{j}{K}} \cdot \left[1 + \frac{\delta}{K} + \frac{\gamma}{K - j} + \mathcal{O}\left(\frac{1}{K}\right)\right]\right)\right] \tag{S.33}$$

$$= \exp\left[\underbrace{\sum_{j=l}^{i-1} \ln\left(1 + \frac{\beta}{K}\frac{j}{K}\right) - \ln\left(1 + \frac{\beta}{K}\left(1 - \frac{j}{K}\right)\right) - \ln\left(1 + \frac{\delta}{K} + \frac{\gamma}{K - j} + \mathcal{O}\left(\frac{1}{K}\right)\right)}_{F}\right] . \tag{S.34}$$

Since $\ln(1 + x) = x + \mathcal{O}(x^2)$ for x close to zero and $-\ln(1 + x) \leq -x + x^2$ for all $x \in (-0.5, \infty)$

$$F \leq \sum_{j=l}^{i-1} \left[\frac{\beta}{K}\frac{j}{K} - \frac{\beta}{K}\left(1 - \frac{j}{K}\right) - \frac{\delta}{K} - \frac{\gamma}{K - j} + \frac{1}{(K - j)^2} + \mathcal{O}\left(\frac{1}{K}\right)\right] . \tag{S.35}$$

and

$$\sum_{j=l}^{i-1} \frac{1}{(K-j)^2} < \sum_{j=1}^{\infty} \frac{1}{j^2} = \frac{\pi^2}{6} = \mathcal{O}(1) \ . \tag{S.36}$$

With this

$$F \leq \sum_{j=l}^{i-1} \left[\frac{\beta}{K}\frac{j}{K} - \frac{\beta}{K}\left(1 - \frac{j}{K}\right) - \frac{\delta}{K} - \frac{\gamma}{K-j} \right] + \mathcal{O}(1) \tag{S.37}$$

$$\leq 2|\beta| + |\delta| - \gamma \ln\left(\frac{K-l}{K-i}\right) + \mathcal{O}(1) \tag{S.38}$$

and inequality (S.32) follows from this. □

Lemma 9. *There exists a function $r(\psi)$ such that*

$$\lim_{K\to\infty} \mathbf{E}_\psi[\sigma_0(X_K)] \leq r(\psi) \tag{S.39}$$

and

$$\lim_{\psi\to 0} r(\psi) = 0 \, . \tag{S.40}$$

Proof.

$$\mathbf{E}_\psi[\sigma_0(X_K)] \leq \frac{\tau_{\lceil K\psi\rceil}}{K} = \frac{1}{K}\sum_{l=1}^{\lceil K\psi\rceil} z_l = \frac{1}{K}\sum_{l=1}^{m} z_l + \frac{z_{\lceil K\psi\rceil}}{K} \tag{S.41}$$

with $m = \lceil K\psi\rceil - 1$ and where $\lceil x\rceil$ denotes the smallest integer larger than or equal to x. τ_i is given by equation (C.4) and z_i by equation (C.3). We first consider the first summand:

$$\frac{1}{K}\sum_{l=1}^{m} z_l = \frac{1}{K}\sum_{l=1}^{m}\sum_{i=l}^{K}\frac{1}{\mu_i}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j} = \frac{1}{K}\sum_{i=1}^{K}\frac{1}{\mu_i}\sum_{l=1}^{\min(i,m)}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j} \tag{S.42}$$

$$= \frac{1}{K}\Bigg(\underbrace{\sum_{i=1}^{m}\frac{1}{\mu_i}\sum_{l=1}^{i}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j}}_{A} + \underbrace{\sum_{i=m+1}^{K-m}\frac{1}{\mu_i}\sum_{l=1}^{m}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j}}_{B} + \underbrace{\sum_{i=K-m+1}^{K-1}\frac{1}{\mu_i}\sum_{l=1}^{m}\prod_{j=l}^{i-1}\frac{\lambda_j}{\mu_j}}_{C} + \underbrace{\frac{1}{\mu_K}\sum_{l=1}^{m}\prod_{j=l}^{K-1}\frac{\lambda_j}{\mu_j}}_{D}\Bigg) \, . \tag{S.43}$$

Note that

$$\mu_i \geq K\cdot\frac{i}{K}\left(1-\frac{i}{K}\right)\frac{\left[1+\frac{\beta}{K}\left(1-\frac{i}{K}\right)\right]\left(1+\frac{\delta}{K}\right)}{1+\frac{\delta}{K}\left(1-\frac{i}{K}\right)} \geq K\cdot\frac{i}{K}\left(1-\frac{i}{K}\right)\underbrace{\frac{\left[1-\frac{|\beta|}{K}\right]\left(1-\frac{|\delta|}{K}\right)}{1+\frac{|\delta|}{K}}}_{=:H} \, . \tag{S.44}$$

With this

$$\frac{A}{K} \leq \frac{E}{K}\cdot\sum_{i=1}^{m}\frac{i}{\mu_i} \leq \frac{E}{HK}\sum_{i=1}^{m}\frac{1}{1-\frac{i}{K}} \leq \frac{E}{HK}\frac{m}{1-\frac{m}{K}} \leq \frac{E}{H}\frac{\psi}{1-\psi} \, , \tag{S.45}$$

and

$$\frac{B}{K} \leq \frac{m \cdot E}{HK} \sum_{i=m+1}^{K-m} \frac{K}{i(K-i)} \leq \frac{2m \cdot E}{HK} \cdot \sum_{i=m+1}^{K/2} \frac{K}{i \cdot \frac{K}{2}} = \frac{4m \cdot E}{HK} \ln\left(\frac{K}{2m}\right) \leq \frac{4\psi \cdot E}{H} \ln\left(\frac{1}{2\psi}\right), \quad \text{(S.46)}$$

and

$$\frac{C}{K} \leq \frac{E}{HK} \sum_{i=K-m+1}^{K-1} \frac{1}{i\left(1-\frac{i}{K}\right)} \sum_{l=1}^{m} \frac{\left(1-\frac{i}{K}\right)^{\gamma}}{\left(1-\frac{l}{K}\right)^{\gamma}} \leq \frac{E \cdot m}{HK \cdot (K-m) \cdot \left(1-\frac{m}{K}\right)^{\gamma}} \sum_{i=K-m+1}^{K-1} \left(1-\frac{i}{K}\right)^{\gamma-1}. \quad \text{(S.47)}$$

Let $f(x) = (1-x)^{\gamma-1}$. Then $f'(x) = (1-\gamma)(1-x)^{\gamma-2}$. $f'(x) < 0$ if $\gamma > 1$ and $f'(x) > 0$ if $\gamma < 1$ for all $x \in (0,1)$. If $f'(x) > 0$

$$\sum_{i=K-m}^{K-1} f\left(\frac{i}{K}\right) \quad \text{(S.48)}$$

is a lower sum of the integral

$$K \cdot \int_{1-\frac{m}{K}}^{1} f(x)dx \quad \text{(S.49)}$$

and if $f'(x) < 0$

$$\sum_{i=K-m+1}^{K} f\left(\frac{i}{K}\right) \quad \text{(S.50)}$$

is a lower sum. Thus, in both cases

$$\sum_{i=K-m+1}^{K-1} \left(1-\frac{i}{K}\right)^{\gamma-1} < K \int_{1-\frac{m}{K}}^{1} (1-x)^{\gamma-1}dx = \frac{K}{\gamma}\left(\frac{m}{K}\right)^{\gamma}. \quad \text{(S.51)}$$

Consequently

$$\frac{C}{K} \leq \frac{E \cdot m}{H \cdot (K-m) \cdot \left(1-\frac{m}{K}\right)^{\gamma}} \frac{1}{\gamma}\left(\frac{m}{K}\right)^{\gamma} \leq \frac{E \cdot (\psi)^{\gamma+1}}{H \cdot \gamma \cdot (1-\psi)^{\gamma+1}}. \quad \text{(S.52)}$$

Since

$$\mu_K = \gamma \cdot \frac{\left(K+1+\frac{\beta}{K}\right) \cdot K}{\left(K+1+\frac{\beta}{K}\right) \cdot K + (K+1+\beta)} > \frac{\gamma}{2}, \quad \text{(S.53)}$$

$$\frac{D}{K} \leq \frac{m \cdot E}{K \cdot \mu_K} \leq \frac{2E\psi}{\gamma}. \quad \text{(S.54)}$$

Turning to the second summand in equation (S.41) and using equation (C.3)

$$\frac{z_{\lceil K\psi \rceil}}{K} = \sum_{i=\lceil K\psi \rceil}^{K} \frac{1}{\mu_i} \prod_{j=\lceil K\psi \rceil}^{i-1} \frac{\lambda_j}{\mu_j} \quad \text{(S.55)}$$

$$\leq \frac{E}{H} \sum_{i=\lceil K\psi \rceil}^{K-1} \frac{1}{i \cdot (K-i)} + \frac{E}{\mu_K \cdot K} \leq \frac{E}{H} \cdot \frac{\ln(K - \lceil K\psi \rceil)}{\lceil K\psi \rceil} + \frac{2E}{\gamma \cdot K} \xrightarrow[K\to\infty]{} 0. \quad \text{(S.56)}$$

In conclusion

$$\lim_{K\to\infty} \mathbf{E}_\psi[\sigma_0(X_K)] \leq \frac{E}{H}\left(\frac{\psi}{1-\psi} + 4\psi\ln\left(\frac{1}{2\psi}\right) + \frac{\psi^{\gamma+1}}{\gamma\cdot(1-\psi)^{\gamma+1}}\right) + \frac{2E\psi}{\gamma} = r(\psi)\,. \tag{S.57}$$

To see that $\lim_{\psi\to 0} r(\psi) = 0$ note that

$$\lim_{\psi\to 0} \psi\cdot\ln\left(\frac{1}{2\psi}\right) = \lim_{\psi\to 0} \frac{\frac{d}{d\psi}\ln\left(\frac{1}{2\psi}\right)}{\frac{d}{d\psi}\frac{1}{\psi}} = \lim_{\psi\to 0} \frac{\frac{1}{\psi}}{\frac{1}{\psi^2}} = \lim_{\psi\to 0}\psi = 0\,. \tag{S.58}$$

□

Proof of Theorem 6.

$$\lim_{K\to\infty} \left|\mathbf{E}_1\left[\sigma_0(X_K)\right] - \mathbf{E}_1\left[\sigma_0(X)\right]\right| \tag{S.59}$$

$$= \lim_{K\to\infty} \left|\mathbf{E}_1\left[\sigma_0(X_K)\right] - \mathbf{E}_1\left[\sigma_l(X_K)\right] + \mathbf{E}_1\left[\sigma_l(X_K)\right] - \mathbf{E}_1\left[\sigma_l(X)\right] + \mathbf{E}_1\left[\sigma_l(X)\right] - \mathbf{E}_1[\,\sigma_0(X)\,]\right| \tag{S.60}$$

$$\leq \lim_{K\to\infty}\left(\underbrace{\mathbf{E}_1\left[\sigma_0(X_K)\right] - \mathbf{E}_1\left[\sigma_l(X_K)\right]}_{=E_l[\sigma_0(X_K)]} + \left|\mathbf{E}_1\left[\sigma_l(X_K)\right] - \mathbf{E}_1\left[\sigma_l(X)\right]\right| + \left|\mathbf{E}_1\left[\sigma_l(X)\right] - \mathbf{E}_1\left[\sigma_0(X)\right]\right|\right) \tag{S.61}$$

$$\leq r(l) + \lim_{K\to\infty} \left|\mathbf{E}_1[\sigma_l(X_K)] - \mathbf{E}_1[\sigma_l(X)]\right| + \left|\mathbf{E}_1[\sigma_l(X)] - \mathbf{E}_1[\sigma_0(X)]\right|\,. \tag{S.62}$$

provided that the limit in the second summand exists. Following Kurtz (1981) p. 13-14 we argue that for a fixed path x, $\sigma_l(x)$ is decreasing in l and can therefore be discontinuous at only a countable number of values of l. Since the diffusion process is a rescaled Brownian motion, there is no point in the interval (0,1) that is exceptional compared to the other points and the points of discontinuity are placed according to some continuous probability distribution on the interval ($P(\text{discontinuity at } l) = 0\ \forall l$). For any discontinuity point thus the collection of paths for which the discontinuity is placed exactly at l is a null set. Since the union of countably many null sets is a null set itself, the probability that a random path has a discontinuity at l is zero. Therefore, the weak convergence of X_K to X implies the convergence of $\sigma_l(X_K)$ to $\sigma_l(X)$ according to the Continuous Mapping Theorem (Theorem 13.25 in Klenke 2008) such that the second limit exists and is equal to 0. See Lemma 3.3 in Chigansky and Klebaner (2012) for a more rigorous proof requiring a positive and increasing scale function $S(x)$, which we can achieve by choosing

$$S(x) = \int_0^x e^{+\beta y(1-y) - \gamma\ln(1-y) + \delta y} dy \tag{S.63}$$

and a positive quadratic variation $[X,X]_t$ for $t > 0$.

After the middle summand vanished, we take the limit $l \to 0$:

$$\lim_{K\to\infty} \left|\mathbf{E}_1\left[\sigma_0(X_K)\right] - \mathbf{E}_1\left[\sigma_0(X)\right]\right| \leq \lim_{l\to 0} r(l) + \lim_{l\to 0} \left|\mathbf{E}_1[\sigma_l(X)] - \mathbf{E}_1[\sigma_0(X)]\right| = 0 + 0 = 0\,. \tag{S.64}$$

The first limit follows from Lemma 9, the second limit from the boundedness of expected times under the diffusion process (Theorem 5) and the application of dominated convergence. □

Consequently, the expected time to extinction under the diffusion process is a valid approximation for the expected time under the rescaled birth and death process. Figure S.1 visualizes the quality of the approximation for different community sizes.

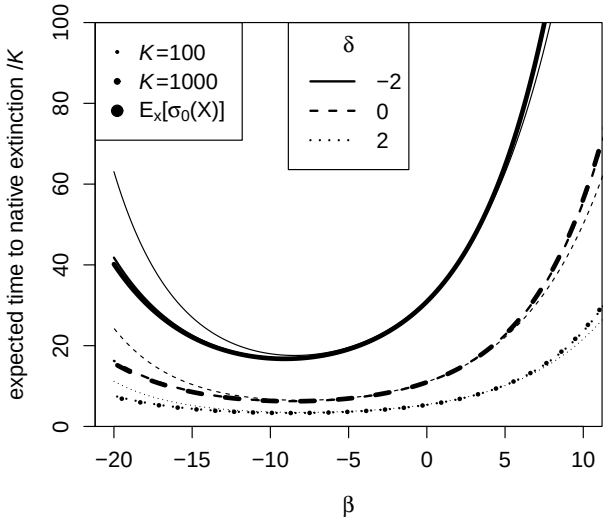

Fig. S.1: The exact solution for the expected time to the extinction of the native species (4) approaches the diffusion approximation (S.15) as the community size increases. ($\gamma = 0.1$).

Derivation of establishment time and exclusion time

The recursion for the expected time $\hat{\tau}_n$ to reach $K/2$ from a population size n above $K/2$ is:

$$\hat{\tau}_n = \begin{cases} 0 & \text{if } n = \frac{K}{2} \\ \frac{1}{\lambda_n+\mu_n} + \frac{\lambda_n}{\lambda_n+\mu_n}\hat{\tau}_{n+1} + \frac{\mu_n}{\lambda_n+\mu_n}\hat{\tau}_{n-1} & \text{if } \frac{K}{2}+1 \leq n \leq K-1 \\ \frac{1}{\mu_K} + \hat{\tau}_{K-1} & \text{if } n = K \end{cases} . \tag{S.65}$$

This can be solved in the same way as recursion (8). For the expected establishment time if the native species starts from its carrying capacity, we obtain

$$\hat{\tau}_K = \sum_{l=K/2+1}^{K} \sum_{i=l}^{K} \frac{1}{\mu_i} \prod_{j=l}^{i-1} \frac{\lambda_j}{\mu_j} . \tag{S.66}$$

We compute the exclusion time $\tilde{\tau}_{K/2}$ (the time to reach either 0 or K from $K/2$) under the assumption of symmetry and set $\gamma = 0$ in model equation 2. The appropriate recursion is:

$$\tilde{\tau}_n = \begin{cases} 0 & \text{if } n = 0 \\ \frac{1}{\lambda_n+\mu_n} + \frac{\lambda_n}{\lambda_n+\mu_n}\tilde{\tau}_{n+1} + \frac{\mu_n}{\lambda_n+\mu_n}\tilde{\tau}_{n-1} & \text{if } 1 \leq n \leq K-1 \\ 0 & \text{if } n = K \end{cases} . \tag{S.67}$$

Equations (C.1) and (C.2) remain valid but now we have $z_1 = \tilde{\tau}_1 - \tilde{\tau}_0 = \tilde{\tau}_1$ and $z_K = \tilde{\tau}_K - \tilde{\tau}_{K-1} = -\tilde{\tau}_{K-1}$, such that

$$z_m = -\tilde{\tau}_{K-1} \prod_{j=m}^{K-1} \frac{\lambda_j}{\mu_j} + \sum_{i=m}^{K-1} \frac{1}{\mu_i} \prod_{j=m}^{i-1} \frac{\lambda_j}{\mu_j} \quad \text{and} \tag{S.68}$$

$$\tilde{\tau}_1 = z_1 = -\tilde{\tau}_{K-1} \prod_{j=1}^{K-1} \frac{\lambda_j}{\mu_j} + \sum_{i=1}^{K-1} \frac{1}{\mu_i} \prod_{j=1}^{i-1} \frac{\lambda_j}{\mu_j} \,. \tag{S.69}$$

Because of symmetry

$$\tilde{\tau}_1 = \tilde{\tau}_{K-1} = \frac{\sum_{i=1}^{K-1} \frac{1}{\mu_i} \prod_{j=1}^{i-1} \frac{\lambda_j}{\mu_j}}{1 + \prod_{j=1}^{K-1} \frac{\lambda_j}{\mu_j}} = \frac{\sum_{i=1}^{K-1} \frac{1}{\mu_i} \prod_{j=1}^{i-1} \frac{\lambda_j}{\mu_j}}{2} \quad \text{and} \tag{S.70}$$

$$\tilde{\tau}_{K/2} = \sum_{l=1}^{K/2} \sum_{i=l}^{K-1} \frac{1}{\mu_i} \prod_{j=1}^{i-1} \frac{\lambda_j}{\mu_j} - \frac{1}{2} \left(\sum_{i=l}^{K-1} \frac{1}{\mu_i} \prod_{j=1}^{i-1} \frac{\lambda_j}{\mu_j} \right) \left(\sum_{l=1}^{K/2} \prod_{j=l}^{K-1} \frac{\lambda_j}{\mu_j} \right) \,. \tag{S.71}$$

Under the diffusion approximation, expressions for establishment time $\hat{g}$ and exclusion time $\tilde{g}$ can be found by using appropriate boundary conditions in equation (S.20). For the establishment time in the symmetric case, we need to use $\hat{g}(1/2) = 0$ and $|\lim_{x \nearrow 1} \hat{g}'(x)| < \infty$, such that

$$\hat{g}(1) = \int_{0.5}^{1} \frac{1}{(1-\xi)^\gamma} \int_{\xi}^{1} \frac{(1-\eta)^{\gamma-1}}{\eta} \cdot e^{\beta[\xi(1-\xi) - \eta(1-\eta)]} d\eta \; d\xi \,. \tag{S.72}$$

For the exclusion time, we need to use the boundary conditions $\tilde{g}(1) = \tilde{g}(0) = 0$ and we set $\gamma = 0$, which leads to

$$\tilde{g}(0.5) = \int_0^{1/2} \frac{e^{-\beta\eta(1-\eta)}}{\eta(1-\eta)} \int_0^{\eta} e^{\beta\xi(1-\xi)} d\xi \; d\eta \,. \tag{S.73}$$

Robustness of results

In this study, we have shown that according to our model for the competitive dynamics of a native and an introduced species, the expected time to the extinction of the native species is minimized for intermediate intensities of interspecific competition. In this section we explore to what extent this result is robust to model modifications.

The assumption of a fixed community size

In our original model, we assumed a fixed total community size. This assumption induces a strong coupling between the population dynamics of the two species. Here we consider a model in which the population dynamics are coupled only because individual death rates are proportional to the competition experienced, which is a function of both population sizes. To describe the system, we then need both the population size n_1 of the native species and the population size n_2 of the

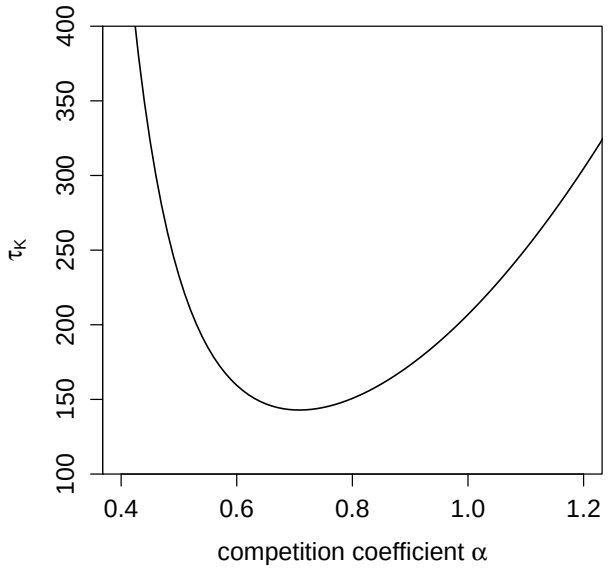

Fig. S.2: The expected time to the extinction of the native species τ_K in the model without a fixed community size with transition rates given by (S.74). ($K = 20, \gamma = 0.1, w_i = 1$)

introduced species. We thus define a Markov process with state space is $\{0, 1, 2, \dots\} \times \{0, 1, 2, \dots\}$ and transition rates

$$(n_1, n_2) \to \begin{cases} (n_1 + 1, n_2): & n_1 \\ (n_1 - 1, n_2): & \frac{c(n_1,n_2)}{K} \cdot n_1 \\ (n_1, n_2 + 1): & w_i n_2 + \gamma \\ (n_1, n_2 - 1): & \frac{c(n_2,n_1)}{K} \cdot n_2 \end{cases} . \tag{S.74}$$

Note that in this model, there is no strict upper limit to the population sizes. However, when the native species is alone its death rate exceeds its birth rate for population sizes larger than K and similarly for the introduced species when its size is larger than $w_i K$. When both species are together, the total community size fluctuates around a value which, if $\alpha < 1$, can exceed both of these quantities and which increases with decreasing competition intensity. The behavior is thus similar to the classical Lotka-Volterra competition model.

Using the same approach as for the eco-genetic model, we solved numerically for the expected time to the extinction of the native species when its initial size is K while the introduced species is initially absent. To obtain a finite transition matrix we needed to assume a maximum community size. We chose $4 \cdot \max(Kw_i, K)$ as excursions of the system to such a high community size are unlikely. For all parameter combinations we examined, this approximation to the expected time to extinction exhibits a minimum at intermediate competition intensities (see Fig. S.2 for an example). Due to the two-dimensional state space, these computations are only possible for small K.

Formulation of transition rates as in neutral community theory

Our model has some parallels to neutral community theory and related non-neutral models. However, most of these models have a different way of incorporating immigration. If a vacancy in the

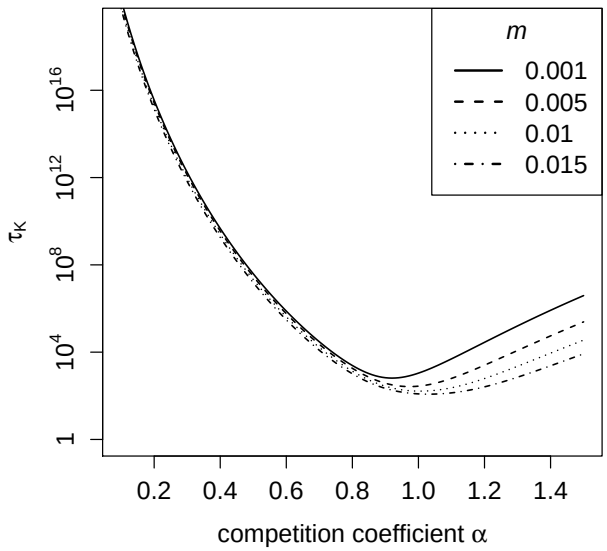

Fig. S.3: The expected time to the extinction of the native species τ_K in a model with transition rates analogous to those in neutral community theory (see equations S.75 and S.76). ($K = 100$, $w = 1$.)

local community is created, with probability $1-m$ it is filled by the offspring of an individual that is already in the community and with probability m by a migrant from the metacommunity (see e.g. Etienne and Alonso, 2007). In our case, immigrations from the metacommunity correspond to introduction events. In analogy to the transition rates in neutral community theory, we can reformulate our transition rates (1) and (2) as:

$$\lambda_n = \underbrace{\frac{c(K-n,n)\cdot(K-n)}{K}}_{\substack{\text{rate at which}\\ \text{members of the introduced}\\ \text{species die}}} \cdot \underbrace{(1-m)\cdot\frac{n}{(K-n)\cdot w+n}}_{\substack{\text{probability that a}\\ \text{native individual}\\ \text{gives birth}}} \tag{S.75}$$

and

$$\mu_n = \underbrace{\frac{c(n,K-n)\cdot n}{K}}_{\substack{\text{rate at which}\\ \text{native individuals die}}} \cdot \Big[\underbrace{(1-m)\cdot\frac{(K-n)\cdot w}{(K-n)\cdot w+n}}_{\substack{\text{probability that an}\\ \text{introduced individual}\\ \text{already in the community}\\ \text{gives birth}}} + \underbrace{m}_{\substack{\text{probability that the}\\ \text{spot is colonized by a}\\ \text{new introduced individual}}} \Big]. \tag{S.76}$$

We explored a range of parameter combinations and found that the expected time to the extinction of the native species under this model (Fig. S.3) behaves very similarly to that under the original model (compare Fig. 1). The minimum is preserved.

Moreover, if we assume the parameter scaling $\gamma = m/K$, then the Markov process converges to the diffusion process specified by the infinitesimal generator in (13). To see this note that for

$x \in [0,1]$

$$\lambda_{Kx} - \mu_{Kx} = K \cdot \left(1 - \frac{\gamma}{K}\right) \cdot \frac{\left(1 + \frac{\beta}{K}x\right)(1-x)x - \left(1 + \frac{\beta}{K}(1-x)\right)x(1-x)\left(1 + \frac{\delta}{K}\right)}{1 + \frac{\delta}{K}(1-x)} - \frac{\gamma}{K} \cdot K \left(1 + \frac{\beta}{K}(1-x)\right) x \tag{S.77}$$

$$= \frac{-\beta(1-2x)(1-x)x - \delta(1-x)x + o_3(1)}{1 + o_4(1)} \cdot (1 + o_5(1)) - \gamma \cdot x + o_6(1)\,, \tag{S.78}$$

and

$$\lambda_{Kx} + \mu_{Kx} = K \cdot \frac{2(1-x)x + o_7(1)}{1 + o_4(1)} \cdot (1 + o_5(1)) + \gamma \cdot x + o_6(1) \tag{S.79}$$

similarly to (S.11) and (S.12). Thus the convergence of generators (S.6) holds. The preconditions for the proofs of lemmata 3 and 4 are not affected by the modification of transition rates and thus by Theorem 1 the Markov process with transition rates (S.75) and (S.76) converges in distribution to the same diffusion process as the original Markov process.

Competition affecting fecundity instead of mortality

An alternative formulation of the transition rates (1) and (2) in which competition affects the rate at which individuals give birth rather than the death rate, is:

$$\lambda_n = \underbrace{\left(2 - \frac{c(n, K-n)}{K}\right) \cdot n}_{\substack{\text{rate at which} \\ \text{native individuals} \\ \text{give birth}}} \cdot \underbrace{\frac{K-n}{K}}_{\substack{\text{probability that an} \\ \text{introduced individual dies}}} \tag{S.80}$$

and

$$\mu_n = \underbrace{w_i \cdot \left(2 - \frac{c(K-n, n)}{K}\right) \cdot (K-n)}_{\substack{\text{rate at which} \\ \text{introduced individuals} \\ \text{give birth}}} \cdot \underbrace{\frac{n}{K}}_{\substack{\text{probability that a} \\ \text{native individual dies}}} + \gamma \cdot \underbrace{\frac{n}{K+1}}_{\substack{\text{probability that the} \\ \text{introduced individual} \\ \text{replaces a native one}}} . \tag{S.81}$$

The phenomenon that the expected time to native extinction is minimal at intermediate competition intensities is apparently not affected by this change in model formulation (Fig. S.4). Again, evaluating $\lambda_{Kx} - \mu_{Kx}$ and $\lambda_{Kx} + \mu_{Kx}$ reveals that this Markov process converges to the same diffusion process as the original Markov process.

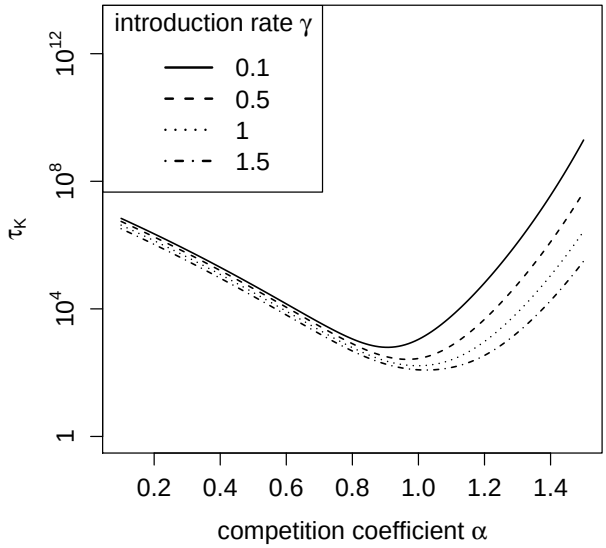

Fig. S.4: The expected time to the extinction of the native species τ_K in a model in which competition influences the birth rate rather than the death rate as specified by (S.80) and (S.81). ($K = 100$, $w = 1$.)

Immigration of native individuals

To allow for an immigration of native individuals at rate γ', we modify (1) by adding an immigration term analogous to the introduction term in (2):

$$\lambda_n = \underbrace{\frac{c(K-n,n)\cdot(K-n)}{K}}_{\substack{\text{rate at which}\\ \text{members of the introduced}\\ \text{species die}}} \cdot \underbrace{\frac{n}{(K-n)\cdot w+n}}_{\substack{\text{probability that a}\\ \text{native individual}\\ \text{gives birth}}}$$
$$+ \underbrace{\gamma'}_{\substack{\text{immigration}\\ \text{rate}}} \cdot \underbrace{\frac{c(K-n,n+1)\cdot(K-n)}{c(n+1,K-n)\cdot(n+1)+c(K-n,n+1)\cdot(K-n)}}_{\substack{\text{probability that an introduced}\\ \text{individual dies}}} . \qquad \text{(S.82)}$$

With this modification of transition rates, the state in which the native species is absent is no longer an absorbing state of the Markov process. Result (4) now gives us the expected time until the first extinction of the native species for realizations starting with K individuals of the native species. Evaluating the expected time to the first extinction for a range of parameter combinations suggests that this quantity is higher than the expected time to extinction in the original model, but still exhibits a minimum at intermediate competition coefficients (Fig. S.5).

One might also be interested in long-term measures of impact based on the stationary distribution of the Markov process, for example the proportion of time during which the native species is absent or the average population size of the native species. Unlike the expected time to the first extinction, these quantities exhibit monotonic relationships with competition intensity (Fig. S.6).

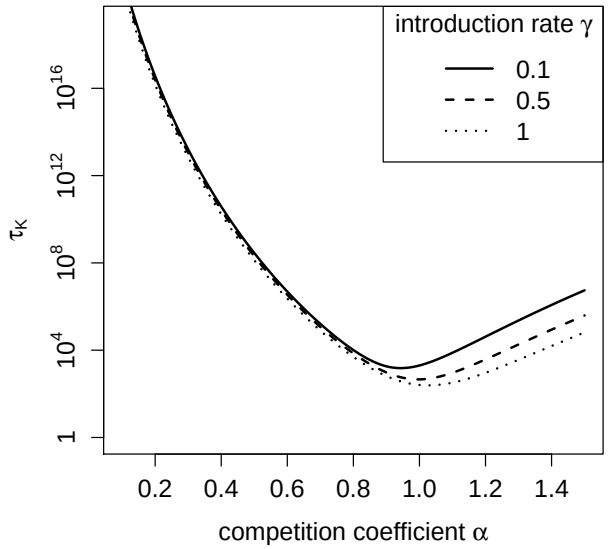

Fig. S.5: The expected time to the first extinction τ_K of the native species if we allow for the immigration of native individuals at rate $\gamma' = 0.5$ as specified in (S.82). ($K = 100$, $w = 1$.)

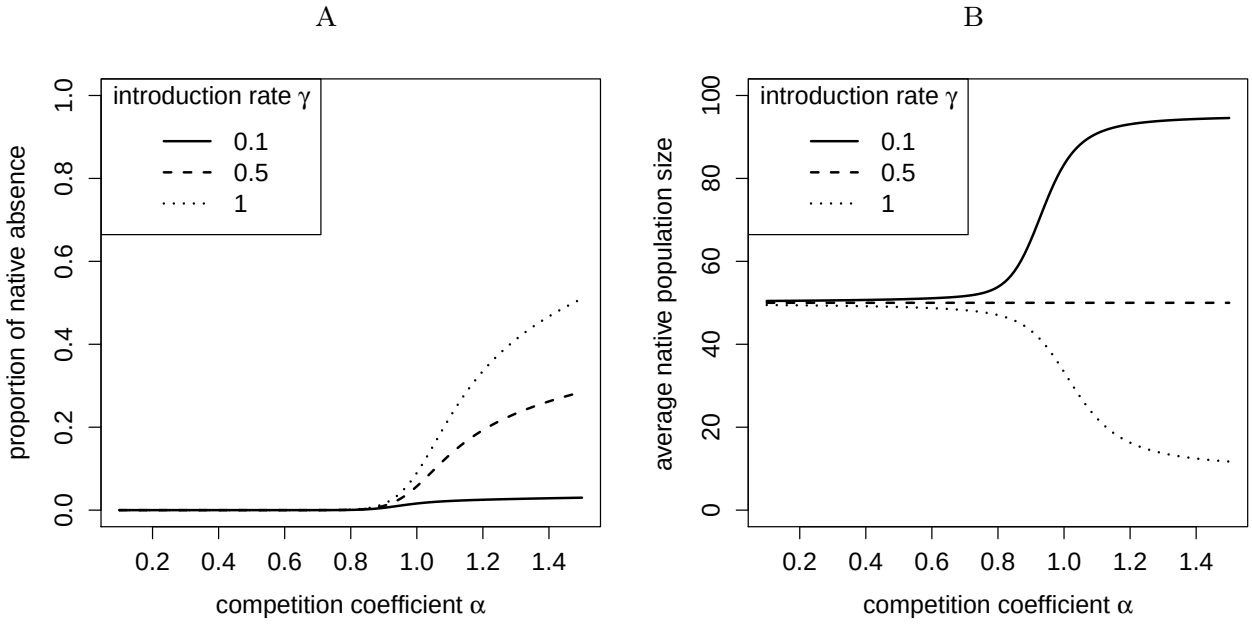

Fig. S.6: Long-term measures of impact in the model with immigration of native individuals: A) the proportion of time during which the native species is absent and B) the average population size of the native species under the stationary distribution. ($K = 100$, $w = 1$, $\gamma' = 0.5$.)

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Chapter 2

Decomposing propagule pressure: the effects of propagule size and propagule frequency on invasion success

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Oikos in press.

Abstract

Propagule pressure quantifies the inflow of individuals to a location and appears to be a key driver of invasion success. It is often defined as the average number of individuals introduced per time unit, or equivalently as the product of the average number of individuals introduced per introduction event (propagule size) and the frequency of introduction events (propagule frequency). Here we study how the influence of propagule size, frequency, and their product depends on the underlying ecological conditions. While previous studies have focused on introductions under environmental heterogeneity or a strong Allee effect, we examine a range of ecological scenarios that differ in the type of density dependence and in the sign of per-capita growth rate. Our results indicate that the relative influence of propagule size and frequency depends mainly on the sign of per-capita growth rate. Given a certain average number of individuals introduced per time unit, a high propagule frequency accelerates invasions under ecological scenarios with positive average per-capita growth rate throughout the invasion process ("easy" scenarios). If per-capita growth rate is negative throughout the invasion process ("difficult" scenarios) or if there is both an easy and a difficult stage ("mixed scenarios"), a high propagule size leads to a faster invasion than a high propagule frequency. To explain this finding, we argue that for a fixed value of the product of propagule size and frequency, an increase in propagule size leads to an increase in demographic variance, which promotes invasion success in difficult and mixed but not in easy scenarios. However, we also show that in many of these cases, the product of propagule size and frequency still correlates more strongly with invasion success than either of the single components. Finally, we illustrate our approach with empirical examples from the literature.

Keywords: alien species, Allee effect, competition, exotic species, invasive species, Markov process, non-native species, propagule pressure, stochasticity

Introduction

Propagule pressure and its components

There are few generalities in invasion biology. Hardly any ecological factor is consistently associated with invasion success of alien species over a wide range of taxonomic groups and studies. A notable exception thus declared "the new frontier in invasion ecology" (Richardson, 2004; Richardson & Pyšek, 2008) is propagule pressure, a concept used to quantify the inflow of individuals of a species into a new habitat. Propagule pressure can be defined as a composite measure with two components: propagule size, which is the average number of individuals introduced in a single introduction event, and propagule frequency, the average number of introduction events per time unit (see Lockwood *et al.*, 2005; Simberloff, 2009). In cases with a finite number of introduction events over a defined time period, many authors use the total number of introduction events—propagule number—instead of propagule frequency. To avoid confusion and because propagule number and frequency are proportional to each other, we use the term propagule frequency throughout the text.

The two-component definition of propagule pressure captures some aspects of the temporal distribution of introduced individuals. However, such detail is not always available in empirical data (see e.g. Cassey *et al.*, 2004). Furthermore, for many applications a single number is desired as measure for propagule pressure. Thus, it is common to define propagule pressure as the average number of individuals introduced per time unit. This measure corresponds to the product of propagule size and propagule frequency and will serve as a basis of comparison throughout this study.

Both propagule size and frequency as well as their product have been shown to positively correlate with invasion success (see e.g. Beirne, 1975; Veltman *et al.*, 1996; Duncan, 1997; Grevstad, 1999a; Forsyth & Duncan, 2001; Colautti, 2005; Simberloff, 2009). Less clear is the relative importance of the different components of propagule pressure for invasion success. Such an understanding is required, for example, for the design of release strategies in biological control (see e.g. Grevstad, 1999b; Shea & Possingham, 2000; Mailleret & Grognard, 2009), to identify the best proxies for propagule pressure in the context of risk analysis (e.g. by comparing different properties of pathways and vectors for the introduction of alien species; Hulme *et al.* 2008), or to assess the reliability of inferences from data sets that contain only a single component of propagule pressure.

In this study, we focus on accidental introductions that occur repeatedly, at random time points, and independently of the incipient population dynamics. We analyze how the role of propagule size and frequency depends on the underlying ecological scenario, as

characterized by the average growth rate of the alien population and its dependence on local population size, and in some cases also on time and spatial location. Specifically, our two main questions are: First, given a certain average number of individuals introduced per time unit, under what ecological scenarios does the alien species reach a given target population size faster with frequent but small introduction events, and under what scenarios is the target reached faster with fewer but large introduction events? To address this question, we fix the product of propagule size and frequency and vary propagule size and thus also propagule frequency. If there are strong differences in invasion success among introduction regimes with the same value of the product, then it may not be reasonable to quantify propagule pressure as the product of propagule size and frequency. Thus, our second question is: Under what ecological conditions do either propagule size or frequency correlate more strongly with invasion success than the product of the two?

Role of propagule size and frequency under different ecological conditions

Thus far, answers to these questions are limited to few specific ecological scenarios. A spatially or temporally varying environment is the best-understood example for an ecological scenario where propagule frequency should have a larger effect. Here, invasion success is expected to be highest when the introduced individuals are spread out as much as possible across space and time because such a strategy maximizes the probability that at least some of the introduced individuals encounter favorable conditions (Haccou & Iwasa, 1996; Haccou & Vatutin, 2003; Schreiber & Lloyd-Smith, 2009; Grevstad, 1999b). All of these studies assume that offspring production is density-independent.

The standard example for an ecological scenario in which propagule size plays the prime role is that of a strong demographic Allee effect. In populations that suffer from this effect, per-capita growth rate increases from negative values at small population sizes, becomes positive at some critical population size, and further increases with population size until competition becomes important (Taylor & Hastings, 2005). Populations with an initial size below the critical size have a high extinction probability. Therefore, it is apparent that in the case of a single introduction event, establishment probability increases with propagule size (see e.g. Dennis, 2002; Drake & Lodge, 2006).

Several studies have extended this result to the case of multiple introduction events subject to a constraint on the total number of individuals introduced. Grevstad (1999b) and Shea & Possingham (2000) investigated optimal release strategies for biological control organisms and found that for severe Allee effects a strategy with few large releases was preferred over a strategy with many small releases. Both studies assumed that each introduction event

occurs at a different location such that individuals arriving in different introduction events cannot interact to jointly overcome the Allee effect. If one allows for such interactions, as did Drury *et al.* (2007) and Mailleret & Lemesle (2009), then a high propagule size can facilitate establishment, even if single releases are not large enough to overcome the critical population size. This appears to result from the fact that larger groups of individuals do not decline as fast as small groups, and because a random accumulation of many introduced individuals is more likely if propagule size is large.

There may also be ecological scenarios where the temporal distribution of introduced individuals plays a minor role. Grevstad (1999b) suggested this to be the case if there is only demographic stochasticity, but no environmental stochasticity or density dependence. In a laboratory introduction experiment with *Daphnia*, the product of propagule size and frequency explained most of the variation in two of the three measures of invasion success considered (Drake *et al.*, 2005, see also the section Empirical examples below). It is not clear, however, which properties of the experimental conditions led to this result.

In summary, previous theoretical studies on the components of propagule pressure either assumed that there is no density dependence, i.e. per-capita demographic rates do not depend on population density, or that there is a strong demographic Allee effect. It is currently not known in general how properties of an ecological scenario such as the type of density dependence and the sign of per-capita growth rate determine the role of propagule size and frequency. For example, it is unclear whether the important role of propagule size under a strong Allee effect carries over to the case of a weak Allee effect, where per-capita growth rate is also positively density-dependent but is positive at all population sizes (Taylor & Hastings, 2005).

Approach and hypotheses

In this study, we therefore consider a range of ecological scenarios that differ in the type of density dependence (positive, negative, neutral) and in the sign of per-capita growth rate. If a scenario has a positive average per-capita growth rate at all population sizes below the target population size we call it an "easy" scenario. On the other hand, "difficult" scenarios are characterized by a negative average per-capita growth rate at all sizes below the target size. We also consider mixed scenarios where per-capita growth rate changes sign at an intermediate population size. In these cases, we say that the invasion process has a "difficult" and an "easy" stage.

To generate the ecological scenarios, we used two stochastic models: a single-population model and a competition model. Our models incorporate several sources of stochasticity.

Apart from the randomness associated with the introduction process, we assumed random timing of birth and death events to include demographic stochasticity, an important factor in the establishment of small alien populations (Fauvergue *et al.*, 2012). Since the effect of environmental heterogeneity on the relative importance of propagule size and frequency has already been well studied (see above), we decided to focus on introductions into constant environments in the main text. In Appendix 2.4, however, we explored how the results of our single-population model change in a varying environment.

We hypothesize that the effect of the temporal distribution of introduced individuals will be determined mostly by whether the underlying ecological scenario is easy or difficult. While under easy scenarios the population grows on average, under a difficult scenario population growth requires unusual events such as an extraordinarily large number of individuals being introduced during a short time interval. A measure for how frequent such unusual events are is the variance of the number of individuals introduced over some time interval, a quantity that may differ between introduction regimes with the same average number of individuals introduced per time unit. The larger propagule frequency is, the more introduction events happen on average, leading to a faster averaging-out of the number of individuals introduced. Consequently, given a fixed value of the product of propagule size and frequency, the associated variance increases with propagule size.

In Appendix 2.1, we provide a formal mathematical explanation for these arguments. We can intuitively understand them by drawing parallels with sports and games where an inferior team can only hope to win through chance events. Such a team should strive to increase the variance of the outcome, for example by playing a risky strategy. In a tournament, an inferior team should favor a mode with few matches (e.g. a knockout tournament system) to take advantage of rare bad days of superior teams. On the other hand, a superior team should favor a mode with many matches (e.g. an all-play-all tournament) to play out its advantage until the end of the competition.

Going back to alien species, we thus hypothesize that after controlling for the product of propagule size and frequency, a high propagule size (and thus low propagule frequency, hence similar to a tournament with few games for an inferior sports team) is advantageous under difficult ecological scenarios. Under easy scenarios, on the other hand, a high propagule frequency might be advantageous since a low demographic variance reduces the risk of sporadic phases of population decline (similar to a superior sports team preferring an all-play-all tournament; see also Drake *et al.*, 2005). After showing how our framework of easy and difficult scenarios helps to classify the results under our various model scenarios, we consider three empirical data sets from the literature to illustrate how it may be applied to

real-world introductions.

Modeling approach and ecological scenarios

Our models have two components: one for the introduction process and one for the local population dynamics of the alien species. While the introduction process is modeled in the same way for all ecological scenarios, the scenarios differ in their population dynamic component. We modeled both components as continuous-time processes, i.e. by specifying the rates at which different kinds of events happen (introduction events, birth events, death events). We also assumed that all demographic processes only depend on the current state of the population or community and not on its history, such that our overall model formally is a continuous-time Markov process.

The introduction process is characterized by the propagule size s and the propagule frequency f, i.e. introduction events with s individuals each happen at rate f. We assumed that introduction events happen sufficiently close to each other in space such that individuals remaining from earlier introduction events have the opportunity to interact with individuals introduced at later times.

As case examples in the main text of this study, we examine seven ecological scenarios with different population dynamics (see Fig. 2.1 for an overview of our ecological scenarios). We consider scenarios in which per-capita growth rate increases (scenarios A, B, and C), stays constant (scenario E), or decreases (scenario G) with increasing population size. In scenarios D and F, the type of density dependence changes with increasing population size.

The seven scenarios also vary in the sign of per-capita growth rate and thus occupy different positions on the spectrum between difficult and easy scenarios. In scenario A, per-capita growth rate is negative at all population sizes below the target size, so this is a difficult scenario. Analogously, scenarios C, E, and G are easy; and scenarios B, D, and F are mixed scenarios. Scenarios with negative per-capita growth rate and neutral or negative density dependence are biologically not very meaningful; thus we do not consider them here.

To fully specify a stochastic population model, average-per capita growth rates need to be combined with information on the distribution of outcomes. These distributions depend on the underlying biological mechanisms and for each of the scenarios in Fig. 2.1 several such mechanistic underpinnings would be possible. Here, we generated the scenarios using either a single-population model or a two-species competition model. In the following, we

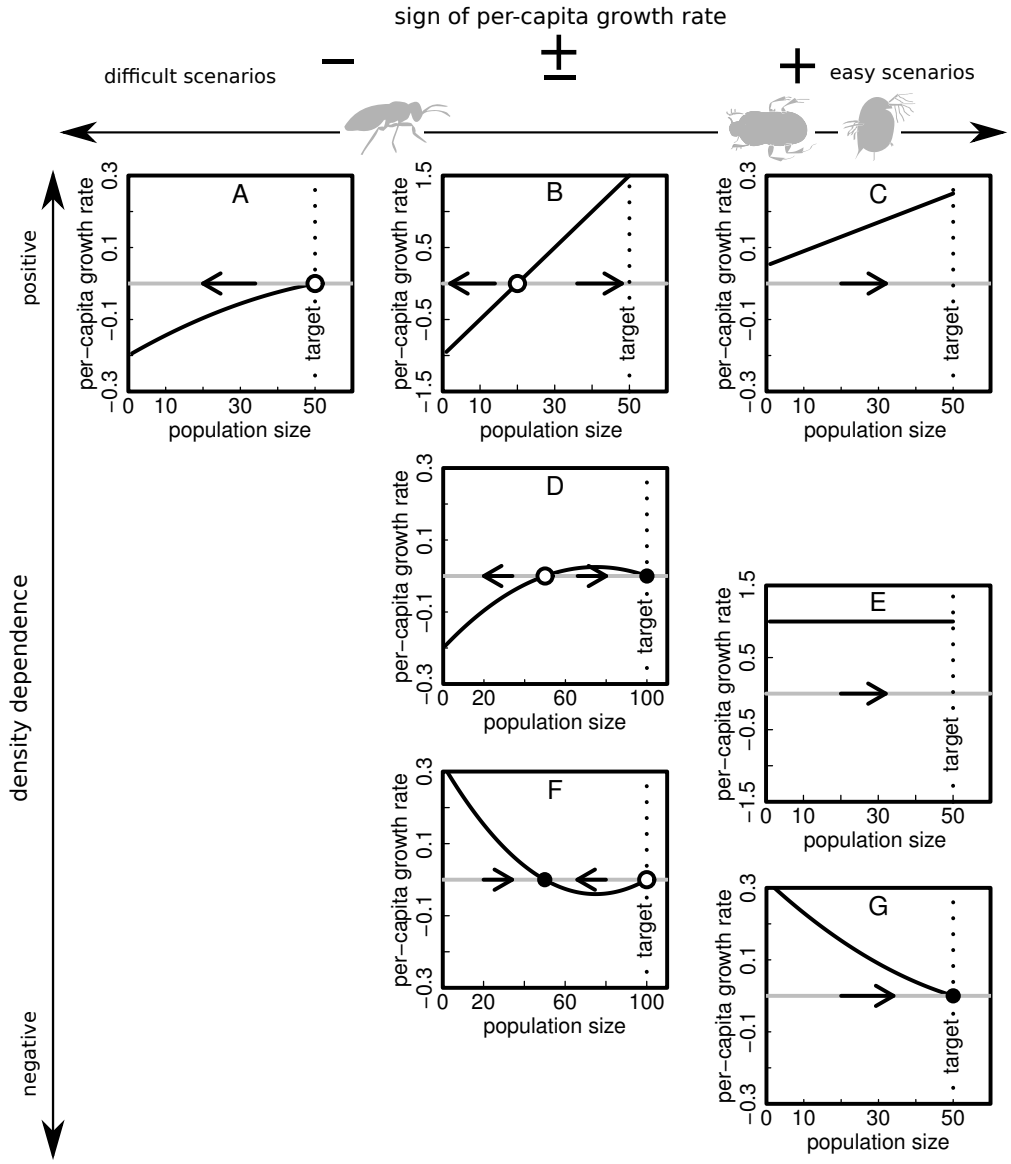

Figure 2.1: Overview of the ecological scenarios considered in this study. Ecological scenarios are characterized by how per-capita growth rate (solid black lines) depends on population size. They differ in the type of density dependence and in the sign of per-capita growth rate. Difficult ecological scenarios (A) have a negative per-capita growth rate; easy scenarios (C, E, G) have a positive per-capita growth rate; and mixed scenarios (B, D, F) have positive and negative per-capita growth rates, depending on population size. Grey lines serve as reference, indicating zero per-capita growth rate, and dotted vertical lines indicate the target population size. If a scenario contains points that would be stable or unstable equilibria in a deterministic system, we indicate them by solid or open circles, respectively. Arrows show the expected direction of population-size change in the different population-size ranges. Please note that the scaling of the y-axis differs among scenarios. Information on the underlying models and parameter values can be found in Table 2.1. The animal symbols refer to the suggested position of our empirical examples (parasitoid wasps, dung beetles, *Daphnia*; see section Empirical examples below) on the spectrum between difficult and easy ecological scenarios.

explain the key assumptions of these models and how we used them to create our ecological scenarios of interest. Detailed information on both models including all parameters and formulas for the transition rates are presented in Appendix 2.2. Table 2.1 summarizes the parameter values used to generate scenarios A-G.

Scenarios B, C, and E stem from a single-population model that focuses on the dynamics of the alien population while it is very small compared to its carrying capacity. In scenario B, the alien population experiences a strong demographic Allee effect, which may for example be caused by mate-finding problems in small populations. There is a threshold population size below which the population tends to decline and above which average per-capita growth rate is positive. Scenario C represents a weak Allee effect in which per-capita growth rate is reduced at small population sizes, but not negative. In scenario E, there is no Allee effect; the population grows exponentially.

To generate scenarios A, D, F, and G, we used a two-species competition model: the alien species is introduced into a habitat that is already occupied by an ecologically similar resident competitor. A version of this model in which only single individuals are introduced is described and analyzed in Wittmann *et al.* (2013a). Depending on the value of the competition coefficient, either the rare species or the more common species has an advantage, i.e. a positive per-capita growth rate.

In scenarios F and G, we have a rare-species advantage. This may occur if there is only partial overlap in the resources used by the two species, such that individuals belonging to the rare species enjoy an abundance of the food items that cannot be used by the other species. In these scenarios, a small alien population has a positive per-capita growth rate that declines with increasing population size as resources become limiting. Scenarios F and G only differ in their target population size. In scenario G, we are interested in the expected time until the alien population reaches half the community size, whereas in Scenario F we computed the expected time until the alien species takes over the whole community and drives the resident competitor to extinction. In scenario F, the community is expected to fluctuate for a long time around a point where both species coexist at intermediate population size.

In scenarios A and D, which again differ only in their target population size, the more common species has an advantage. This may occur if there is intraspecific cooperation, if each species modifies the habitat in a way that is detrimental to the other species, or if the two species compete for two resources and each species consumes more of the resource that is most limiting for the other species. A common-species advantage can also result from interspecific interference or aggression, because as long as the population is small, most of

the encounters of alien individuals are with members of the other species. As the population increases in size and that of the competitor decreases, the number of such harmful encounters decreases and per-capita growth rate becomes positive.

Analysis

To compare introduction regimes with different values of propagule size and frequency, we calculate the respective expected times until the alien species first reaches the target population size R, given that it is initially absent. Due to stochasticity in the model and the infinite sequence of introduction events, the alien species always reaches its target eventually.

Computing the expected time to reach the target

To compute the expected time until the alien species reaches the target population size R, we employed first-step analysis, a standard technique for the analysis of Markov processes (see e.g. Karlin & Taylor, 1975). This technique allowed us to compute our quantity of interest, the expected time to reach the target population size, without having to run simulations. The key idea is that we can write the expected time $\mathbf{E}[T_i]$ when starting at some initial population size i as

$$\mathbf{E}[T_i] = \mathbf{E}[\tau_i] + \mathbf{E}[T_{i'}] \,, \tag{2.1}$$

where $\mathbf{E}[\tau_i]$ is the expected time until the population size first changes from i to some other size i', and $\mathbf{E}[T_{i'}]$ is the remaining time. Due to the Markov property—the population dynamics in our model only depend on the current state of the population, not on its history—the remaining time is just the expected time to reach the target population size if we would restart the process with initial population size i'. As detailed in Appendix 2.2, taking into account all possible next states i' and writing down such an equation for all possible initial states i, leads to a system of linear equations with the expected times belonging to the different initial population sizes as unknowns. We used R (version 2.14.1, R Development Core Team, 2011) to numerically solve this system given the parameter settings of interest.

Comparison of expected times

For each of our ecological scenarios, we compared the expected times to reach the target population size among introduction regimes in two different ways. To address our first research question, we fixed the product of propagule size and frequency, i.e. the average number of individuals introduced per time unit, to some value Π. We then computed the expected times for all propagule sizes $1, 2, \ldots, 20$ and corresponding propagule frequencies $\Pi, \Pi/2, \ldots, \Pi/20$.

To answer our second question and find the single measure of propagule pressure that correlates most with invasion success, we calculated the expected time to reach the target population size on a grid of combinations of propagule size and frequency. We then computed Spearman's rank correlation coefficient between the expected times and each of propagule size, frequency, or their product. Since the expected time to reach the target population size decreases with propagule pressure, the observed correlation coefficients are between 0 and -1, where -1 corresponds to the strongest possible correlation.

Results

The results on the seven ecological scenarios that we selected as case examples are compiled in Fig. 2.2. Overall, the expected time to reach the target population size differed strongly between scenarios (see Table 2.2). To facilitate a comparison of results between scenarios, we thus divided all expected times for a particular scenario by the respective expected time belonging to a propagule size of 1.

Whether the expected time to reach the target size increased or decreased when we increased propagule size while keeping the product constant depended mostly on the sign of per-capita growth rate rather than on the type of density dependence. All easy scenarios exhibited an increase in expected times with increasing propagule size, independently of the type of density dependence (positive in scenario C, neutral in scenario E, and negative in scenario G). The opposite effect, a decrease in expected time with increasing propagule size, can be observed in the difficult scenario A. In the mixed scenarios B, D, and F, the effect of the difficult phase dominated. In Appendix 2.3, we examine in detail how the role of propagule size and frequency changes as we continuously vary the parameters of the single-population and the competition model, thereby generating a continuum of ecological scenarios. In accordance with the results on our example scenarios, the relative effect of propagule size and frequency changed at parameter values at the boundary between easy

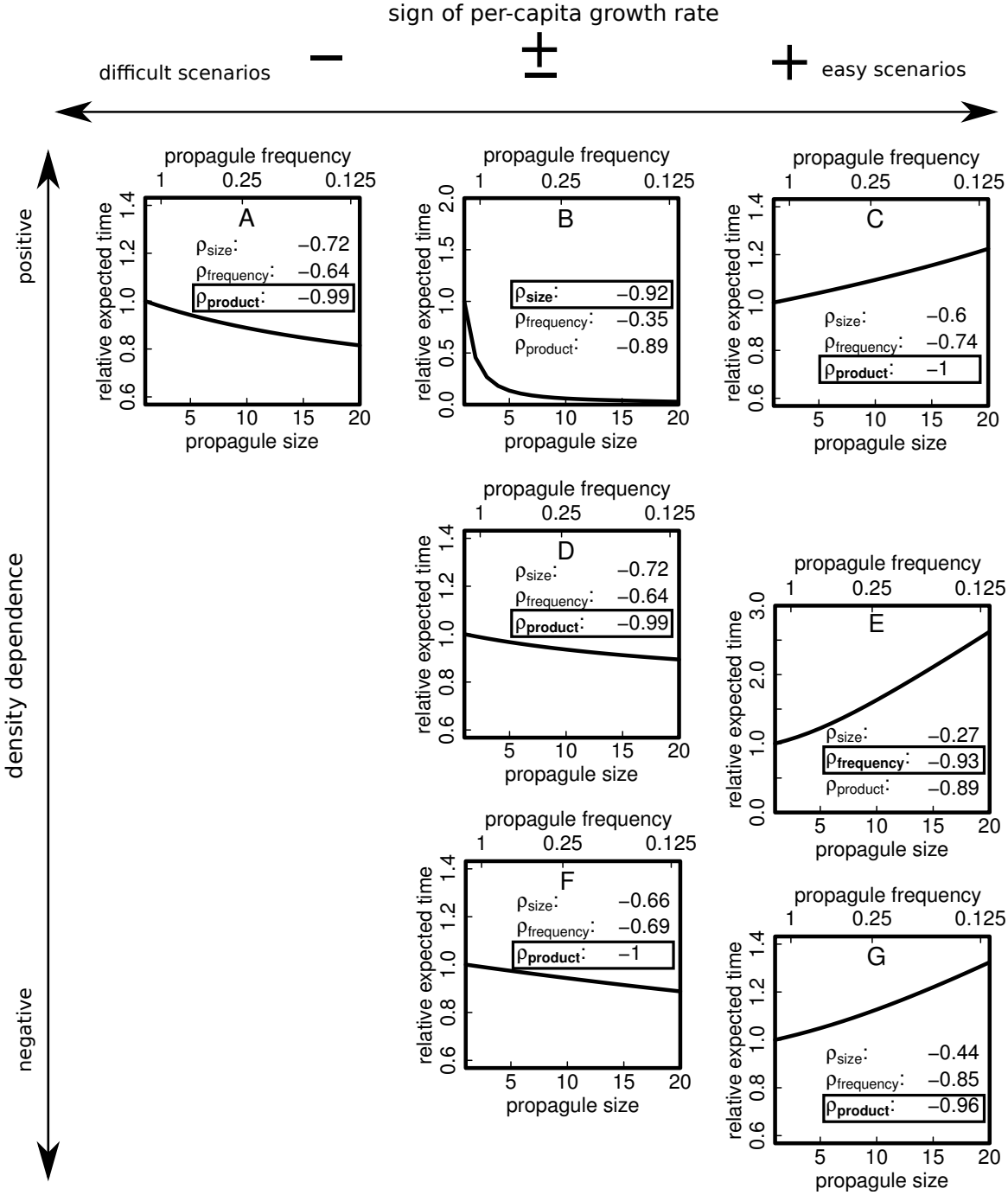

Figure 2.2: Expected times to reaching the target population size for different values of propagule size and frequency with a fixed value of their product ($s \cdot f = 2.4$). All expected times are given relative to the corresponding expected time for a propagule size of 1 (see Table 2.2). Please note that the scaling of the y-axis differs among scenarios. ρ_{size}, $\rho_{\text{frequency}}$, and ρ_{product} represent the rank correlation coefficients between the expected times and the respective measure of propagule pressure across parameter combinations $(s, f) \in \{1, \ldots, 10\} \times \{0.005, 0.010, \ldots, 0.095, 0.1\}$. The measure with the strongest rank correlation coefficient is highlighted. For an explanation of the ecological scenarios A-G please refer to Fig. 2.1 and the section Modeling approach and ecological scenarios.

and difficult scenarios (Figs. 2.4 and 2.6).

The rank correlation coefficients displayed in Fig. 2.2 (ρ_{size}, $\rho_{\text{frequency}}$, and ρ_{product}) indicate that in five of our seven ecological scenarios the product of propagule size and frequency correlated more strongly with the expected times than either propagule size or frequency alone. In one case, the strong Allee-effect scenario B, propagule size was the strongest correlator and in another case, the exponential-growth scenario E, propagule frequency was the strongest correlator. In Appendix 2.3, we outline in detail how the rank correlation coefficients depend on parameters of each of the two models. These results confirm that although the product of propagule size and frequency is the strongest correlator across wide regions of parameter space, there are also regions where the expected time to reach the target state correlates more strongly with either propagule size or frequency alone, for example cases with very strong Allee effects or cases where per-capita growth rate takes large positive values (Figs. 2.5 and 2.9). When comparing just the correlation coefficients of propagule size and frequency, we observe that in difficult or mixed scenarios, with the exception of scenario F, propagule size was a stronger correlator than propagule frequency. Under easy scenarios, on the other hand, propagule frequency was a stronger correlator. Again, the detailed results in Figs. 2.5 and 2.9 confirm this conclusion.

Empirical examples

To illustrate how our results might be applied in practice, we now consider three empirical examples from the literature, two historical data sets and one experimental study. These data sets contain information on propagule size, propagule frequency, and a measure of invasion success such that we can attempt to place them into our framework of ecological scenarios in Fig. 2.1 by examining the relative effect of the components of propagule pressure. Since our results suggest that the response of an ecological scenario to different introduction regimes depends mostly on whether the scenario is easy or difficult, and much less on the type of density dependence, we only attempt to place our examples along the per-capita growth rate axis. Please note, however, that this analysis is not a formal validation of our modeling results, since there is no independent information on the kind of ecological scenario into which these examples fall, although one type of scenario usually appears more plausible than the other.

Experimental *Daphnia* introductions

Our first example is an introduction experiment performed in the laboratory by Drake *et al.* (2005). Implementing 16 different combinations of propagule size and propagule frequency, some of which had the same value for the product of the two components, Drake *et al.* (2005) introduced the crustacean *Daphnia magna* into experimental microcosms. Their experimental approach is very similar to the assumptions in our modeling framework, with two exceptions: First, the introduction events in their experiment happen at regular intervals and not at random time points. The second difference is that once a population went extinct in their experiment, no more individuals were introduced, whereas we assume that introduction events happen independently of the population dynamics.

Their response variables were population growth, population persistence, and time to extinction, and for each of them they performed model selection using multiple linear regression, logistic regression, and Cox proportional hazards regression (Cox & Oakes, 1984), respectively. The product of propagule size and frequency had a significant positive effect on all three measures for invasion success. For population growth, they additionally detected a significant positive effect of propagule frequency. In all other cases, neither propagule size nor frequency significantly contributed to explaining the variation in experimental outcomes. These results address our first research question.

To also obtain an answer to our second question, we computed Spearman's rank correlation coefficients between the different measures of propagule pressure and average population size at the end of the experiment (data provided on J.M. Drake's laboratory website). The final population size had the strongest correlation to the product of propagule size and frequency (0.86), a slightly weaker correlation to propagule frequency (0.81), and a much weaker correlation to propagule size (0.40). These findings are in line with what would be expected in an easy scenario (see Fig. 2.2, placement in Fig. 2.1), which is not surprising, since the individuals were introduced into a suitable medium under constant conditions. This is also consistent with the observation that the introduced populations persisted until the end of the experiment in 89 % of the experimental units.

Parasitoids released for biological control

The second example is taken from Hopper & Roush (1993) who studied a historical data set on the establishment success of three taxonomic groups of parasitoids (chalcidoids, ichneumonoids, and tachinids) introduced as biological control agents against Lepidoptera. For each of the three groups, Hopper & Roush (1993) fit binomial generalized linear models

with number of individuals collected (a proxy for genetic diversity), total number released (analogous to the product of size and frequency), mean number released at each site in each year (propagule size), and number of releases (proportional to propagule frequency) as predictors. To select the most important predictors, they performed stepwise model selection using a likelihood ratio testing approach. For two of the groups (ichneumonoids and tachinids), propagule size was the only predictor included into the model, for one group the total number released (chalcidoids). Thus, at least for the former two groups, these parasitoid introductions apparently fall into our difficult or mixed scenario class (see Fig. 2.1). The average establishment probabilities in these two groups were 25 % and 36 %, whereas chalcidoids had an average establishment probability of 45 %. Hopper & Roush (1993) discussed a mate finding Allee effect as a possible explanation for the important role of propagule size, but our results indicate that other ecological scenarios may also lead to a large effect of propagule size.

The Australian dung beetle project

Our last example is a historical data set on the Australian dung beetle project (Tyndale-Biscoe, 1996). Between 1968 and 1984, 1.73 million beetles belonging to 43 species were introduced to decompose the large quantities of dung produced by alien cattle (Edwards, 2007). This data set is particularly suitable for our study, as many species were introduced to different places with different combinations of propagule size and propagule number. Despite the overall high numbers of individuals introduced in this project, there is variation in propagule pressure among species that has been suggested as an explanation for the differential establishment success (Edwards, 2007).

We extracted data on the original dung beetle releases in Australia (not on the later redistributions) from Tyndale-Biscoe (1996). We considered introduction events within the same town as belonging to one location. For each species and each location, we scored propagule size and propagule frequency. We computed propagule frequency as the number of distinct introduction events in the data set divided by the duration of the whole project, i.e. 16 years. To compute propagule size, we ignored the introduction events for which the number of individuals introduced was unknown and averaged over all other introduction events. The data set by Tyndale-Biscoe (1996) also contains information on whether individuals of the alien species were found at the release site in a later year. If individuals were later encountered at at least one release site within a location, we scored the establishment at that location as successful. For our analysis, we selected the seven species for which we had more than 20 locations with information on propagule size, frequency, and success.

Averaged across these seven species and across locations, the mean propagule frequency was 0.14 and the mean propagule size 663 (see Table 2.3 for more detailed information). Successful establishment occurred on average at 49 % of locations.

For each of the seven species, we used R (R Development Core Team, 2011) to fit five candidate binomial generalized linear models (GLMs) for establishment success: three models with only a single predictor (propagule size, frequency, or their product) and two models that included the product and either propagule size or frequency. Using Akaike's information criterion (AIC), we selected one model for each species (see Appendix 2.5 for details on the statistical analyses). For five species, the selected model contained only the product, and for one species only propagule frequency. For one species, *Euoniticellus africanus*, the selected model contained the product and propagule size as predictors. However, the coefficient of propagule size was negative and the difference in AIC values to the model with only frequency was not significant (simulation-based p-value: 0.14, see Appendix 2.5 for details). These results imply that also in this case a high propagule frequency was beneficial for establishment success. Thus, at least for the latter two species, the results indicate an easy ecological scenario (Fig. 2.1). This finding is in accordance with our expectations, as the dung beetles were released with the intention to establish them, and thus the responsible agency tried to optimize the conditions encountered by the released individuals (Edwards, 2007).

Discussion

Effect of propagule size and frequency in difficult and easy scenarios

As hypothesized, given a certain average number of individuals introduced per time unit, the relationship between the temporal distribution of introduced individuals and invasion success depends mainly on whether the alien population has a negative or a positive per-capita growth rate in its new environment. Under difficult ecological scenarios, i.e. those with a negative average population growth rate, the population grows faster if propagule size is high, that is if introduced individuals are clustered in time. On the other hand, for easy ecological scenarios, i.e. those with a positive average growth rate, a high propagule frequency leads to a faster population growth. Less relevant than the sign of per-capita growth rate was the type of density dependence. Interestingly, the effects of propagule size and frequency under a weak Allee-effect (scenario C) were very different from those under a strong Allee effect (scenario B) and more similar to those in scenarios E (exponential growth) and even

G (negative frequency dependence). Since introduction regimes with few large introduction events are associated with more variability than those with many small introduction events, our results are consistent with those of Rajakaruna *et al.* (2013) who found that stochasticity in immigration rate increases establishment probability in unfavorable habitats but is disadvantageous in favorable habitats.

Although the expected times to reach the target population size differ substantially between introduction regimes with the same value of the product of propagule size and frequency, our results on the rank correlations indicate that in many cases these differences are slight enough for the product to remain a better single predictor of invasion success than either propagule size or frequency. This does not imply, however, that the product is the optimal single-number measure for propagule pressure. It is conceivable that a product in which size and frequency have different exponents depending on their relative effect would perform even better.

Furthermore, the results for scenarios B and E in in Fig. 2.2 and those in Appendix 2.3 indicate that there are ecological scenarios in which either propagule size or frequency is the strongest correlator of invasion success. For parameter combinations with a strongly positive per-capita growth rate under which the target state is reached very rapidly, propagule frequency appears to be the strongest correlator (see scenario E and Figs. 2.5 and 2.9). Propagule size, on the other hand, can be the strongest correlator under very strong Allee effects (see scenario B and Fig. 2.5) and possibly other very difficult scenarios.

Relative strength of effects and invasion processes with both difficult and easy stages

Our results indicate that in ecological scenarios where per-capita growth rate is positive in one population-size range and negative in another, the target state is reached faster for larger and thus less frequent introduction events, as is the case in difficult scenarios. Hence in such mixed scenarios, the difficult phase seems to dominate. This may simply result from the fact that the difficult phase is usually much longer such that reducing it by a certain proportion has a larger effect on the overall time than the same proportional reduction of the easy phase would have.

Furthermore, the model developed in Appendix 2.1 suggests that the positive effect of a high propagule size under a difficult ecological scenario may be more substantial than the positive effect of a high propagule size under an easy scenario. We could attribute the latter phenomenon to two kinds of edge effects: Introduction events with a high propagule frequency lead to a faster invasion because (1) the waiting time until the first introduction event is shorter, and (2) when the population size is already very close to the target, the

waiting time to the next introduction event that will bring the population to the target is shorter. We acknowledge that these effects might be specific to the way we measure invasion success, i.e. as expected time to reach a target population size in the case of a sequence of introduction events. In any case, these effects are probably weak except for very small propagule frequencies (see Fig. 2.8). Thus, as long as there is one stage of the invasion process in which the per-capita population growth rate is negative, we would expect this stage to determine the influence of propagule size and frequency. The results for the extension of the single-population model with environmental fluctuations (see Appendix 2.4) suggest that the important role of propagule size under difficult scenarios also dominates the tendency of propagule frequency to have a larger effect in varying environments, at least for the parameter combinations we examined.

Ecological relevance of difficult vs. easy scenarios

Since in absolute terms invasions are less likely or proceed more slowly under difficult scenarios than under easy scenarios, one could argue that the relative effect of the components of propagule pressure under difficult ecological scenarios lacks relevance. In our opinion, however, the results on difficult scenarios are at least as relevant for invasion biology, because they are concerned with the cases for which propagule pressure matters most. From a management perspective, it is very hard to prevent population growth of an alien species under an easy scenario. Propagule pressure would have to be reduced to zero, since few individuals would be sufficient to trigger population growth. In difficult scenarios, in contrast, it is feasible to prevent or delay population growth by a reduction of propagule pressure or a change in the temporal distribution of introduced individuals.

Stochastic vs. deterministic models

While deterministic and stochastic models would yield similar results for easy scenarios, their predictions may be quite different for difficult scenarios. Under a strong Allee effect, for example, a single introduction below the critical population size would inevitably go extinct in standard deterministic models, whereas it has a small probability of succeeding under stochastic models (Dennis, 2002). In the case of multiple introductions, the deterministic modeling results by Mailleret & Lemesle (2009) imply that a population below the Allee threshold can only grow if propagule pressure is high enough to compensate the population decline. In stochastic models, on the other hand, a random accumulation of introduction events can lead to a phase of population growth even if average propagule pressure

is low. Unlike deterministic models, stochastic models thus predict that difficult phases in the invasion process can be overcome, although this may take a long time. This prediction of stochastic models may account for some of the commonly observed time lags in which an alien population stays at a relatively low population size for a long time until it suddenly grows and proceeds to further invasion stages (Crooks, 2005). In our opinion, stochastic models are therefore a key tool for understanding how alien species proceed from one stage to the next in the invasion process.

Empirical data sets

Of the three empirical examples considered in this study, two—the experimental *Daphnia* introductions by Drake *et al.* (2005) and at least some of the dung beetles introduced to Australia—appear to represent easy ecological scenarios while the introductions of ichneumonoid and tachinid parasitoids discussed by Hopper & Roush (1993) seem to come from a difficult scenario. Although we do not have independent measurements of per-capita growth rates, these results are consistent with what we know about the underlying ecological conditions and also agree with the relative average success probabilities: The parasitoids had the lowest average success probability, the dung beetles were intermediate, and the *Daphnia* introductions had the highest success probability.

When drawing conclusions from historical data sets such as those from Hopper & Roush (1993) or Tyndale-Biscoe (1996), some caution is warranted. One problem is that a seemingly causal effect of a predictor variable (e.g. propagule size or frequency) on invasion success may be due to a correlation with an actual causal variable that is not included in the analysis. This might happen, for example, if more releases are made at sites where the alien species encounters good conditions. Furthermore, whether or not a predictor is chosen by some model selection procedure can also depend on the magnitude of variation in the predictors themselves, which can vary considerably in historical data (see e.g. Table 2.3). Thus the component of propagule pressure that explains most variation as part of a statistical model may not always be the component with the strongest effect on invasion success in general.

These limitations of inference from historical data demonstrate that experimental studies are important to improve our understanding of how the temporal distribution of introduced individuals affects invasion success. Apart from the experimental *Daphnia* introductions by Drake *et al.* (2005), we know of only one other such study: Hedge *et al.* (2012) performed colonization experiments with larvae of the pacific oyster *Crassostrea gigas* and found higher population growth with frequent small introduction events compared to fewer

large introduction events, a phenomenon that they attributed to reduced intraspecific competition or increased facilitation if individuals arrive at different time points.

Outlook

Future extensions of our modeling approach might take into account aspects of propagule pressure beyond propagule size and frequency. While we assumed that introduction events took place at well-defined independent locations within which all individuals had the ability to interact, for some species it might be more reasonable to explicitly model the spatial configuration of the habitat in the introduced range and the spatial distribution of introduced individuals (see Drury *et al.*, 2007, for an example of such models).

Another simplifying assumption we made is that successive introduction events all have the same propagule size. As, for example, in the dung beetle example, propagule size varied considerable between introduction events within the same location (see Table 2.3), it would be interesting to model how variation in propagule size among introduction events affects invasion success. We would expect that this additional variation would contribute to accelerating invasions under difficult ecological scenarios. In some cases (see e.g. Gamfeldt *et al.*, 2005; Burgess & Marshall, 2011; Hufbauer *et al.*, 2013), it might not even be sufficient to consider the quantity of introduced individuals, but one might also need to include aspects of quality such as the genetic composition of propagules, their body condition, or life-history stage.

Acknowledgments

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Appendix 2.1 Formal development of hypotheses

To formalize the hypotheses developed in the main text, let us consider the following stochastic process: Some entity, e.g. a population or a vehicle, starts at position 0 and, at rate f, makes jumps of magnitude s. Between jumps, the entity moves with constant velocity v, but only at positions greater than 0 (see Fig. 2.3 for example trajectories). In analogy to our overall research question, we ask: Is the expected time until the entity reaches some target position R shorter with many small jumps or with fewer large jumps?

To address this question, let N_t be a random variable for the number of jumps during some time interval of length t. Then N_t is Poisson distributed with parameter $f \cdot t$. Further, let $X_t = s \cdot N_t$ be the displacement of the entity due to these jumps. Then

$$\mathbf{E}[X_t] = \mathbf{E}[s \cdot N_t] = s \cdot \mathbf{E}[N_t] = s \cdot f \cdot t \,. \tag{2.2}$$

Thus, the average velocity with which the entity moves is $\bar{v} = s \cdot f + v$, and so it depends only on the product of s and f.

If the average velocity is positive, i.e. in easy scenarios, the expected time to reach the target state R should be approximately $R/\bar{v}$. The observed advantage of a high propagule frequency or jump rate f in easy scenarios can be explained by two kinds of edge effects (Fig. 2.3 a). The first edge effect results from the fact that movement only starts after the first jump out of position 0. Thus the higher the jump rate is, the earlier the system starts to move deterministically towards its target. To understand the second edge effect, compare the scenario where jumps of magnitude $2s$ occur at rate $f/2$ to the scenario with magnitude s and rate f. Assume that the system is already within distance s of the target state. Then the expected time until the target is reached is $2/f$ in the former scenario, and only $1/f$ in the latter scenario with the higher jump rate. In the former scenario, the system overshoots the target, an effort that can be considered wasted if only the time to reach the target state is of interest. While the first edge effect is particularly strong if the jump rate is small compared to the velocity v, the second becomes important if the distance between start and target position is small.

In contrast, in difficult scenarios with a negative average velocity, our entity of interest would never reach the target state under a deterministic model. In our stochastic model, however, the target state will eventually be reached when the displacement during some time interval t is considerably larger than expected due to a chance accumulation of jumps (Fig. 2.3 b). A measure of how frequent such unusual events are is the variance of the

displacement

$$\mathbf{Var}(X_t) = \mathbf{Var}(s \cdot N_t) = s^2 \cdot \mathbf{Var}(N_t) = s^2 \cdot f \cdot t\,. \tag{2.3}$$

Thus for a fixed value of the product $s \cdot f$, the variance increases with the magnitude of jumps s and thus the expected time to reach the target state decreases. The more negative the average growth rate is, the stronger is this effect (see Fig. 2.4).

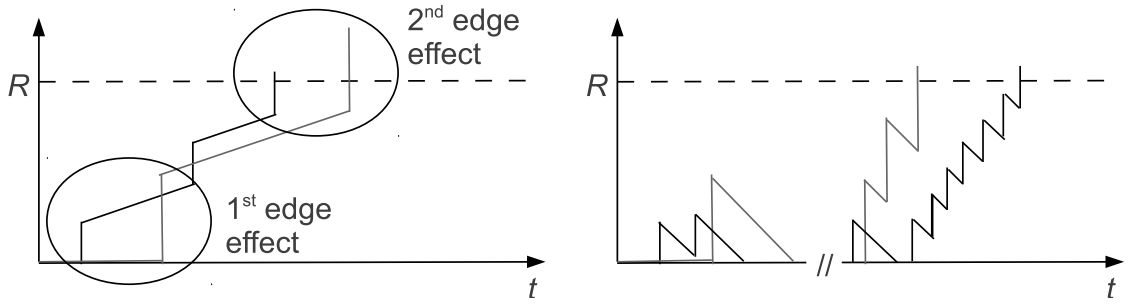

Figure 2.3: Example trajectories illustrating the heuristic arguments for (a) "easy" ecological scenarios and (b) "difficult" ecological scenarios. The gray lines correspond to introduction regimes with low propagule frequency and high propagule size whereas the black lines represent scenarios with high propagule frequency and low propagule size.

Appendix 2.2 Details on the Markov processes and their analysis

Single-population model

Each state of this Markov process is characterized by the current population size of the alien species $i \in \{0, 1, 2, \dots\}$. From state i, the system jumps to other states $j \in \{0, 1, 2, \dots\}$ at rates

$$\bar{\lambda}_{i,j} = \begin{cases} b(i) & \text{for } j = i + 1 \\ d(i) & \text{for } j = i - 1\,, \\ 0 & \text{otherwise} \end{cases} \tag{2.4}$$

where $b(i)$ and $d(i)$ are the rates at which birth and death events, respectively, occur in the population. Transitions due to introduction events happen at rate

$$\hat{\lambda}_{i,j} = \begin{cases} f & \text{for } j = i + s \\ 0 & \text{otherwise} \end{cases} . \tag{2.5}$$

We assumed a constant per capita death rate of 1, such that $d(i) = i$. The birth rate at population size i is $\beta_q \cdot i^2 + \beta_l \cdot i$, where $\beta_q \geq 0$ and $\beta_l \geq 0$ quantify the birth rates due to processes that, respectively, do or do not require interactions such as cooperation or mate finding for reproduction. With different choices of the two parameters, this model produces a range of different scenarios. Scenarios B, C, and E in Fig. 2.1 are three such examples (See Table 2.1 for the corresponding parameter values). For $\beta_q > 0$, the birth rate is positively density-dependent. If, at the same time, $\beta_l + \beta_q < 1$, the birth rate is smaller than the death rate at small population sizes. Hence, the population experiences a strong demographic Allee effect with critical population size $(1 - \beta_l)/\beta_q$. Below this critical size, the population tends to decline and for small propagule sizes we thus obtain an ecological scenario with a difficult initial stage (Scenario B in Fig. 2.1). If, on the other hand, $\beta_q > 0$ and also $\beta_l + \beta_q > 1$, the population is expected to grow even at small sizes but its per capita growth rate increases with population size. This so-called weak Allee effect is an easy scenario (scenario C in Fig. 2.1). If $\beta_q = 0$ and $\beta_l > 1$, the population grows exponentially (scenario E in Fig. 2.1).

Competition model

This model is a modified version of the competition model in Wittmann *et al.* (2013a) and also has parallels to the model by Duncan & Forsyth (2006). The competition model is characterized by the fixed total community size K, the fecundity of the alien species relative to the native species w, and by the competition coefficient α, which specifies the strength of interspecific competition relative to intraspecific competition. Thus, the competition experienced by an individual whose own species has size x is $c(x, y) = x + \alpha \cdot y$ if the other species has population size y. We assume that the rate at which individuals die is proportional to the competition they experience. A dead individual is immediately replaced by an individual drawn at random from a large offspring pool to which individuals contribute in proportion to their fecundity.

For consistency with Wittmann *et al.* (2013a), here the state n of the Markov process represents the current number of native individuals in the population, thus $n \in \{0, 1, \ldots, K\}$.

The transition rates due to birth and death events are then:

$$\bar{\lambda}_{n,n+1} = \underbrace{\frac{c(K-n,n)\cdot(K-n)}{K}}_{\substack{\text{rate at which}\\ \text{members of the alien}\\ \text{species die}}} \cdot \underbrace{\frac{n}{(K-n)\cdot w+n}}_{\substack{\text{probability that a}\\ \text{native individual}\\ \text{gives birth}}} \text{ for } n < K \tag{2.6}$$

and

$$\bar{\lambda}_{n,n-1} = \underbrace{\frac{c(n,K-n)\cdot n}{K}}_{\substack{\text{rate at which}\\ \text{native individuals die}}} \cdot \underbrace{\frac{(K-n)\cdot w}{(K-n)\cdot w+n}}_{\substack{\text{probability that an}\\ \text{alien individual}\\ \text{gives birth}}} \text{ for } n > 0 \tag{2.7}$$

and $\bar{\lambda}_{n,m} = 0$ for $m \notin \{n-1, n+1\}$.

The introduction process here is the same as in the single-population scenario. However, after each introduction event, the number of individuals in the community is truncated to K by randomly removing s individuals in proportion to the competition they experience. Thus, transitions due to introduction events happen at rates

$$\hat{\lambda}_{n,n-k} = f \cdot H[k, n, K-n+s, s, c(n, K-n+s), c(K-n+s, n)] \text{ for } n-k \in \{0, 1, \ldots, K\}, \tag{2.8}$$

where $H[k, n, K-n+s, s, c(n, K-n+s), c(K-n+s, n)]$ is the probability mass function of Wallenius' noncentral hypergeometric distribution (Fog, 2008), i.e. the probability that in a community with n native and $K - n + s$ alien individuals, k native individuals are selected to be killed when drawing s individuals without replacement, and where native individuals have weight $c(n, K - n + s)$ and alien individuals weight $c(K - n + s, n)$. We computed H using the package BiasedUrn (Fog, 2011) in R (R Development Core Team, 2011).

We used the competition model to create scenarios A, D, F, and G in Fig. 2.1. The underlying parameter values can be found in Table 2.1.

Table 2.1: Models and parameter values underlying the ecological scenarios considered in the main text (see Fig. 2.1)

Scenarios generated by the single-population model				
Scenario		density-independent birth rate β_l	density-dependent birth rate β_q	target population size R
B		0	0.05	50
C		1.05	0.004	50
E		2	0	50
Scenarios generated by the competition model				
Scenario	competition coefficient α	alien fecundity w	carrying capacity K	target population size R
A	1.2	1	100	50
D	1.2	1	100	100
F	0.68	1	100	100
G	0.68	1	100	50

Analysis

For each model, the total transition rate from state i to state j, $\lambda_{i,j}$, is the sum of the transition rate due to introduction events and the transition rate due to other events:

$$\lambda_{i,j} = \hat{\lambda}_{i,j} + \bar{\lambda}_{i,j} \text{ for } i \neq j \, . \tag{2.9}$$

λ_i is the total rate at which the Markov process leaves state i. These transition rates can be organized into a rate matrix $\mathbf{\Lambda}$ whose diagonal entries are given by $\lambda_{i,i} = -\lambda_i$.

When computing the expected time to reach the target population size R, only transitions from states with alien population sizes smaller than the target population size are relevant. We denote this set of states J. For the competition scenario, $J = \{K - R + 1, \ldots, K\}$ and for the single-population scenario $J = \{0, 1, \ldots, R - 1\}$. Now, consider a realization of the Markov process that starts in a state $i \in J$. Then the time T_i to reach state R can be decomposed into the time until the Markov process first leaves state i and the remaining time. Taking expectations and using the Markov property, we obtain:

$$\mathbf{E}[T_i] = \frac{1}{\lambda_i} + \sum_{j \in J, j \neq i} \frac{\lambda_{i,j}}{\lambda_i} \cdot \mathbf{E}[T_j]. \tag{2.10}$$

Note that we do not need to include summands for states outside J, because once the process leaves J it has reached the target and the remaining time is 0.

The system of linear equations that consists of one such equation for each $i \in J$ was

solved numerically in R (R Development Core Team, 2011) for T_{i_0}, the expected time belonging to the initial state i_0 ($i_0 = 0$ in the single-population scenario and $i_0 = K$ in the competition scenario). This corresponds to solving the matrix equation $\tilde{\mathbf{\Lambda}}\,\mathbf{E}[T] = -\mathbf{1}$, where $\tilde{\mathbf{\Lambda}}$ is the matrix obtained by removing from $\mathbf{\Lambda}$ all rows and columns belonging to the states that are not in J. $\mathbf{E}[T]$ is a column vector of expected times and $\mathbf{1}$ is a column vector with a 1 in each element. The expected times to reach the target state for the seven scenarios considered in the main text and for a propagule size of 1 are shown in Table 2.2.

Table 2.2: Expected times to reach the target population size for a propagule size of 1. The relative expected times in Fig. 2.2 refer to these values.

scenario	expected time
A	72.0
B	586.2
C	11.0
D	118.4
E	3.5
F	11254.6
G	13.0

Relative difference in expected time to reach the target

In the following sections, we explore the continuous dependence of the results on the parameters of our two models. To be able to visualize the results, we summarized the relative effect of propagule size and frequency within one quantity. To this end, we first computed the expected time of interest $\mathbf{E}[T]$ with propagule size s and propagule frequency f, and then computed the corresponding expectation $\mathbf{E}[T^*]$ with propagule size $s+1$ and propagule frequency $f \cdot s/(s+1)$. We then defined the sensitivity to this perturbation, our desired single quantity, as

$$\Delta := \frac{\mathbf{E}[T^*] - \mathbf{E}[T]}{\mathbf{E}[T]}\,, \tag{2.11}$$

i.e. as the relative difference between the two expected times.

Under the hypothesis that only the product of propagule size and propagule frequency matters, we would expect $\Delta = 0$. Negative values of Δ represent cases where the introduction regime with the larger propagule size led to a faster invasion, whereas for positive Δ invasion was faster for the scenario with the higher propagule frequency. In the remainder of the text, we will abbreviate these two outcomes by saying that propagule size or propag-

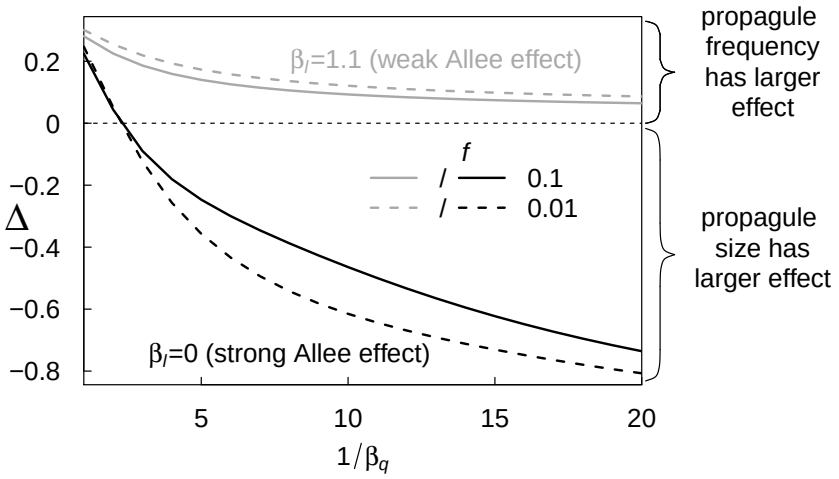

Figure 2.4: The sensitivity score Δ (see equation 2.11) as a function of the intensity of the Allee effect ($1/\beta_q$) in the single-population model with a strong ($\beta_l = 0$, black lines) or a weak Allee effect ($\beta_l = 1.1$, gray lines) and propagule frequencies f of 0.1 and 0.01. Note that with increasing values of $1/\beta_q$, the weak Allee-effect scenario approaches the exponential growth scenario. $R = 50, s = 3$.

ule frequency have a larger effect, respectively. In addition to providing relative differences in expected times, we also show the rank correlation coefficients as a function of the model parameters.

Appendix 2.3 Results in dependence on model parameters

Single-population model

In the single-population model, the sensitivity score Δ (see equation 2.11) decreases with decreasing density-dependent birth rate β_q, or increasing $1/\beta_q$ (Fig. 2.4). In other words, the more interactions between individuals are required to produce an offspring in a density-dependent manner, the larger is the effect of propagule size. Under a weak Allee effect, however, propagule frequency remained the component with the larger effect for all values of β_q. If the Allee effect is strong, on the other hand, propagule size has a larger effect than propagule frequency for all but the smallest critical population sizes (small values of $1/\beta_q$). The latter cases actually represent easy ecological scenarios because the propagule size is larger than the critical population size. For easy scenarios, propagule frequency had a larger effect when it was small, whereas for difficult scenarios, a decrease in propagule frequency increased the effect of propagule size even more.

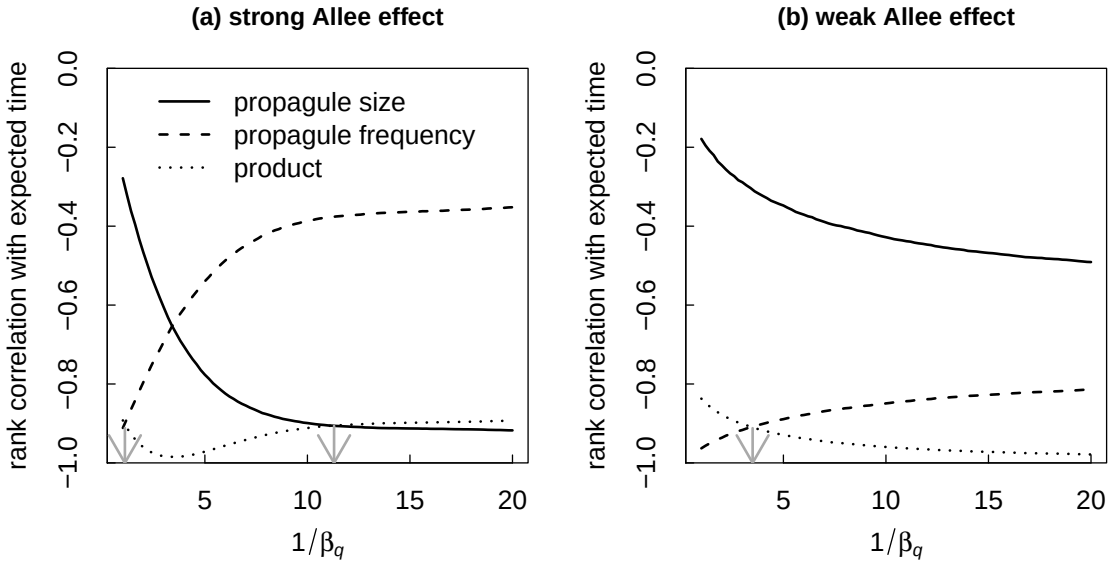

Figure 2.5: Rank correlation of the expected time to reach the target state and the three measures of propagule pressure as a function of the intensity of the Allee effect ($1/\beta_q$) in the single-population model. The strong Allee effect in (a) is produced by setting $\beta_l = 0$, whereas in (b) $\beta_l = 1.1$. The gray arrows indicate the points where a different measure of propagule pressure becomes the strongest correlator. The analysis is based on the introduction regime $(s, f) \in \{1, \ldots, 10\} \times \{0.005, 0.010, \ldots, 0.095, 0.1\}$. Note that with increasing values of $1/\beta_q$, the weak Allee-effect scenario approaches the exponential growth scenario. $R = 50$.

With increasing intensity of the Allee effect (increasing $1/\beta_q$) the expected time to reach the target population size became more strongly correlated to propagule size, while the correlation to propagule frequency became weaker (Fig. 2.5). Interestingly, for the parameter combination in Fig. 2.5 a, which represents a strong Allee effect, each of the three measures of propagule pressure had the strongest correlation with expected time in some range of $1/\beta_q$: propagule frequency for very small values, i.e. small critical population sizes, the product for intermediate values, and propagule size for very high values, i.e. large critical population sizes. In the weak Allee effect scenario of Fig. 2.5 b, the measure with the strongest correlation to the expected times changed from propagule frequency to the product with increasing $1/\beta_q$, but over the parameter range we examined, propagule size always exhibited the weakest correlation.

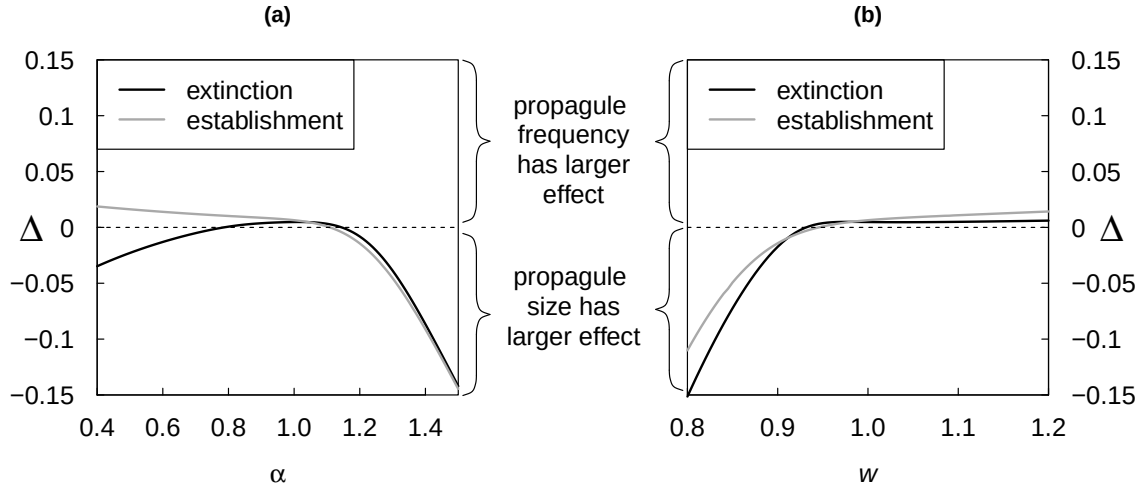

Figure 2.6: The sensitivity score Δ (see equation 2.11) of establishment time ($R = 50$) and native species extinction time ($R = 100$) in the competition model (a) for different competition coefficients α and (b) for different alien species fecundities w. $K = 100$, $f = 0.8$, $s = 3$. In a) $w = 1$; in b) $\alpha = 1$.

Competition model

In the competition model, the sensitivity of expected times to perturbations in propagule size and frequency depends on the competition coefficient α and the alien species fecundity w (Fig. 2.6). In a symmetric competition situation ($w = 1$) with an advantage for the rare species ($\alpha < 1$), a high propagule frequency is more relevant for fast establishment, whereas a high propagule size helps the alien species to rapidly exclude the native species from the community. When the more common species has an advantage ($\alpha > 1$), propagule size has a larger effect for both establishment and exclusion of the native species. If $\alpha = 1$ and the alien species has a lower fecundity than the native species ($w < 1$), propagule size has a larger effect on both times of interest, whereas propagule frequency has a larger effect if the alien species has an advantage over the native species ($w > 1$). Other combinations of α and w are explored in Figs. 2.7 and 2.8.

According to the competition model, with the parameter combination examined in Fig. 2.9, the product always had the strongest correlation to the expected time to the extinction of the native species, whereas for the establishment time of the alien species, propagule frequency correlated more strongly for small competition coefficients. In general, the correlation coefficients for establishment time and extinction time behaved similarly for competition coefficients $\alpha > 1$, but diverged for $\alpha < 1$.

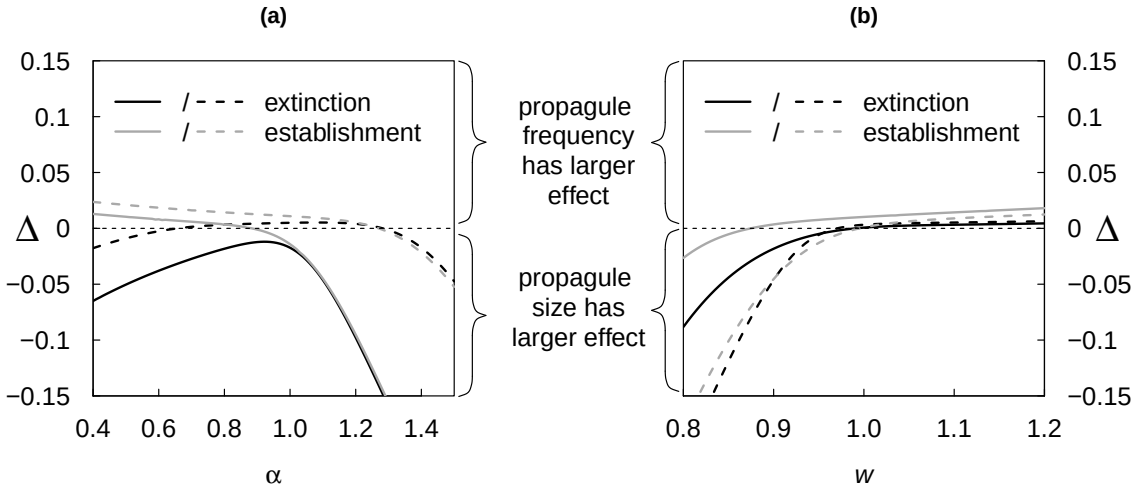

Figure 2.7: The sensitivity score Δ (see equation 2.11) of establishment time ($R = 50$) and native species extinction time ($R = 100$) in the competition model (a) for different competition coefficients α with $w = 0.9$ (solid lines) and $w = 1.1$ (dashed lines) and (b) for different alien species fecundities w with $\alpha = 0.8$ (solid lines) and $\alpha = 1.1$ (dashed lines). $K = 100, f = 0.8, s = 3$.

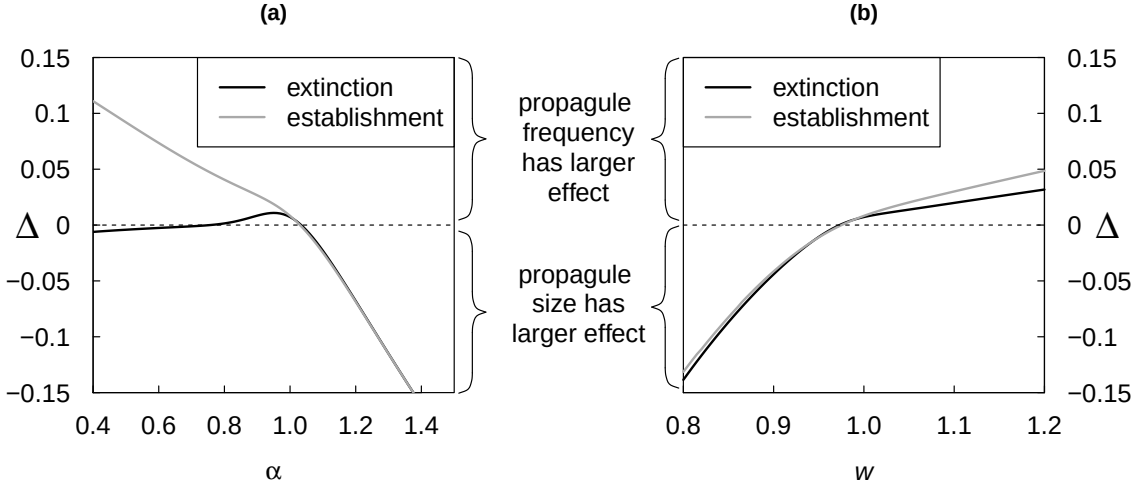

Figure 2.8: The sensitivity score Δ (see equation 2.11) of establishment time ($R = 50$) and native species extinction time ($R = 100$) in the competition model (a) for different competition coefficients α and (b) for different alien species fecundities w. The propagule frequency $f = 0.1$ is smaller than in Fig. 2.6. This leads to larger absolute values of Δ in the positive half-plane. $K = 100, s = 3$. In a) $w = 1$; in b) $\alpha = 1$.

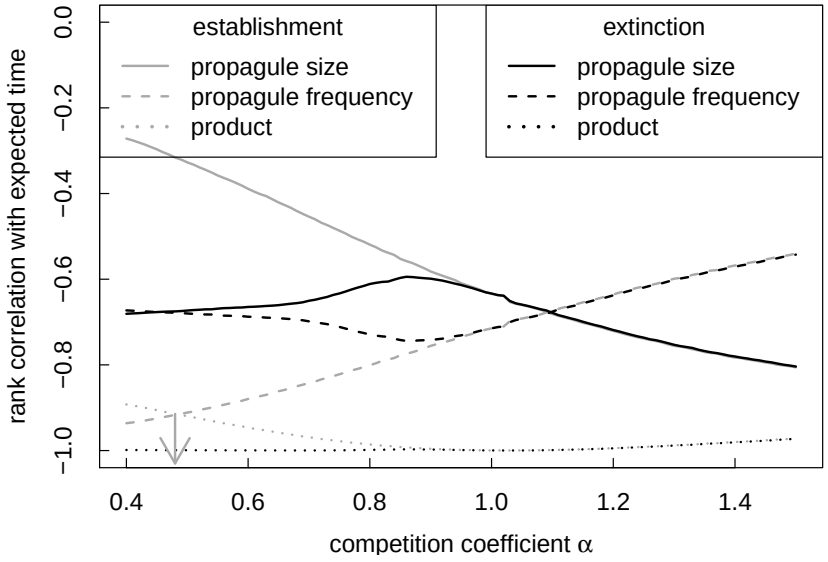

Figure 2.9: Rank correlation of the expected time to reach the target state ($R = 50$ for establishment and $R = 100$ for native species extinction) and the three measures of propagule pressure in the competition model for different competition coefficients α. The gray arrows indicate the points where a different measure of propagule pressure becomes the strongest correlator. The analysis is based on the introduction regimes $(s, f) \in \{1, \ldots, 10\} \times \{0.005, 0.010, \ldots, 0.095, 0.1\}$. $K = 100, w = 1$.

Appendix 2.4 Results for the single-population model with environmental change

Here we consider an extension of the single-population model in which there are two possible environmental states, 0 and 1, transitions between which happen at rate ϵ. The per capita birth rates in environment 0 are $\beta_{l,0} = \beta_l \cdot \phi$ and $\beta_{q,0} = \beta_q \cdot \phi$. $\beta_{l,1} = \beta_l/\phi$ and $\beta_{q,1} = \beta_q/\phi$ are the corresponding rates in environment 1. Thus β_l and β_q are now the geometric averages of the birth rate parameters and $\phi \geq 1$ quantifies the magnitude of environmental change. With $\phi = 1$, the environment is constant and we get back to the original model. To summarize, in environment j and with a current population size of i, birth events happen at rate

$$b(i) = \beta_{l,j} \cdot i + \beta_{q,j} \cdot i^2 \,. \tag{2.12}$$

To characterize the current state of the process, we now need two numbers: the population size i and the environmental state j. However, we can transform the model into a one-dimensional Markov process with the help of a one-to-one map between the two-

dimensional states and the natural numbers $\{1, \ldots, 2R\}$. After transformation, we can apply the same methods for its analysis as for the other models (described in Appendix 2.2). The results, corresponding to Figs. 2.4 and 2.5, are shown in Figs. 2.10 and 2.11. An increase in the magnitude of environmental change increases the sensitivity of expected times to propagule frequency. However, at least for the parameter combinations we considered, this effect is not strong enough to compensate the larger effect of propagule size under the difficult scenario of a strong Allee effect.

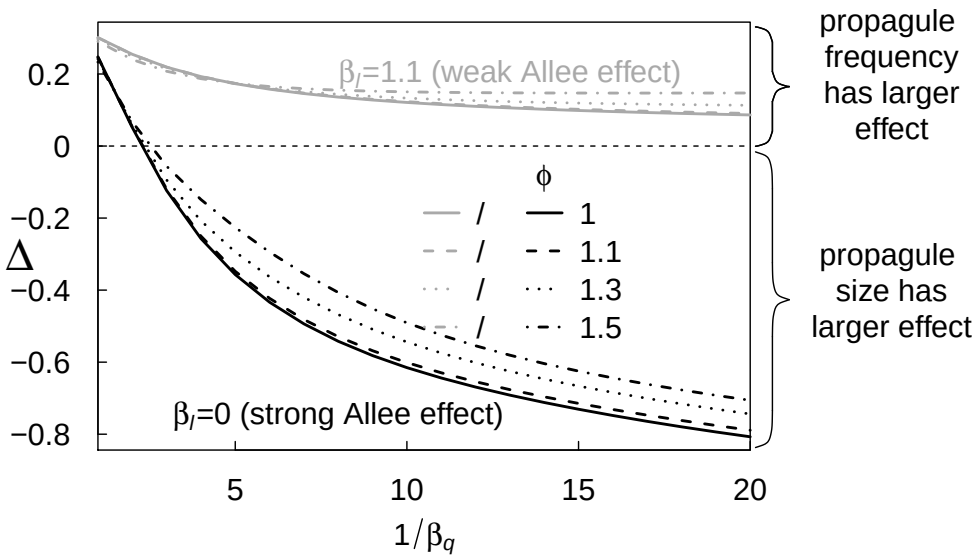

Figure 2.10: The sensitivity score Δ (see equation 2.11) as a function of the intensity of the Allee effect ($1/\beta_q$) in the single-population model with a strong ($\beta_l = 0$, black lines) or a weak Allee effect ($\beta_l = 1.1$, gray lines) and different magnitudes of environmental change ϕ. $R = 50, s = 3, f = 0.01, \epsilon = 0.1$, initial environment: 0.

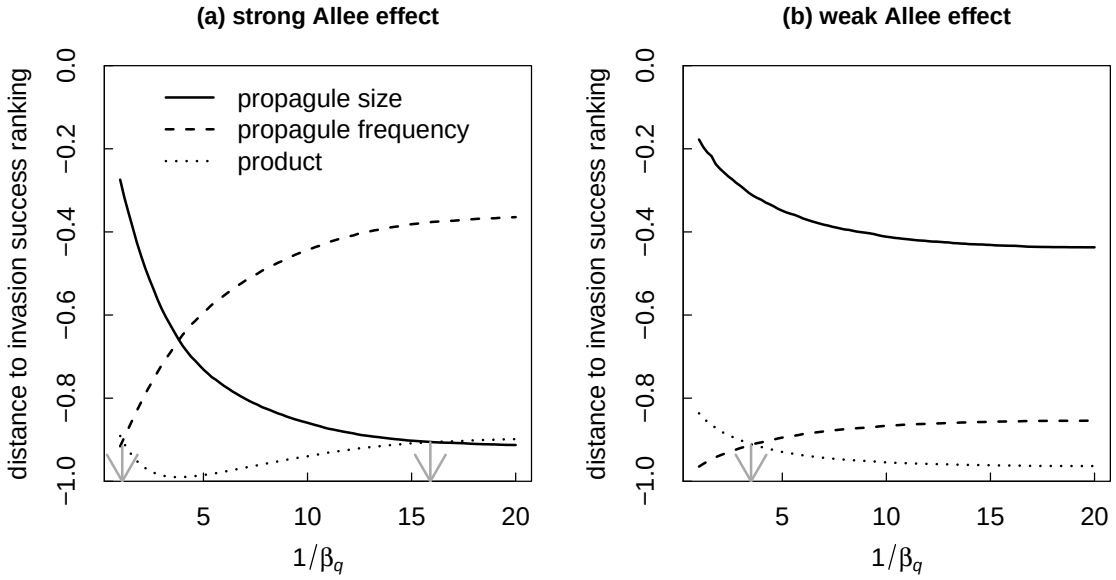

Figure 2.11: Rank correlation of the expected time to reach the target state and the three measures of propagule pressure as a function of the intensity of the Allee effect ($1/\beta_q$) in the single-population model with environmental change. The strong Allee effect in (a) is produced by setting $\beta_l = 0$, whereas in (b) $\beta_l = 1.1$. The gray arrows indicate the points where a different measure of propagule pressure becomes the strongest correlator. The analysis is based on the introduction regimes $(s, f) \in \{1, \ldots, 10\} \times \{0.005, 0.010, \ldots, 0.095, 0.1\}$. $R = 50, \phi = 1.5, \epsilon = 0.1$, initial environment: 0.

Appendix 2.5 Statistical models for the Australian dung beetle project

Here we provide details on the statistical analysis of the dung beetle data set from Tyndale-Biscoe (1996). Descriptive statistics for the seven species that we selected for our analysis are shown in Table 2.3. For each of the seven species, we fit logit-link binomial generalized linear models using the function glm (family "binomial") in R (R Development Core Team, 2011). The models were of the form

$$\log\left(\frac{p_i}{1-p_i}\right) = c_{intercept} + c_{size} \cdot s_i + c_{frequency} \cdot f_i + c_{product} \cdot s_i \cdot f_i, \tag{2.13}$$

where p_i is the success probability at location i, and s_i and f_i are the corresponding values for propagule size and propagule frequency. $c_{intercept}$, c_{size}, $c_{frequency}$, and $c_{product}$ are the model coefficients. In each of the five candidate models, we set some of these coefficients to zero while estimating the others. As criterion for model selection, we used AIC (Burnham

& Anderson, 2002), i.e. $-2 \cdot \text{log-likelihood} + 2 \cdot (\text{number of parameters})$, as implemented in the R function AIC (see Table 2.4). The model coefficients for the selected model are given in Table 2.5.

Note that whenever a one-factor model is chosen according to AIC, as was the case for all but one species, a likelihood-ratio test comparing it to a model with an additional parameter would never reject the simpler model on a 5 % level. The converse does not necessarily hold, but in the case of *Euoniticellus africanus*, a likelihood ratio test comparing the model with product and size to the model including only the product would reject the latter ($p = 3.6 \cdot 10^{-5}$).

In the case of *Euoniticellus africanus*, we also tested whether the model including the product and propagule size as predictors fits significantly better than the model with only propagule frequency. Since the two models are not nested, we could not use a likelihood-ratio test. To evaluate the significance of the observed difference in AIC values between the two models, we therefore ran simulations in R (R Development Core Team, 2011). The model assumed under the null hypothesis was the propagule-frequency model as fit to the observed data for *Euoniticellus africanus* ($c_{intercept} = -7.9$, $c_{frequency} = 74.4$, $c_{size} = c_{product} = 0$). Using this model and the same values for propagule size and frequency as in the observed data set, we generated 10,000 invasion success data sets. For each of them, we fit the two competing models and recorded their AIC values. In 14 % of the simulations, the model with the product and propagule size as predictors had an AIC advantage at least as large as the observed advantage. Thus, we conclude that this AIC difference is not significant on a 5 % level and we cannot reject the hypothesis that propagule frequency is the only influencing factor (p-value 0.14).

Table 2.3: Descriptive statistics on the biological control introductions of seven species of dung beetles (data from Tyndale-Biscoe, 1996). $\overline{f}$, $\overline{s}$, and $\overline{f \cdot s}$ give the average across locations of propagule frequency, propagule size, and their product, respectively. $\mathrm{sd}(f)$, $\mathrm{sd}(s)$, and $\mathrm{sd}(f \cdot s)$ are the corresponding standard deviations. $\overline{\mathrm{sd}}(s)$ is the average across locations of the estimated standard deviation of release sizes within one location.

Species	$\overline{f}$	$\mathrm{sd}(f)$	$\overline{s}$	$\mathrm{sd}(s)$	$\overline{f \cdot s}$	$\mathrm{sd}(f \cdot s)$	$\overline{\mathrm{sd}}(s)$
Onthophagus gazella	0.201	0.207	841.9	729.3	217.09	420.53	792.3
Onitis alexis	0.109	0.103	415.8	176.9	44.01	41.99	108.58
Onthophagus binodis	0.078	0.041	824.9	675.9	65.38	68.22	537.32
Euoniticellus intermedius	0.231	0.204	629.5	644.5	129.75	117.04	18.99
Onthophagus taurus	0.111	0.067	896.7	445	96.55	61.1	285.34
Euoniticellus africanus	0.088	0.057	512.7	308.4	44.5	38.37	11.79
Hister nomas	0.165	0.165	516.2	184.2	88.66	111.93	194.13

Table 2.4: AIC values for the different candidate binomial GLMs for the success of seven species of dung beetles. AIC values of the respective selected models are printed in bold face.

Species	product	frequency	size	product + frequency	product + size
Onthophagus gazella	**78.10**	88.38	119.49	80.04	79.50
Onitis alexis	**78.19**	82.51	94.82	80.10	80.17
Onthophagus binodis	89.70	**89.29**	89.87	89.77	91.27
Euoniticellus intermedius	**59.38**	61.03	75.98	60.33	61.18
Onthophagus taurus	**51.97**	52.11	52.07	53.75	53.97
Euoniticellus africanus	31.59	17.56	41.56	19.07	**16.52**
Hister nomas	**25.45**	26.85	35.97	26.91	27.35

Table 2.5: Coefficients of the selected binomial GLM (see equation 2.13) for the success of seven species of dung beetles. Missing entries indicate that the corresponding predictor was not part of the selected model.

Species	$c_{intercept}$	$c_{frequency}$	c_{size}	$c_{product}$
Onthophagus gazella	-2.65			0.047
Onitis alexis	-2.08			0.058
Onthophagus binodis	-1.65	19.6		
Euoniticellus intermedius	-0.96			0.022
Onthophagus taurus	-0.66			0.002
Euoniticellus africanus	-3.83		-0.013	0.219
Hister nomas	-2.84			0.048

Chapter 3

Genetic diversity in introduced populations with Allee effect

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arXiv:1311.4469

Abstract

A phenomenon that strongly influences the demography of small introduced populations and thereby potentially their genetic diversity is the Allee effect, a reduction in population growth rates at small population sizes. We take a stochastic modelling approach to investigate levels of genetic diversity in populations that successfully overcame a strong demographic Allee effect, a scenario in which populations smaller than a certain critical size are expected to decline. Our results indicate that compared to successful populations without Allee effect, successful Allee-effect populations tend to 1) derive from larger founder population sizes and thus have a higher initial amount of genetic variation, 2) spend fewer generations at small population sizes where genetic drift is particularly strong, and 3) spend more time around the critical population size and thus experience more drift there. Altogether, the Allee effect can either increase or decrease genetic diversity, depending on the average founder population size. In the case of multiple introduction events, there is an additional increase in diversity because Allee-effect populations tend to derive from a larger number of introduction events than other populations. Finally, we show that given genetic data from sufficiently many populations, we can statistically infer the critical population size.

Keywords: critical population size, founder effect, genetic variation, invasive species, stochastic modelling

Introduction

The amount of genetic diversity in a recently established population is strongly shaped by its early history: While the founder population size determines the amount of genetic variation imported from the source population, the population sizes in the following generations influence how much of this variation is maintained and how much is lost through genetic drift. A phenomenon that strongly affects this early history is the demographic Allee effect, a reduction in per-capita growth rate in small populations (Stephens *et al.*, 1999; Fauvergue *et al.*, 2012). Allee effects have been detected in species from many different taxonomic groups (Kramer *et al.*, 2009). Apart from cooperation between individuals, the study subject of the effect's eponym (Allee, 1931), they can result from a variety of other mechanisms such as difficulties to find mating partners, increased predation pressure in small populations, or biased dispersal towards large populations (Kramer *et al.*, 2009). In this study, we focus on the so-called strong demographic Allee effect, in which the average per-capita growth rate is negative for populations smaller than a certain critical population size (Taylor & Hastings, 2005).

A population whose founder size is below this threshold has a high probability of going extinct. With more and more transport of goods around the world, however, many species are introduced to a location not just once, but again and again at different time points. Eventually, a random excess in the number of birth events may cause one of these small introduced populations to grow exceptionally fast, surpass the critical population size, and then grow further to reach high population sizes. Whereas most failed introductions pass unnoticed, the rare successful populations can be detected and sampled and may have substantial impact on native communities and ecosystems.

Our main question in this study is how expected levels of genetic diversity differ between successful populations that either did or did not have to overcome an Allee effect. Answering this question would help us to understand the ecology and evolution of introduced and invasive populations in several ways. On the one hand, the amount of genetic variation is an indicator for how well an introduced population can adapt to the environmental conditions encountered at the new location. Therefore, the Allee effect—if it influences genetic diversity—could shape the long-term success and impact even of those populations that are successful in overcoming it. On the other hand, genetic patterns created by the Allee effect could help to complete a task that is very challenging when only ecological data are available (Courchamp *et al.*, 2008; Kramer *et al.*, 2009): detecting Allee effects in field populations or even estimating the critical population size. Information on the criti-

cal population size would be very valuable in practice, for example to identify maximum release rates for species whose establishment is to be prevented, or minimum release rates for those whose establishment is desired, for example in biological control or for species reintroductions (Deredec & Courchamp, 2007). Furthermore, an important task in statistical population genetics is to reconstruct the demographic history of a population and to infer parameters such as founder population sizes, times since the split of two populations, or migration rates. Should the Allee effect have long-lasting effects on patterns of genetic diversity in established populations, it would have to be taken into account in such analyses.

To our knowledge, there have not been any empirical studies on the population genetic consequences of the Allee effect and the few theory-based results are pointing into different directions. There are arguments suggesting that a strong Allee effect may lead to an increase in genetic diversity, and others that suggest a decrease. An increase in genetic diversity due to the Allee effect is predicted for populations that expand their range in a continuous habitat (Hallatschek & Nelson, 2008; Roques *et al.*, 2012). In the absence of an Allee effect, mostly alleles in individuals at the colonisation front are propagated. Under an Allee effect, the growth rate of individuals at the low-density front is reduced and more individuals from the bulk of the population get a chance to contribute their alleles to the expanding population. This leads to higher levels of local genetic diversity and weaker spatial genetic structure. A similar effect has been discussed in the spatially discrete case: Kramer & Sarnelle (2008) argued that without Allee effect even the smallest founder populations would be able to grow, leading to populations with very little genetic diversity. The Allee effect, they conclude, sets a lower limit to feasible founder population sizes and thus does not allow for extreme bottlenecks.

The Allee effect not only influences whether a population will reach high population sizes, but also how fast this happens. So far, the genetic consequences of this change in population dynamics have not been explored theoretically. However, it is often stated that the Allee effect can lead to time lags in population growth (Drake & Lodge, 2006; Simberloff, 2009; McCormick *et al.*, 2010), i.e. initial population growth rates that are small compared to growth rates attained later (Crooks, 2005). Such time lags follow almost directly from the definition of the Allee effect and would imply an increased opportunity for genetic drift and thus a reduction in genetic diversity. However, it is not clear whether time lags are still present if we consider the subset of populations that is successful in overcoming the Allee effect.

In this study, we propose and analyse stochastic models to elucidate and disentangle the various ways in which the Allee effect shapes expected levels of neutral genetic diversity.

Furthermore, we investigate under what conditions genetic diversity would overall be lower or higher compared to populations without Allee effect. First, we compare successful populations with and without Allee effect with respect to two aspects of their demography: the distribution of their founder population sizes, i.e. the distribution of founder population sizes conditioned on success, and the subsequent population dynamics, also conditioned on success and meant to include both deterministic and stochastic aspects. In a second step, we will then consider what proportion of neutral genetic variation from the source population is maintained under such a demography. Focusing throughout on introductions to discrete locations rather than spread in a spatially continuous habitat, we first consider the case of a single founding event, and then the case of multiple introductions at different time points. Finally, we explore whether the genetic consequences of the Allee effect could be employed to estimate the critical population size from genetic data.

Model

In our scenario of interest, a small founder population of size N_0 (drawn from a Poisson distribution) is transferred from a large source population of constant size k_0 to a previously uninhabited location. Assuming non-overlapping generations and starting with the founder population at $t = 0$, the population size in generation $t+1$ is Poisson-distributed with mean

$$\mathbf{E}[N_{t+1}] = N_t \cdot \lambda(N_t) = N_t \cdot \exp\left\{ r \cdot \left(1 - \frac{N_t}{k_1}\right) \cdot \left(1 - \frac{a}{N_t}\right) \right\}, \tag{3.1}$$

where k_1 is the carrying capacity of the new location, r is a growth rate parameter, and a is the critical population size. Unless otherwise noted, we use the parameter values $k_0 = 10,000$ and $k_1 = 1000$. To model Allee-effect populations, we set $a = 50$, otherwise $a = 0$. Under this model, the average per-capita number of surviving offspring per individual $\lambda(N_t)$ is smaller than one for population sizes below the critical population size a and above the carrying capacity k_1 and greater than one between critical population size and carrying capacity (figure 3.1). With $a = 0$, this model is a stochastic version of the Ricker model (see e.g. de Vries *et al.*, 2006). Its deterministic counterpart can exhibit stable oscillations or chaotic behaviour for large values of r, but here we will only consider values of r between 0 and 2, where k_1 is a locally stable fixed point (de Vries *et al.*, 2006, p. 29).

We follow the population-size trajectory until the population either goes extinct (unsuccessful population) or reaches target population size $z = 100$ (successful population).

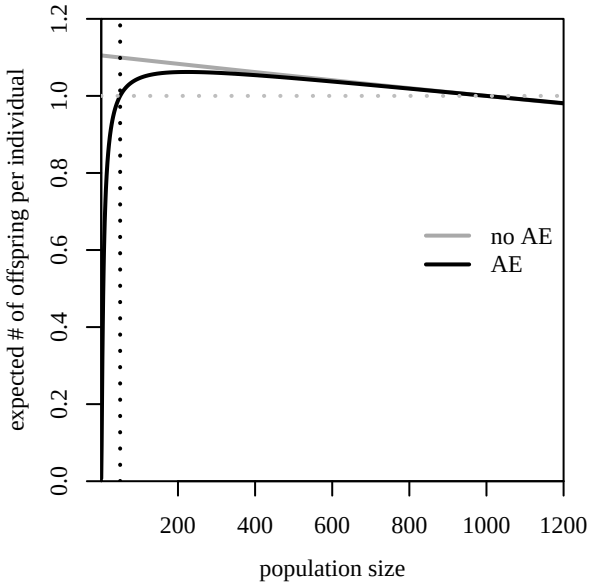

Figure 3.1: The expected number of surviving offspring per individual ($\lambda(n)$, see equation (3.1)) as a function of the current population size n without Allee effect (no AE, grey line) or with an Allee effect (AE, black line) of critical size $a = 50$ (indicated by dotted vertical line). $k_1 = 1000, r = 0.1$.

When a successful population reaches size z, we sample n_s individuals from the population and trace their ancestry backwards in time. This allows us to quantify the proportion of genetic variation from the source population that is maintained in the newly founded population. Since the impact of the Allee effect as well as the strength of genetic drift and random population-size fluctuations decline with increasing population size, the particular choice of z and k_1 should have little influence on the results as long as they are sufficiently large.

The assumption that each population goes back to a single founding event and then either goes extinct or reaches the target population size z is justified as long as introduction events are rare. Then the fate of a population introduced in one event is usually decided before the respective next event. However, many species are introduced to the same location very frequently (Simberloff, 2009). Therefore, we also consider a scenario with multiple introduction events: In each generation, an introduction event occurs with probability p_{intro}, each time involving n_{intro} individuals. We considered a population successful and sampled it if it had a population size of at least z after the first 200 generations. We fixed the number of generations rather than sampling the population upon reaching z as before, because this would introduce a bias: Populations that would take longer to reach z would be likely to re-

ceive more introduction events and thus have higher levels of diversity. With a fixed number of 200 generations and our default choice of migration probability $p_{intro} = 0.05$, all populations receive on average ten introduction events. All other parameters were unchanged compared to the case with just one founding event.

Methods

We formulated our demographic model as a Markov chain with transition probabilities

$$P_{ij} = \Pr(N_{t+1} = j | N_t = i) = \frac{e^{-\lambda(i)\cdot i} \cdot (\lambda(i) \cdot i)^j}{j!}, \tag{3.2}$$

where $\lambda(i)$ is given by equation (3.1). We used first-step analysis and Bayes' formula to compute 1) the probability of a population being successful, i.e. reaching some target size z before going extinct, 2) the conditioned distribution of founder population sizes, i.e. the distribution of founder population sizes among successful populations, and 3) the transition probability matrix $\mathbf{P}^c$ of the Markov chain conditioned on reaching z before 0. The conditioned Markov chain serves two purposes. First, we can use it to directly simulate trajectories of successful populations, which is more efficient than simulating from the original Markov chain and then discarding unsuccessful runs. Second, we can use $\mathbf{P}^c$ to compute the expected number of generations that successful populations with or without Allee effect spend at each of the population sizes from 1 to $z - 1$ before reaching z, and the expected number of offspring per individual in successful populations with and without Allee effect. Thereby we characterised the population dynamics of successful populations with and without Allee effect. These computations are described in detail in Appendix 3.1. In the case of multiple introduction events, we simulated from the original Markov chain and discarded unsuccessful runs.

Given a successful population size trajectory $N_0, N_1, \ldots, N_{T_z}$, we then simulated the genealogies of a sample of $n_s = 10$ individuals genotyped at both copies of $n_l = 10$ freely recombining loci. We constructed the genealogies by tracing the sampled lineages back to their their most recent common ancestor (see Appendix 3.2 for details). These simulations are based on the assumption that each individual in the offspring generation is formed by drawing two parents independently and with replacement from the parent population. Equivalently, we could assume that each individual is the mother of a Poisson-distributed number of offspring with mean $\lambda(N_t)$ and that the father of each offspring individual is drawn independently and with replacement from the population. Our algorithm for the sim-

ulation of genealogies is a discrete-time version of the ancestral recombination graph (see e.g. Griffiths, 1991; Griffiths & Marjoram, 1997; Wakeley, 2009, Chapter 7) with a few modifications to better represent the genetics of very small populations. For each simulation run, we stored the average pairwise coalescence time G_2 between sampled chromosomes. To compute the expected proportion of variation from the source population that is maintained in the newly founded population, we divided G_2 by $2k_0$, the expected coalescence time for two lineages sampled from the source population.

We implemented all simulations in C++ (Stoustrup, 1997), compiled using the g++ compiler (gcc.gnu.org, 2012, version 4.7.2), and relied on the boost library (version 1.49) for random number generation (boost.org, 2013). We used R (R Development Core Team, 2011, version 2.14.1) for all other numerical computations and for data analysis.

Results

(a) Shift towards larger founder sizes

To compare the demography of successful populations with and without Allee effect, we first examine the distribution of their founder population sizes. These success-conditioned distributions (see Appendix 3.1 for how to compute them) differ from the original distribution because the success probability is higher for some founder population sizes than for others. Without Allee effect, small populations can still go extinct by chance, but this quickly becomes very unlikely as the founder population size increases (see Dennis, 2002, and figure 3.7). Thus, there is a shift towards larger founder population sizes in the conditioned distribution, but this shift is only noticeable for very small average founder sizes (figure 3.2a). With Allee effect, the success probability is overall lower, even above the critical population size, and has a sigmoid shape with a sharp increase around the critical size (see Dennis, 2002, and figure 3.7). Consequently, the conditioned distribution of founder population sizes is more strongly shifted to larger population sizes than without Allee effect (figure 3.2). This shift is particularly strong if the mean of the original distribution is small compared to the critical population size (figure 3.2a). As the mean founder size approaches the critical population size and a larger proportion of populations is successful (see figure 3.7), the shift becomes smaller (figure 3.2b,c).

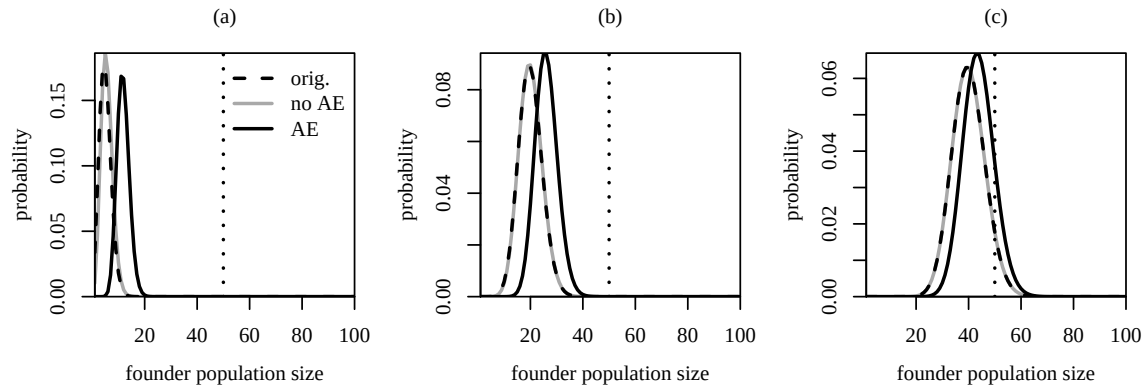

Figure 3.2: Success-conditioned distributions of founder population sizes with Allee effect (AE, solid black lines) and without (no AE, solid grey lines). The original distribution (dashed) is Poisson with mean 5 (a), 20 (b), or 40 (c) and is almost indistinguishable from the the conditioned distribution without Allee effect in B and C. The dotted vertical line indicates the critical size for Allee-effect populations. Note the differences in the scale of the y-axes. $r = 0.1$.

(b) Dynamics of successful populations

Upon reaching the target population size z, a successful Allee-effect population has on average spent fewer generations at small population sizes than a successful population that did not have to overcome an Allee effect (figure 3.3), particularly if the founder population size is small compared to the critical population size (figure 3.3a,b). Thus, although the average Allee-effect population declines at small population sizes (see figure 3.1), those populations that successfully overcome the critical population size must have grown very fast in this population-size range. Allee-effect populations, however, spend more time at larger population sizes than populations without Allee effect (figure 3.3). Note that the small peak figure 3.3a and the kink in figure 3.3b are due to the fact that the population necessarily spends some time around its founder population size.

(c) Population genetic consequences

We have now seen two ways in which the Allee effect modifies the demography of successful populations: it shifts the distribution of founder population sizes and it affects the time they spend in different population-size ranges. In this section, we examine the separate and combined effect of these two features on levels of genetic diversity. Our quantity of interest is the expected proportion of genetic variation from the source population that is maintained

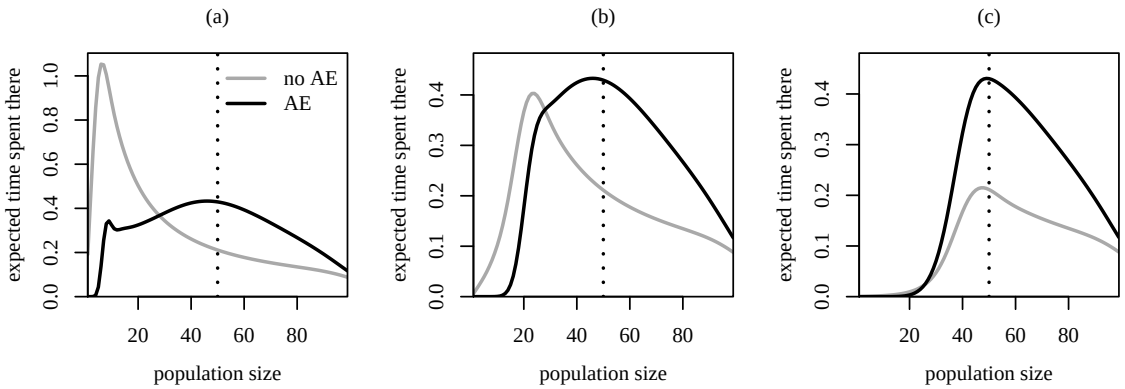

Figure 3.3: The expected number of generations that successful populations spend at each of the population sizes from 0 to $z-1$ before reaching population size z (here 100). The initial population sizes are 5 in (a), 20 in (b), and 40 in (c). The grey lines represent population dynamics conditioned on success in the absence of an Allee effect (no AE) whereas the black lines represent the conditioned population dynamics with Allee effect (AE) and a critical population size of 50 (indicated by dotted vertical line). Note the differences in the scale of the y-axes. $r = 0.1$.

by the newly founded population when it reaches size z. For different values of the growth rate parameter r, we compare four sets of successful populations (figure 3.4) representing all possible combinations of a founder-size distribution with or without Allee effect (solid and dashed lines in figure 3.4, corresponding to black and grey lines in figure 3.2, respectively) and subsequent population dynamics with or without Allee effect (black and grey lines in figure 3.4, corresponding to black and grey lines in figure 3.3, respectively).

There are three comparisons to be made in each subplot of figure 3.4. We first focus on figure 3.4b where the growth rate parameter r is the same as in figures 3.1–3.3. We first compare populations with the same dynamics but different distributions of founder population sizes (dashed vs. solid grey lines and dashed vs. solid black lines) and observe that those whose founder population size was drawn from the Allee-effect distribution maintained more genetic variation. This increase was strong for small mean founder population sizes and became weaker with increasing mean founder population size, in accordance with the lessening shift in the conditioned distribution of founder population sizes (see figure 3.2). Second, among populations that share the founder-size distribution but differ in their population dynamics (black dashed vs. grey dashed lines and black solid vs. grey solid lines), those with Allee-effect dynamics maintained more diversity at small founder population sizes, but less diversity for large founder population sizes.

Finally, the biologically meaningful comparison is between successful populations with

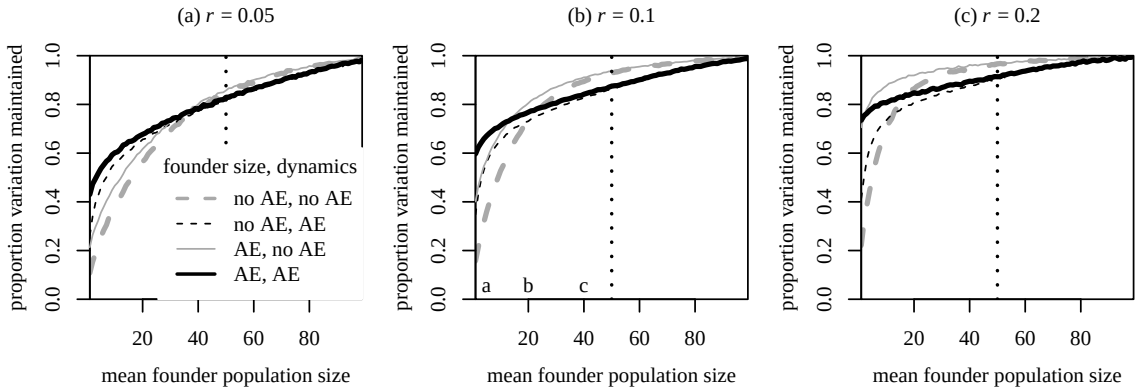

Figure 3.4: Average proportion of genetic variation from the source population that is maintained by an introduced population upon reaching size z. The subplots differ in the value of the growth rate parameter r. The values on the x-axes correspond to the mean of the original founder-size distribution. In each subplot, the four displayed scenarios differ in the underlying demography and represent all possible combinations of success-conditioned founder-size distributions either with Allee effect (solid lines) or without (dashed lines) and success-conditioned population dynamics either with Allee effect (black lines) or without (grey lines). Thick lines correspond to the biologically meaningful scenarios with Allee effect (AE, AE) and without (no AE, no AE), whereas thin lines represent combinations that have no direct biological interpretation but help us to decompose the genetic consequences of the Allee effect (no AE, AE and AE, no AE). The letters a, b, and c in subplot (b) refer to the subplots in figures 3.2 and 3.3, where we examined for $r = 0.1$ and the respective (mean) founder population sizes how the Allee effect influences the conditioned distribution of founder population sizes and the conditioned population dynamics. The critical size for Allee-effect populations ($a = 50$) is indicated by a dotted vertical line. Each point represents the average over 20,000 successful populations. Across all points in the plots, standard errors were between 0.0009 and 0.0020, and standard deviations between 0.141 and 0.274.

an Allee effect in both aspects of their demography (black solid lines) and successful populations without any Allee effect (grey dashed lines). This comparison reveals the strong and population-size dependent genetic consequences of the Allee effect: For small mean population sizes, successful populations with Allee effect in figure 3.4b maintained up to 3.8 times more genetic variation than populations without Allee effect. For mean population sizes close to the critical population size, on the other hand, Allee-populations maintained up to 6.6 % less genetic variation. Figures 3.4a and c show the corresponding results for a smaller and a larger growth rate parameter, respectively. For the smaller growth rate parameter, the Allee effect has a positive effect on genetic diversity over a wider range of mean founder population sizes (figure 3.4a), whereas for a higher growth rate parameter the Allee effect starts to have a negative effect already at relatively small mean founder population sizes (figure 3.4c). The results in figure 3.4 are based on average pairwise coalescence times, a measure related to the average number of pairwise differences in a sample. Results based on the average total length of genealogies were qualitatively similar (see Appendix 3.3).

(d) Multiple introductions

Populations with Allee effect maintained a larger proportion of genetic variation than did populations without Allee effect if the number of individuals introduced per event was smaller than the critical population size (figure 3.5a). In this parameter range, successful populations with Allee effect had received more introduction events than successful populations without Allee effect (figure 3.5b). Since in the case of multiple migrations the population can go temporarily extinct, not all introduction events necessarily contribute to the genetic diversity in the sample. However, for small founder population sizes, lineages sampled from an Allee-effect population also had a smaller probability to trace back to the same introduction event than lineages sampled from a population without Allee effect (figure 3.5c). If a single introduction event was sufficient to overcome the critical population size, there was no noticeable difference between populations with and without Allee effect, neither in the amount of genetic variation maintained nor in the number of introduction events they received.

(e) Estimating the critical population size from genetic data

The results in the last sections have shown that the Allee effect can have substantial impact on the expected amount of genetic variation in a recently founded population. However, due

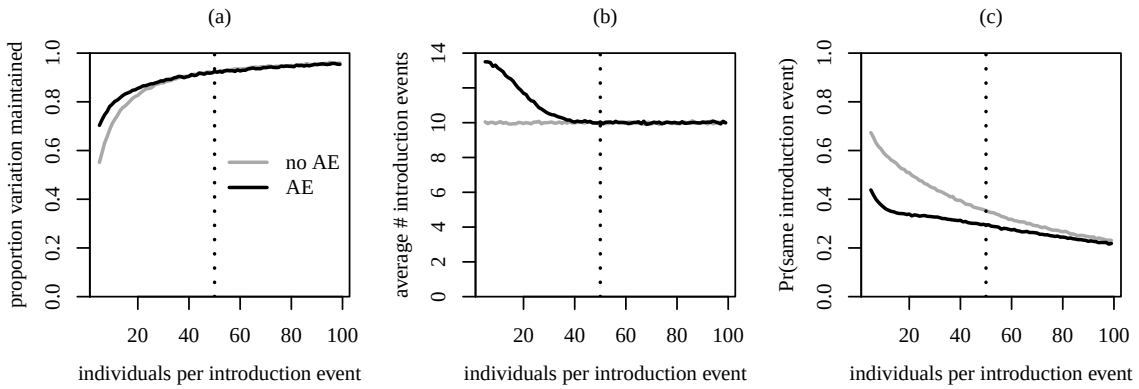

Figure 3.5: Genetic consequences of the Allee effect in the case of multiple introduction events. (a) Proportion of variation maintained by populations with Allee effect (AE) and without (no AE). Standard deviations were between 0.155 and 0.160 and standard errors between 0.0010 and 0.0012. (b) Average number of introduction events that happened in successful simulation runs. (c) Probability that two lineages in the sample trace back to the same introduction event. Allee-effect populations had a critical population size of 50, as indicated by a dotted vertical line. The migration probability per generation was 0.05. Each point represents the average over 20,000 successful populations. $r = 0.1$.

to stochasticity in the population dynamics and genetics, the associated standard deviations are so large that there always is considerable overlap between the underlying distributions with and without Allee effect. Using Approximate Bayesian Computation (ABC), a flexible statistical framework for simulation-based parameter estimation (Beaumont, 2010; Csilléry *et al.*, 2010, see Appendix 3.4 for the detailed methodology), we explored under what conditions it would be feasible to infer the critical population size from genetic data. We found that it is indeed possible to obtain reasonably accurate estimates of the critical population size, but only if we have information from sufficiently many independent replicates of the process, for example genetic data from several populations that have independently colonised a number of ecologically similar locations (figures 3.6 and 3.12).

Discussion

Our results indicate that the Allee effect strongly influences the expected amount of genetic diversity in a population that recently established from a small founder population size. In the case of a single introduction event, we can attribute this influence to the joint action of three mechanisms: 1) Compared to other successfully established populations, those that have overcome an Allee effect tend to derive from larger founder populations and hence

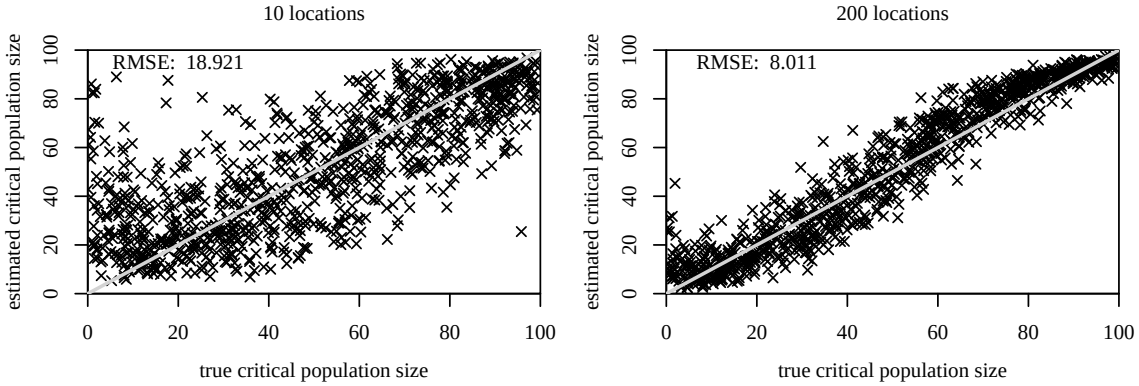

Figure 3.6: Estimated vs. true values of the critical population size for either 10 or 200 independent locations. On the diagonal grey line, the estimated critical population size is equal to the true one. The value in the upper left corner of each plot is the root mean squared error (RMSE) across the 1000 data sets.

start on average with more genetic diversity. 2) To successfully overcome the critical population size, small Allee-effect populations must grow very fast initially. Therefore, they spend fewer generations in the range of population sizes where genetic drift is strongest, which leads to an increase in genetic diversity relative to populations without Allee effect. 3) Successful Allee-effect populations experience a time lag in population growth around and above the critical population size, leading to increased opportunity for genetic drift and thus a negative effect on genetic diversity. The first and—to some extent—the third mechanism have been suggested before (see Kramer & Sarnelle, 2008; McCormick *et al.*, 2010). In this study, we have clarified the role of the third mechanism and first described the second mechanism in the context of the Allee effect.

Taken together, the second and third mechanism suggest a peculiar relationship between the original population growth rate and the growth rate among successful populations: Successful populations that are originally expected to decline rapidly (Allee effect-populations substantially below the critical size) grow the fastest, followed by those populations that are expected to increase moderately (populations without Allee effect). The slowest-growing populations are those that are expected to weakly increase or decrease (Allee-effect populations around the critical size). In summary, the per-capita population growth rate conditioned on success (see figure 3.8) seems to depend more on the absolute value of the original growth rate, $(E[N_{t+1}]/N_t) - 1$, than on its sign, a phenomenon that is also present in simpler models (see Appendix 3.6 for an example from diffusion theory). Thus, if we wish to predict the population genetic consequences of the Allee effect, it is not sufficient to know

the critical population size, but it may be even more important to determine the absolute value of the average-per capita growth rate at small population sizes.

As the mean founder population size increases, the two mechanisms leading to an increase in diversity (1 and 2) become weaker, whereas the mechanism leading to a decrease in diversity (3) becomes stronger. Therefore, the Allee effect appears to have a positive influence on levels of genetic diversity if typical founder population sizes are small, but a negative effect for large mean founder population sizes. The mean founder population size at which the direction of the effect changes depends on the magnitude of the growth rate parameter r. In the case of multiple introduction events, successful populations that have overcome an Allee effect tend to go back to more introduction events than do successful populations without Allee effect, a fourth mechanism that may tip the balance of the genetic consequences of the Allee effect into the positive direction. Exceptionally high levels of genetic diversity caused by Allee effects may contribute to explaining why established alien or invasive populations often harbour a large amount of genetic diversity relative to their source populations (Roman & Darling, 2007) although they supposedly established from small numbers.

As we have seen, the genetic consequences of the Allee effect can be used to estimate the critical population size from genetic data. We conducted our analysis with SNP data in mind, but with different choices of summary statistics other types of genetic data could also be accommodated. To achieve reasonable accuracy, however, we would need independent data from many different locations. Since we found magnitude and direction of the Allee effect's influence to be very context-dependent, it would also be important to know the other demographic parameters fairly well in order to be able to infer the critical population size from genetic data. It could also be worthwhile to perform a joint analysis combining genetic data with relevant ecological information, e.g. on propagule pressure and establishment success (Leung *et al.*, 2004). As demonstrated by previous studies that addressed other questions in invasion biology with a combination of genetic and ecological data (e.g. Estoup *et al.*, 2010), Approximate Bayesian Computation (ABC) provides a flexible statistical framework for such a task.

Even if it is difficult to detect an Allee effect in genetic data from a single population, neglecting its presence might affect the inference of other demographic parameters such as founder population size, growth rate, and time since the founding event. We explored this possibility in Appendix 3.5, but found no consistent differences in the quality of parameter estimation between populations with and without Allee effect. In both cases, the quality of the inference was rather poor, indicating that the stochastic dynamics in the true model

posed a greater challenge to parameter inference than did the Allee effect itself.

Stochastic population models such as the one in this study are not only characterised by their average behaviour, but also by the stochastic variability among outcomes. This seems to be particularly important for the genetic consequences of the Allee effect because of several reasons. First, the successful establishment of populations whose size is initially below the critical population size would not be possible in a deterministic model; it requires at least some variability. Second, the extent to which the population dynamics conditioned on success can deviate from the original population dynamics should also depend on the amount of variability. Third, even for a given demographic history, the amount of genetic drift depends on one source of variability, namely that in offspring number among individuals. In this study, we have worked with the standard assumption of Poisson-distributed offspring numbers. However, there is evidence that many natural populations do not conform to this assumption (Kendall & Wittmann, 2010). Especially in small populations with Allee effect, we would expect more variation in offspring number because many individuals do not encounter a mating partner (Kramer *et al.*, 2009), whereas those that do can exploit abundant resources and produce a large number of offspring. In a second paper (Wittmann *et al.*, 2013d, Chapter 4), we therefore investigate how the genetic consequences of the Allee effect depend on the distribution of the number of offspring produced by individuals or families. Since the magnitude of the growth rate parameter r affects the relative strength of deterministic and stochastic forces, our results in Chapter 4 (Wittmann *et al.*, 2013d) will shed additional light on the role of r for the genetic consequences of the Allee effect.

Acknowledgements

We would like to thank Raphael Gollnisch and Shankari Subramaniam for assistance with simulations and Pablo Duchén for sharing ABC scripts. MJW is grateful for a scholarship from the Studienstiftung des deutschen Volkes.

Appendix 3.1 The conditioned Markov chain and its properties

This section explains how to obtain the transition probability matrix of the Markov chain conditioned on the event that the population reaches size z before going extinct (reaching size 0), i.e. conditioned on the event $T_z < T_0$. We also explain how to derive further properties of the conditioned Markov chain. We first restrict our Markov chain to the states $0, 1, \ldots, z-1, z$, where 0 and z are absorbing states and $1, \ldots, z-1$ are transient, that is the Markov chain will leave them at some time. We can write the transition probability matrix of the original Markov chain as

$$\mathbf{P} = \begin{pmatrix} \mathbf{Q} & \mathbf{R} \\ \mathbf{0} & \mathbf{I} \end{pmatrix}, \tag{3.3}$$

where $\mathbf{Q}$ is a $(z-1) \times (z-1)$ matrix representing the transitions between transient states, $\mathbf{R}$ is a $(z-1) \times 2$ matrix with the transition probabilities from the transient states to the absorbing states z (first column) and 0 (second column), $\mathbf{0}$ is a $2 \times (z-1)$ matrix filled with zeros, and $\mathbf{I}$ is an identity matrix (in this case 2×2).

Following Pinsky & Karlin (2010), we then computed the fundamental matrix $\mathbf{W} = (\mathbf{I} - \mathbf{Q})^{-1}$. W_{ij} gives the expected number of generations a population starting at size i spends at size j before reaching one of the absorbing states. This matrix operation is based on first-step analysis, i.e. on a decomposition of expected quantities according to what happens in the first step (see Pinsky & Karlin, 2010, Section 3.4 for details).

The probabilities of absorption in either of the two absorbing states can then be computed as $\mathbf{U} = \mathbf{WR}$. The first column of $\mathbf{U}$ contains the success probabilities $\Pr(T_z < T_0 | N_0 = i)$ shown in figure 3.7. For a given original distribution of founder population sizes (given by the probabilities $\Pr(N_0 = n)$ for different founder population sizes n), we used the success probabilities together with Bayes' formula to compute the distribution of founder population sizes among successful populations:

$$\Pr(N_0 = n | T_z < T_0) = \frac{\Pr(N_0 = n) \cdot \Pr(T_z < T_0 | N_0 = n)}{\sum_{i=1}^{\infty} \Pr(N_0 = i) \cdot \Pr(T_z < T_0 | N_0 = i)}. \tag{3.4}$$

The resulting distributions are shown in figure 3.2.

Using the success probabilities and Bayes' formula, we then computed the transition

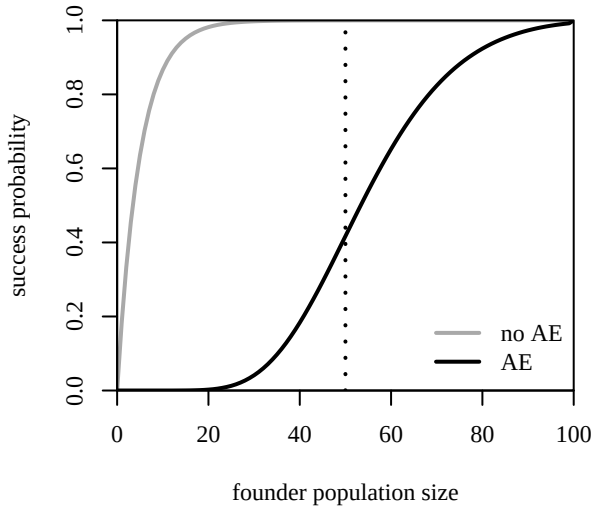

Figure 3.7: Success probabilities $\Pr(T_{100} < T_0)$ without Allee effect (no AE, grey line) or with an Allee effect (AE, black line) and critical size $a = 50$ (indicated by dotted vertical line). $k_1 = 1000, r = 0.1$.

probabilities of the Markov chain conditioned on $T_z < T_0$:

$$Q^c_{ij} = \Pr(N_{t+1} = j | N_t = i, T_z < T_0) = \frac{Q_{ij} \cdot \Pr(T_z < T_0 | N_0 = j)}{\Pr(T_z < T_0 | N_0 = i)}. \tag{3.5}$$

As z is the only absorbing state of this new Markov chain, the full transition probability matrix is

$$\mathbf{P^c} = \begin{pmatrix} \mathbf{Q^c} & \mathbf{R^c} \\ \mathbf{0} & 1 \end{pmatrix}, \tag{3.6}$$

where $\mathbf{R^c}$ contains the transition probabilities from the transient states to z. These probabilities are chosen such that each row sums to 1. In this case, $\mathbf{0}$ stands for a $1 \times (z-1)$ vector filled with zeros. We used this transition probability matrix to simulate the population dynamics conditioned on success.

To further study the conditioned Markov chain, we computed its fundamental matrix $\mathbf{W^c} = (\mathbf{I} - \mathbf{Q^c})^{-1}$. W^c_{ij} gives the number of generations a population starting at size i spends at size j before reaching z, conditioned on reaching z before going extinct. These are the values shown in figure 3.3. Note that in these plots we did not include the first

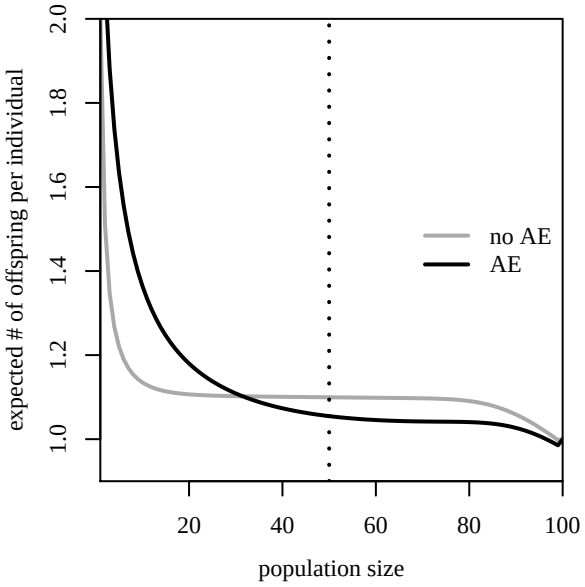

Figure 3.8: The expected number of surviving offspring per individual in successful populations (see equation (3.7)) as a function of the current population size without (no AE, grey line) or with an Allee effect (AE, black line) and critical size $a = 50$ (indicated by dotted vertical line). $k_1 = 1000, r = 0.1$.

generation, which the population necessarily spends at its founder size.

We also computed the expected number of surviving offspring per individual at population size i under the conditioned population dynamics (figure 3.8):

$$\frac{1}{i}\sum_{j=1}^{z} j \cdot P_{ij}^{c}. \tag{3.7}$$

This is an approximation because our Markov chain is restricted to population sizes up to z whereas actual populations would be able to grow beyond z. However, in the range of population sizes that is most relevant for our study, i.e. at small and intermediate population sizes, equation (3.7) should give an accurate approximation of the expected number of surviving offspring per individual.

Appendix 3.2 Details on the simulation of genealogies

The genealogies are constructed by tracing the ancestry of the sampled genetic material backwards in time. One special feature of our algorithm is the possibility of multiple and simultaneous mergers. This means that in one generation several coalescent events can happen and each of them can possibly involve more than two lineages. Such events are very rare in large populations as assumed by standard coalescent models but they can be quite frequent in small populations as we are considering here. Another special feature is that the genealogical process takes into account explicitly that individuals are diploid and biparental and thus avoids logical inconsistencies that may occur when independently simulating the genealogies at different loci (Wakeley *et al.*, 2012). However, this realism comes at a computational cost and in cases where we are only interested in average levels of genetic diversity, i.e. for the analysis underlying figures 3.4, 3.5, and 3.10 we resorted to independently simulating the genealogies at the different loci.

The current state of the ancestry is defined by a set of lineage packages for each population (source population and newly founded population). Such a lineage package contains all the genetic material that is travelling within the same individual at that time point. It has two sets of slots, one set for each genome copy. Each set has a slot for each locus. If the genetic material at a certain locus and genome copy is ancestral to the sample, the slot is occupied by a node, otherwise it is empty.

The ancestral history starts with $2 \cdot n_s$ lineage packages in the newly founded population. Initially all slots in the lineage packages are occupied by nodes. From there, the ancestry is modelled backwards in time until at each locus there is just one node left. Given the state of the ancestry in generation t, the state in generation $t-1$ is generated as follows: Backward in time, each generation starts with a migration phase (figure 3.9). All lineage packages that are currently in the newly founded population choose uniformly without replacement one of the Y_t migrants from the source population, or one of the $N_t - Y_t$ residents. Note that in our simulations with a single founding event, $Y_0 = N_0$ and $Y_t = 0$ for all $t > 0$, whereas in the case of multiple introductions, Y_t can be positive also at $t > 0$. According to their choice in this step, lineage packages either remain in the newly founded population or are transferred to the source population.

Then each lineage package splits into two because the two genome copies (sets of slots) each derive from a possibly different parent (see figure 3.9). Lineage packages that do not contain ancestral material are discarded immediately. For each of the remaining lineage packages, recombination is implemented by independently constructing a stochastic map

$R : \{1, \ldots, n_l\} \to \{0, 1\}$ such that

$$R(1) = \begin{cases} 0 & \text{with probability } \frac{1}{2} \\ 1 & \text{with probability } \frac{1}{2} \end{cases} \tag{3.8}$$

and then

$$R(n+1) = \begin{cases} R(n) & \text{with probability } 1 - \rho_n \\ 1 - R(n) & \text{with probability } \rho_n \end{cases} \tag{3.9}$$

is drawn recursively for $n \in \{1, \ldots, n_l - 1\}$. The recombination probability ρ_n between loci n and $n + 1$ was 0.5 for all analyses in this study. A node at locus l in the new lineage package is placed into the first genome copy if $R(l) = 0$ and into the second genome copy if $R(l) = 1$.

After each lineage package underwent splitting and recombination, all resulting lineage packages uniformly pick one of the N_{t-1} or k_0 individuals as ancestor, depending on whether they are in the newly founded or in the source population, this time with replacement (see figure 3.9). Lineage packages that chose the same ancestor are merged. If there is more than one node at the same genome copy and slot, a coalescent event takes place.

Because genetic drift is strong in small populations, many pairs of lineages will already encounter their common ancestor within the newly founded population. The lineages that did not coalesce until time 0 must all be in the source population which is assumed to be of constant size k_0 at all times. To efficiently simulate the genealogical process of the remaining lineages, we follow one of two procedures: As long as there is still lineage packages that carry more than one node or if the number of pairs of remaining lineages is larger than $k_0/10$, we continue as before, going backwards generation-by-generation. Each lineage package can split due to recombination and merge with others that choose the same ancestor. However, as the source population is large it would take a long time until all lineages find their most recent common ancestor (MRCA) and in most generations nothing would happen. Furthermore, nodes within the same lineage package typically become separated by recombination relatively fast. Thus whenever there is no lineage package with more than one node, we switch to a second and more efficient simulation mode: If n_{total} is the number of lineage packages, we draw the number of generations T until the next merger

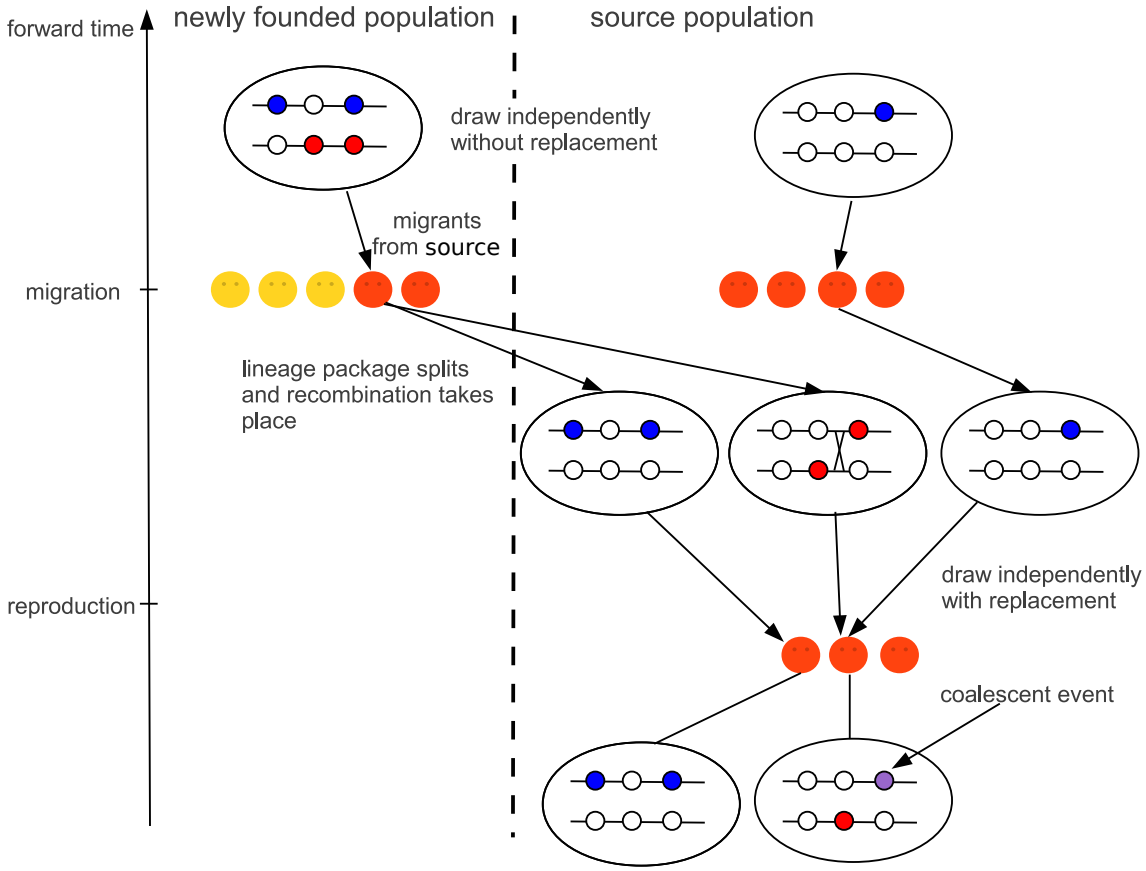

Figure 3.9: Illustration of the backward-in-time simulation of genealogies.

of two lineage packages from a geometric distribution with success probability

$$p_{merge} = \frac{\binom{n_{total}}{2}}{k_0} \tag{3.10}$$

and update the current time to $t - T$. Then one of the $\binom{n_{total}}{2}$ pairs of lineage packages is picked at random and the lineage packages are merged. If the two lineage packages have their node at the same slot, a coalescent event happens and we continue in the efficient simulation mode. If the nodes are at different slots we again have a lineage package with more than one node and we switch back to the more accurate simulation mode. Note that the last recombination event in the accurate simulation mode ensures that each node has a 50 % chance to be in the first or second genome copy. This leads to a 50 % chance of coalescence if two lineage packages with a node at the same locus merge. Thus, there is no need to implement recombination in the efficient simulation mode. This efficient simulation mode excludes multiple and simultaneous mergers, events that should be very rare for a reasonably large source population size k_0.

We switch between the two simulation modes until eventually there is only one node left at each locus, the MRCA of all sampled genetic material at the respective locus. Throughout the simulation, we store all information needed to provide the topology and branch lengths (in number of generations) for the genealogies at each locus.

Appendix 3.3 Results based on total length of the genealogy

In the main text, we use average pairwise coalescence times to assess genetic diversity. Here we show the corresponding results for the average total length of the genealogy G_{total}, a measure related to the number of segregating sites or the number of alleles in a sample. To measure the proportion of variation maintained, we divided G_{total} by $4k_0 \cdot \sum_{i=1}^{2n_s-1} \frac{1}{i}$, the expected total length of the sample genealogy if all lineages would have been sampled in the source population (Wakeley, 2009, p. 76). The results (figure 3.10) were qualitatively similar to the results based on average pairwise coalescence times (see figure 3.4), except that the proportion of variation maintained more slowly approached one with increasing founder population size.

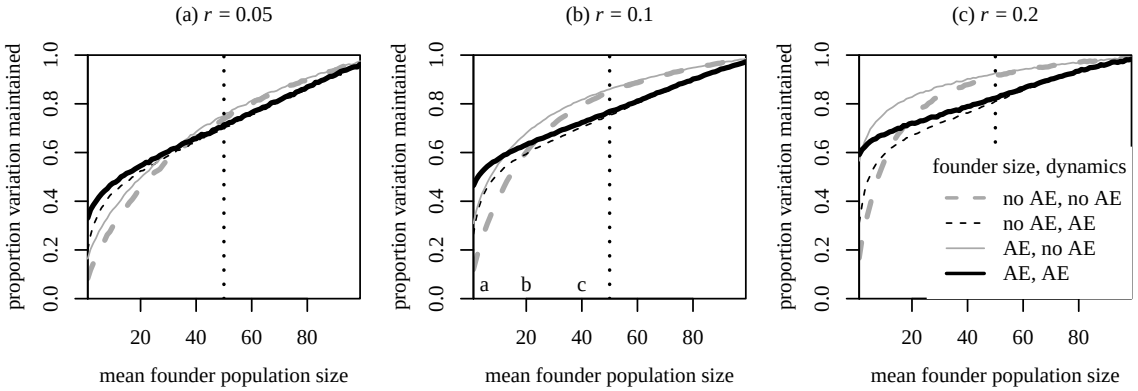

Figure 3.10: Average proportion of genetic variation maintained (based on the average total length of sample genealogies) as a function of the mean of the original (unconditioned) founder population size distribution. The four scenarios differ in the underlying demography and represent all possible combinations of success-conditioned founder-size distributions either with Allee effect (solid lines) or without (dashed lines) and success-conditioned population dynamics either with Allee effect (black lines) or without (grey lines). Thick lines correspond to the biologically meaningful scenarios with Allee effect (AE, AE) and without (no AE, no AE), whereas thin lines represent combinations that have no direct biological interpretation but help us to decompose the genetic consequences of the Allee effect (no AE, AE and AE, no AE). The letters a, b, and c in subplot (b) refer to the subplots in figures 3.2 and 3.3, where we examined for the respective (mean) founder population sizes how the Allee effect influences the conditioned distribution of founder population sizes and the conditioned population dynamics. The critical size for Allee-effect populations ($a = 50$) is indicated by a dotted vertical line. Each point represents the average over 20,000 successful populations. Across all points, standard errors were between 0.0007 and 0.0016, and the corresponding standard deviations between 0.103 and 0.221.

Appendix 3.4 Methodology for estimating the critical population size

We generated 1000 pseudo-observed data sets and 100,000 simulated data sets, each with independent introductions to 200 locations. The critical population sizes were drawn from a uniform distribution on [0,100]. We fixed the other parameters of the population dynamics ($k_0 = 10,000, k_1 = 1000, r = 0.1$) and assumed them to be known with certainty. We further assumed that the original distribution of founder population sizes was Poisson with mean 20, and sampled the founder population sizes independently for each location from the conditioned distribution of founder population sizes for the respective critical population size. Given the selected founder population size, we simulated the population dynamics at

each location from the conditioned Markov chain until the population reached size 200, i.e. twice the largest possible critical population size.

At this point, we sampled $n_l = 10$ individuals at both genome copies, resulting in a sample of 20 chromosomes from a given location. We generated genealogies for 10 freely recombining loci. To obtain a more differentiated picture of patterns of genetic variation and capture as much information as possible, we did not use the average pairwise coalescence times or total lengths of the genealogy as before. Instead, for $i \in 1, 2, \ldots, 19$ we took the combined length of all branches B_i that have i descendants in the sample. Using these branch lengths and assuming that the number of mutations on a branch of length b is Poisson-distributed with parameter $\mu \cdot b$, we estimated the mean and variance across loci of the entries of the site-frequency spectrum (SFS) ξ_i, i.e. the number of mutations that appear in i chromosomes in the sample, as

$$\hat{\mathbf{E}}[\xi_i] = \mu \cdot \bar{B}_i \tag{3.11}$$

and, using the law of total variance,

$$\hat{\mathbf{Var}}[\xi_i] = \hat{\mathbf{E}}\left[\mathbf{Var}[\xi_i|B_i]\right] + \hat{\mathbf{Var}}\left[\mathbf{E}[\xi_i|B_i]\right] = \mu \cdot \bar{B}_i + \mu^2 \cdot s^2(B_i), \tag{3.12}$$

where the $\bar{B}_i$ are the average branch lengths across the n_l loci and the $s^2(B_i)$ are the corresponding empirical variances. We assumed $\mu = 0.001$. Note that we do not take into account variability introduced by the mutation process because we assume that we have enough loci to estimate the means and variances of the SFS entries with reasonable accuracy.

We further summarised the data for each SFS entry $i \in 1, 2, \ldots, 19$ by computing the averages and empirical standard deviations of the quantities in eqs. (3.11) and (3.12) across locations. To investigate how the quality of the estimation depends on the number of independent locations available, we took into account either only 10, 25, 50, 100, or all 200 of them to compute these statistics. Using the pls script from abctoolbox (Wegmann *et al.*, 2010) and the pls package in R (Mevik & Wehrens, 2007), we then conducted partial least squares regression on the first 10,000 simulated data sets to condense the information contained in the 76 summary statistics to a smaller number of components. To decide on the number of components, we examined plots of the root mean squared error of prediction (RMSEP) as a function of the number of components (figure 3.11). For none of the different numbers of locations did the RMSEP change substantially beyond 20 components. Thus, we decided to include 20 components as summary statistics for ABC.

We used these 20 PLS components as summary statistics for parameter estimation with

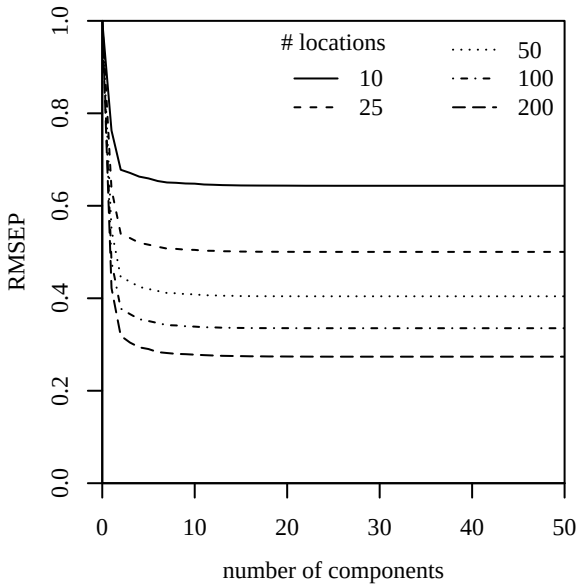

Figure 3.11: Root mean squared error of prediction (RMSEP) as a function of the number of PLS components for various numbers of locations. In no case did the RMSEP substantially change beyond 20 components. Thus, we decided to include 20 components as summary statistics for ABC.

the R package abc (Csilléry *et al.*, 2012). We chose a tolerance of 1 % and used the option "loclinear" implementing the local linear regression method Beaumont *et al.* (2002). To avoid estimated parameter values that fall outside the prior, we estimated $\ln(a/(100-a))$ and then back-transformed the estimated values. For each pseudo-observed data set, we thus used the 100,000 simulated data sets to approximate the posterior distribution of the critical population size given a uniform prior on [0,100]. For each data set, we stored the mean of the posterior, which we take as our point estimator, and the 50 % and 95 % credibility intervals. We observed that the quality of parameter inference improved with an increasing number of locations (figures 3.6 and 3.12). An examination of the percentage of pseudo-observed data sets for which the true parameter value falls into the respective 50 % or 95 % credibility interval suggests that ABC approximates Bayesian inference reasonably well in this case (figure 3.13).

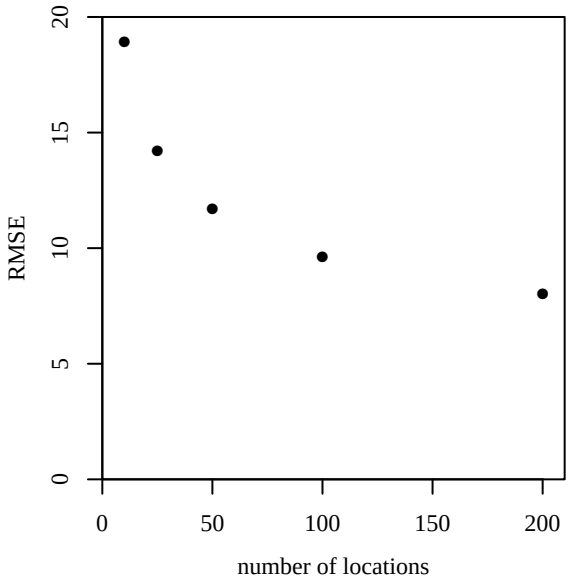

Figure 3.12: The root mean squared error (RMSE) of the estimated critical population size as a function of the number of independent locations used for the estimation.

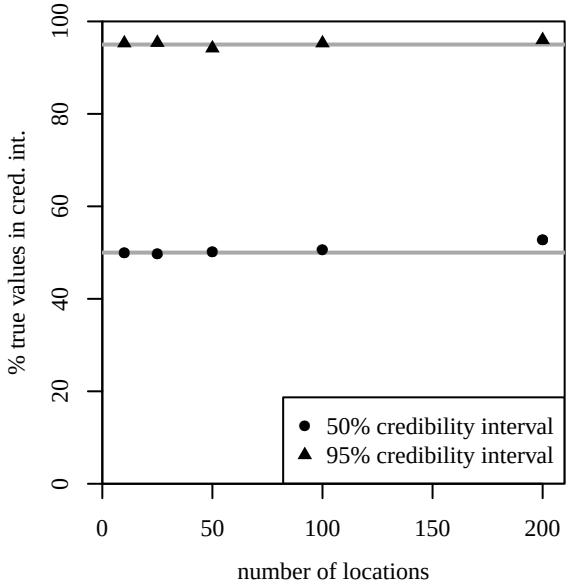

Figure 3.13: Percentage of true parameter values that fall within the 50% and 95% credibility interval, an indicator for how well Approximate Bayesian Computation approximates Bayesian inference. The grey lines are at 50% and 95%.

Appendix 3.5 Consequences of a neglected Allee effect

Using the ABC framework again, we explored the consequences of neglecting the Allee effect when estimating other demographic parameters: the founder population size N_0, the growth parameter r, and the number of generations since the founding event. We generated 2000 pseudo-observed data sets from our stochastic model, 1000 without Allee effect and 1000 with an Allee effect and a critical population size of 50. As the basis for estimation in ABC, we used 100,000 data sets that were simulated from a model without Allee effect. To also explore the consequences of neglecting stochasticity, we considered two versions of the model without Allee effect: our stochastic model with $a = 0$ and a modified version where we removed as much stochasticity as possible. That is, the population size in the next generation was not drawn from a Poisson distribution, but was set to $\mathbf{E}[N_{t+1}]$ if this value was an integer. Otherwise, we randomly set N_{t+1} to the next smallest or next largest integer with the respective probabilities chosen such that equation (3.1) was fulfilled.

The priors for the demographic parameters of interest were as follows:

$$\ln(N_0) \sim \text{unif}([\ln(5), \ln(80)]), \tag{3.13}$$

$$r \sim \text{unif}([0.01, 0.1]) \tag{3.14}$$

and

$$n_g \sim \text{unif}(\{20, \ldots, 500\}), \tag{3.15}$$

where unif stands for the uniform distribution. The other parameters were fixed: $k_0 = 10,000, k_1 = 1000, \mu = 0.001, n_s = 10$. For each data set, we retried simulating with the same parameter combination until we obtained a successful population with $N_{n_g} \geq n_s$. We generated 100 independent genealogies for samples of size n_s taken at time n_g and computed means and variances of the entries of the site-frequency spectrum as described in Appendix 3.4. Using partial least squares regression on the first 10,000 simulated data sets, we reduced this information to 20 components that served as summary statistics for ABC. As above, we used the R package abc (Csilléry *et al.*, 2012) with a tolerance of 1 % and the option "loclinear'.

In figure 3.14, we compare the quality of parameter estimation across the four possible

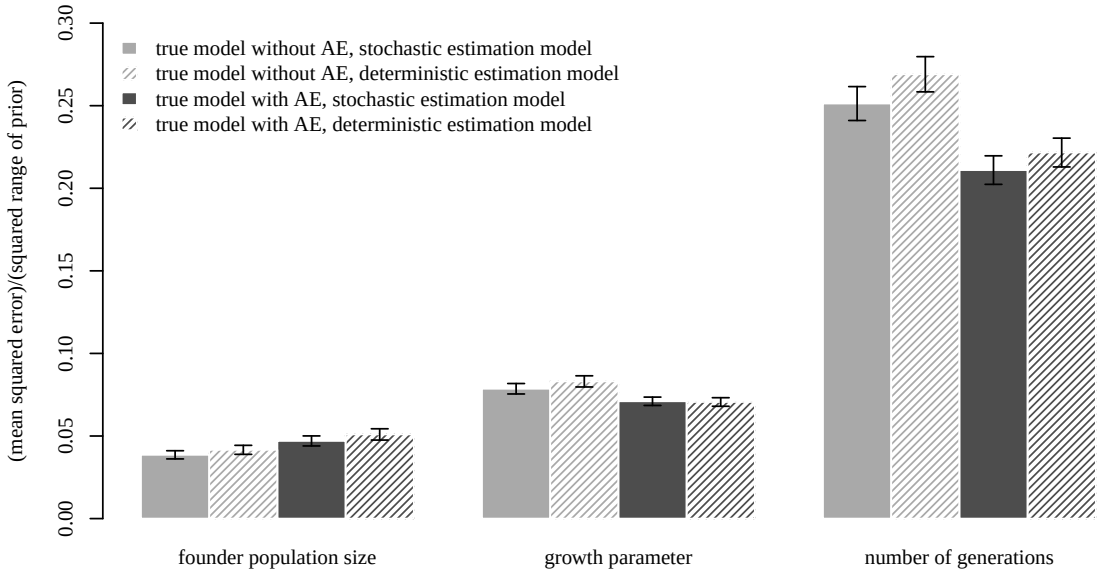

Figure 3.14: Mean squared error (MSE) $\pm$ its standard error, relative to the squared range of the prior for different demographic parameters in an Approximate Bayesian Computation analysis that neglects the Allee effect.

combinations of whether or not the true model includes an Allee effect and whether the model used for estimation was stochastic or deterministic. The differences in quality between the four combinations were not consistent across estimated parameters. Overall, the quality of the estimation was poor, with root mean squared errors of up to half the range of the corresponding prior. Note that these problems cannot only result from model misspecification since the case where the correct model was used (solid light grey bars in figure 3.14) also produced large errors. Thus, it appears that the amount of stochasticity in the model is so large as to prevent accurate parameter inference based on genetic data from a single population.

Appendix 3.6 A conditioned diffusion process

Our results in the main text indicate that the growth rate under the conditioned population dynamics depends mostly on the absolute value and not so much on the sign of the growth rate under the unconditioned population dynamics. For mathematically interested readers, we now explore a simple model where this fact can be proven easily. We consider a diffusion process on the interval $[0, 1]$ with constant infinitesimal mean $\mu(x) = \mu$ and constant infinitesimal variance $\sigma^2(x) = \sigma^2$. We will show that the associated diffusion process conditioned on hitting 1 before 0 is independent of the sign of μ and that its infinitesimal mean increases with $|\mu|$.

Our task is to compute the infinitesimal mean and variance of the conditioned diffusion process. Following the formulas on p. 263 in Karlin & Taylor (1981), the infinitesimal mean of the conditioned diffusion process is

$$\mu^*(x) = \mu(x) + \frac{s(x)}{S(x)} \cdot \sigma^2(x), \tag{3.16}$$

where $S(x)$ is the scale function and $s(x)$ is its derivative. Using the definitions of these functions (e.g. Karlin & Taylor, 1981, p. 262) and plugging in the parameters of our diffusion, we obtain

$$s(x) = \exp\left(-\int_0^x \frac{2\mu(\eta)}{\sigma^2(\eta)} d\eta\right) = \exp\left(-\frac{2\mu x}{\sigma^2}\right) \tag{3.17}$$

and

$$S(x) = \int_0^x s(\eta) d\eta = \frac{\sigma^2}{2\mu} \cdot \left[1 - \exp\left(-\frac{2\mu x}{\sigma^2}\right)\right]. \tag{3.18}$$

Substituting eqs. (3.17) and (3.18) into equation (3.16), we obtain

$$\mu^*(x) = \mu \cdot \frac{\exp(2\mu x/\sigma^2) + 1}{\exp(2\mu x/\sigma^2) - 1} =: f(\mu), \tag{3.19}$$

a function that is symmetric about 0, i.e. $f(-\mu) = f(\mu)$, and increases with the absolute value of μ (figure 3.15). The variance $\sigma^{2*}(x)$ equals the original variance $\sigma^2(x)$.

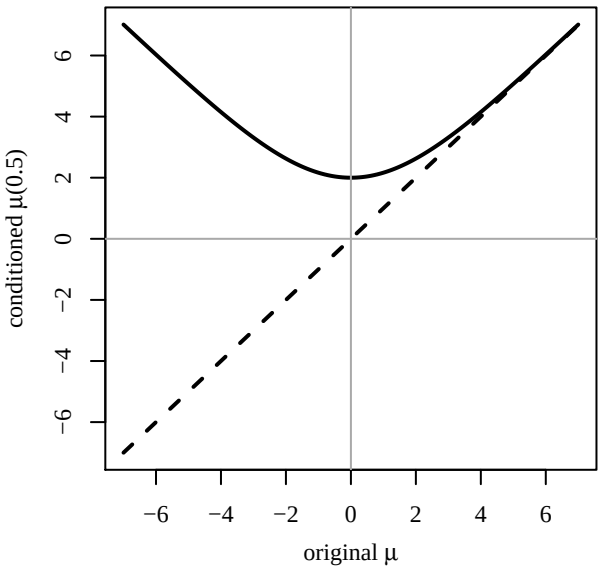

Figure 3.15: The infinitesimal mean of the conditioned diffusion process at 0.5 (solid line), i.e. in the middle of the interval, as a function of the infinitesimal mean of the original process. On the dashed line, the infinitesimal means of original and conditioned process would be equal.

Chapter 4

Population genetic consequences of the Allee effect and the role of offspring-number variation

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Abstract

A strong demographic Allee effect in which the expected population growth rate is negative below a certain critical population size can cause high extinction probabilities in small introduced populations. However, many species are repeatedly introduced to the same location and eventually one population may overcome the Allee effect by chance. With the help of stochastic models, we investigate how much genetic diversity such successful populations harbour on average and how this depends on offspring-number variation, an important source of stochastic variability in population size. We find that with increasing variability, the Allee effect increasingly promotes genetic diversity in successful populations. Successful Allee-effect populations with highly variable population dynamics escape rapidly from the region of small population sizes and do not linger around the critical population size. Therefore, they are exposed to relatively little genetic drift. We show that here—unlike in classical population genetics models—the role of offspring-number variation cannot be accounted for by an effective-population-size correction. Thus, our results highlight the importance of detailed biological knowledge, in this case on the probability distribution of family sizes, when predicting the evolutionary potential of newly founded populations or when using genetic data to reconstruct their demographic history.

Keywords: critical population size, family size, founder effect, genetic variation, invasive species, stochastic modelling

Introduction

The demographic Allee effect, a reduction in per-capita population growth rate at small population sizes (Stephens *et al.*, 1999), is of key importance for the fate of both endangered and newly introduced populations, and has inspired an immense amount of empirical and theoretical research in ecology (Courchamp *et al.*, 2008). By shaping the population dynamics of small populations, the Allee effect should also strongly influence the strength of genetic drift they are exposed to and hence their levels of genetic diversity and evolutionary potential. In contrast to the well-established ecological research on the Allee effect, however, research on its population genetic and evolutionary consequences is only just beginning (Kramer & Sarnelle, 2008; Hallatschek & Nelson, 2008; Roques *et al.*, 2012). In this study, we focus on the case where the average population growth rate is negative below a certain critical population size. This phenomenon is called a strong demographic Allee effect (Taylor & Hastings, 2005). Our goal is to quantify levels of genetic diversity in introduced populations that have successfully overcome such a strong demographic Allee effect. Of course, the population genetic consequences of the Allee effect could depend on a variety of factors, some of which we investigated in Chapter 3 (Wittmann *et al.*, 2013c). Here we focus on the role of variation in the number of offspring produced by individuals or pairs in the population.

There are several reasons why we hypothesise offspring-number variation to play an important role in shaping the population genetic consequences of the Allee effect. First, variation in individual offspring number can contribute to variability in the population dynamics and this variability influences whether and how introduced populations can overcome the Allee effect. In a deterministic model without any variation, for instance, populations smaller than the critical size would always go extinct. With an increasing amount of stochastic variability, it becomes increasingly likely that a population below the critical population size establishes (Dennis, 2002). Depending on the amount of variability, this may happen either quickly as a result of a single large fluctuation or step-by-step through many generations of small deviations from the average population dynamics. Of course, the resulting population-size trajectories will differ in the associated strength of genetic drift. Apart from this indirect influence on genetic diversity, offspring-number variation also directly influences the strength of genetic drift for any given population-size trajectory. In offspring-number distributions with large variance, genetic drift tends to be strong because the individuals in the offspring generation are distributed rather unequally among the individuals in the parent generation. In distributions with small variance, on the other hand,

genetic drift is weaker.

In Wittmann *et al.* (2013c), we have studied several aspects of the population genetic consequences of the Allee effect for Poisson-distributed offspring numbers, a standard assumption in population genetics. However, deviations from the Poisson distribution have been detected in the distributions of lifetime reproductive success in many natural populations. Distributions can be skewed and multimodal (Kendall & Wittmann, 2010) and, unlike in the Poisson distribution, the variance in the number of surviving offspring is often considerable larger than the mean, as has been shown for example for tigers (Smith & McDougal, 1991), cheetahs (Kelly *et al.*, 1998), and steelhead trout (Araki *et al.*, 2007). Several sources contribute to this variance, for example variation in environmental conditions, sexual selection, and predation. For several bird species, there is evidence that pairs individually optimise their clutch size given their own body condition and the quality of their territories (Högstedt, 1980; Davies *et al.*, 2012). Variation in offspring number may also depend on population size or density and thus interact with an Allee effect in complex ways. A mate finding Allee effect, for example, is expected to lead to a large variance in reproductive success among individuals (Kramer *et al.*, 2009) because many individuals do not find a mating partner and thus do not reproduce at all, whereas those that do find a partner can take advantage of abundant resources and produce a large number of offspring. In this study, we therefore investigate how the genetic consequences of the Allee effect depend on offspring-number variation. With the help of stochastic simulation models, we generate population-size trajectories and genealogies for populations with and without Allee effect and with various offspring-number distributions, both models with a smaller and models with a larger variance than the Poisson model.

Although probably only few natural populations conform to standard population genetic assumptions such as that of a constant population size and a Poisson-distributed number of offspring per individual, many populations still behave as an idealised population with respect to patterns of genetic variation (Charlesworth, 2009). The size of this corresponding idealised population is called the effective population size and is often much smaller (but can, at least in theory, also be larger) than the size of the original population, depending on parameters such as the distribution of offspring number, sex ratio, and population structure. Because of this robustness and the tractability of the standard population genetics models, it is common to work with these models and effective population sizes, instead of using census population sizes in conjunction with more complex and realistic models. For example, when studying the demographic history of a population, one might estimate the effective current population size, the effective founder population size etc. If one is inter-

ested in census population size, one can then use the biological knowledge to come up with a conversion factor between the two population sizes. Therefore, if we find differences in the genetic consequences of the Allee effect between different family size distributions but it is possible to resolve these differences by rescaling population size or other parameters, those differences might not matter much in practice. If, on the other hand, such a simple scaling relationship does not exist, the observed phenomena would be more substantial and important in practice. We therefore investigate how closely we can approximate the results under the various offspring-number models by rescaled Poisson models.

Methods

(a) Scenario and average population dynamics

In our scenario of interest, N_0 individuals from a large source population of constant size k_0 migrate or are transported to a new location. The average population dynamics of the newly founded population are described by a modified version of the Ricker model (see e.g. Kot, 2001) with growth parameter r, carrying capacity k_1, and critical population size a. Given the population size at time t, N_t, the expected population size in generation $t+1$ is

$$\mathbf{E}[N_{t+1}|N_t] = N_t \cdot \exp\left\{ r \cdot \left(1 - \frac{N_t}{k_1}\right) \cdot \left(1 - \frac{a}{N_t}\right) \right\}. \tag{4.1}$$

Thus, below the critical population size and above the carrying capacity, individuals produce on average less than one offspring, whereas at intermediate population sizes individuals produce on average more than one offspring and the population is expected to grow (figure 4.1). We compared populations with critical population size $a = a_{AE} > 0$ to those without Allee effect, i.e. with critical size $a = 0$. In all our analyses, the growth parameter r takes values between 0 and 2, that is in the range where the carrying capacity k_1 is a locally stable fixed point of the deterministic Ricker model and there are no stable oscillations or chaos (de Vries *et al.*, 2006, p. 29).

(b) Offspring-number models

Our goal was to construct a set of offspring-number models that all lead to the same expected population size in the next generation (equation (4.1)) but which represent a range of values for the variability in population dynamics and the strength of genetic drift c (ta-

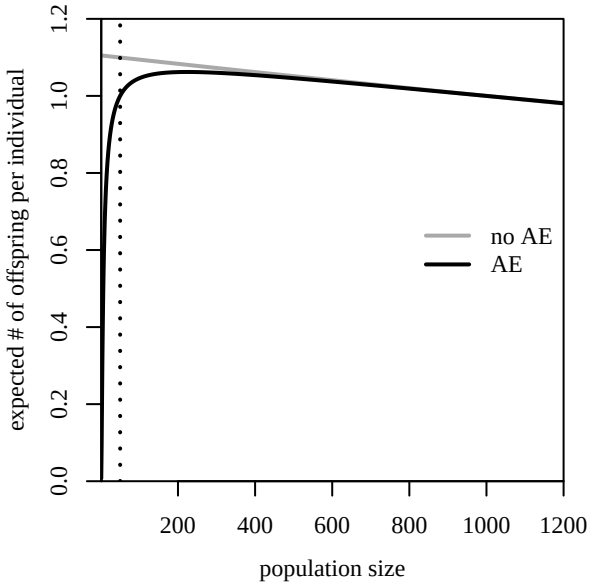

Figure 4.1: The expected number of surviving offspring per individual (see equation (4.1)) as a function of the current population size without Allee effect (no AE, grey line) or with an Allee effect (AE, black line) and critical size $a = 50$ (indicated by a dotted vertical line). $k_1 = 1000, r = 0.1$.

ble 4.1). All our models have in common that individuals are diploid and biparental, and, for simplicity, hermaphroditic. The models differ in how pairs are formed, in whether individuals can participate in multiple pairs, in whether or not selfing is possible, and in the distribution of the number of offspring produced by a pair.

Poisson model This is the model underlying the results in Wittmann *et al.* (2013c) and we use it here as a basis of comparison. Given a current population size N_t,

$$N_{t+1} \sim \text{Poisson}\left(\mathbf{E}[N_{t+1}|N_t]\right), \tag{4.2}$$

Table 4.1: Properties of the offspring-number models considered in this study. The values for c, the relative strength of genetic drift in equilibrium, are derived in Appendix 4.1.

model	$\mathbf{Var}[N_{t+1}\|N_t]$	relative strength of genetic drift c
binomial	$\mathbf{E}[N_{t+1}\|N_t] \cdot \left(1 - \frac{\mathbf{E}[N_{t+1}\|N_t]}{4 \cdot \lfloor N_t/2 \rfloor}\right)$	$\frac{3}{4}$
Poisson	$\mathbf{E}[N_{t+1}\|N_t]$	1
Poisson-Poisson	$3 \cdot \mathbf{E}[N_{t+1}\|N_t]$	2
Poisson-geometric	$5 \cdot \mathbf{E}[N_{t+1}\|N_t]$	3

such that $\mathbf{Var}[N_{t+1}|N_t] = \mathbf{E}[N_{t+1}|N_t]$.

Poisson-Poisson model Under this model, first a Poisson-distributed number of pairs

$$P_{t+1} \sim \text{Poisson}\left(\frac{1}{2} \cdot \mathbf{E}[N_{t+1}|N_t]\right) \tag{4.3}$$

are formed by drawing two individuals independently, uniformly, and with replacement from the members of the parent generation. That is, individuals can participate in multiple pairs and selfing is possible. Each pair then produces a Poisson-distributed number of offspring with mean 2. The offspring numbers of the P_{t+1} pairs are stored in the vector of family sizes $\mathbf{F_{t+1}} = (f_1, f_2, \ldots, f_{P_{t+1}})$. This vector is required to simulate the genealogies backward in time, unlike in the Poisson model where we only needed to store the total population size in each generation.

To compute the variance of N_{t+1} given N_t, we used the formula for the variance of the sum of a random number of independent and identically distributed random variables (Karlin & Taylor, 1975, p. 13)

$$\mathbf{Var}[N_{t+1}|N_t] = \mathbf{E}[X]^2 \cdot \mathbf{Var}[P_{t+1}|N_t] + \mathbf{E}[P_{t+1}|N_t] \cdot \mathbf{Var}[X], \tag{4.4}$$

where $\mathbf{E}[X]$ and $\mathbf{Var}[X]$ are mean and variance of the number of offspring produced by a single pair. The resulting variance (see table 4.1) is larger than that under the Poisson model.

Poisson-geometric model This model is identical to the Poisson-Poisson model except that the number of offspring of a given pair is geometrically distributed with mean 2, rather than Poisson-distributed. Using equation (4.4) again, we obtain an even larger variance than under the Poisson-Poisson model (table 4.1).

Binomial model Here, individuals can participate in only one pair and selfing is not possible. First, the individuals from the parent generation t form as many pairs as possible, i.e. $P_{t+1} = \lfloor N_t/2 \rfloor$. Then, each pair produces a binomially distributed number of offspring with parameters $n = 4$ and $p = \frac{\mathbf{E}[N_{t+1}|N_t]}{4 \cdot P_{t+1}}$, such that the population size in the offspring generation

$$N_{t+1} \sim \text{Binom}\left(n = 4 \cdot P_{t+1},\ p = \frac{\mathbf{E}[N_{t+1}|N_t]}{4 \cdot P_{t+1}}\right). \tag{4.5}$$

The reason for our choice of n was that it leads to a variance $np(1-p)$ that is smaller than the variance under the Poisson model (see table 4.1). As was the case for the previous two models, also here we needed to store the vector of family sizes to be able to simulate the genealogies backward in time.

(c) Demographic simulations

As we are only interested in populations that successfully overcome demographic stochasticity and the Allee effect, we discarded simulation runs in which the new population went extinct before reaching a certain target population size z. Here, we used $z = 2 \cdot a_{AE}$, i.e. twice the critical population size in populations with Allee effect. We generated 20,000 successful populations with and without Allee effect for each offspring-number model and for a range of founder population sizes between 0 and z. The population-size trajectories $N_0, N_1, \ldots, N_{T_z}$, where N_{T_z} is the first population size larger or equal to z, and the family-size vectors were stored for the subsequent backward-in-time simulation of genealogies. We also used the population-size trajectories to compute the average number of generations that the 20,000 replicate populations spent at each population size before reaching z. The complete simulation algorithm was implemented in C++ (Stoustrup, 1997), compiled using the g++ compiler (gcc.gnu.org, 2012), and uses the boost random number library (boost.org, 2013). We used R (R Development Core Team, 2011) for the analysis of simulation results.

(d) Simulation of genealogies

From each successful model population, we simulated ten independent single-locus genealogies, each for ten individuals sampled at both genome copies at the time when the population first reaches z. To construct the genealogies, we trace the ancestral lineages of the sampled individuals backward in time to their most recent common ancestor. For the Poisson model, we applied the simulation strategy of Wittmann *et al.* (2013c): Given the population-size trajectory $N_0, N_1, \ldots, N_{T_z}$ we let all lineages at time $t+1$ draw an ancestor independently, with replacement, and uniformly over all N_t individuals in the parent generation. For the other offspring-number models considered in this study, we use a modified simulation algorithm (see Appendix 4.1 for details) that takes into account the family-size information stored during the demographic simulation stage. Both simulation algorithms account for the possibility of multiple and simultaneous mergers of lineages and other particularities of genealogies in small populations. All lineages that have not coalesced by generation 0 are transferred to the source population. As in Wittmann *et al.* (2013c), we

simulated this part of the ancestral history by switching between two simulation modes: an exact and a more efficient approximative simulation mode (see Appendix 4.1). At the end of each simulation run, we stored the average time to the most recent common ancestor $\overline{G_2}$ for pairs of gene copies in the sample.

To visualise our results and compare them among the offspring-number models, we divided $\overline{G_2}$ by the average time to the most recent common ancestor for two lineages sampled from the source deme ($2k_0/c$). The quotient $\overline{G_2}/(2k_0/c)$ can be interpreted as the proportion of genetic diversity that the newly founded population has maintained relative to the source population. We also computed the per-cent change in expected diversity in populations with Allee effect (AE) compared to those without:

$$\left(\frac{\overline{G_2} \text{ with AE}}{\overline{G_2} \text{ without AE}} - 1 \right) \cdot 100. \tag{4.6}$$

(e) Effective-size rescaled Poisson model

Given a population size n in an offspring-number model with relative strength of genetic drift c (see table 4.1), we define the corresponding effective population size as $n_e(n) = n/c$. In this way, a population of size n in the target offspring-number model experiences the same strength of genetic drift as a Poisson population of size n_e. To approximate the various offspring-number models by a rescaled Poisson model, we thus set the population size parameters of the Poisson model (a, k_0, k_1, z, and N_0) to the effective sizes corresponding to the parameters in the target model. For example, to obtain a Poisson model that corresponds to the Poisson-geometric model we divided all population size parameters by 3. In cases where the effective founder population size $n_e(N_0)$ was not an integer, we used the next-larger integer in a proportion $q = n_e(N_0) - \lfloor n_e(N_0) \rfloor$ of simulations and the next-smaller integer in in the remainder of simulations. For the target population size, we used the smallest integer larger or equal to the rescaled value. All other parameters were as in the original simulations.

Results

The main results on the population dynamics and genetic diversity of populations with and without Allee effect are compiled in figure 4.2. The upper two rows show the population genetic consequences of the Allee effect for different founder population sizes, the lower row the average number of generations that successful populations spend in different

population-size ranges. Each column stands for one offspring-number model. Variation in offspring number and variability in the population dynamics increases from left to right. A first thing to note in figure 4.2 is that with increasing offspring-number variation the amount of genetic variation maintained in newly founded populations decreases, both for populations with and without Allee effect (solid black and grey lines in figure 4.2a–d). In populations without Allee effect, however, the decrease is stronger. As a result, the magnitude and direction of the Allee effect's influence on genetic diversity changes as variation in offspring number increases. For the binomial model, the model with the smallest variability in population dynamics and genetics, the Allee effect has a negative influence on the amount of diversity maintained for all founder population sizes we considered (figure 4.2a,e). For the model with the next-larger variation, the Poisson model, the Allee effect increases genetic diversity for small founder population sizes but decreases genetic diversity for large founder population sizes (figure 4.2b,f). These results on the Poisson model are consistent with those in Wittmann *et al.* (2013c). As variability further increases, the range of founder population sizes where the Allee effect has a positive effect increases (figure 4.2c,g). For the model with the largest offspring-number variation, the Poisson-geometric model, the Allee effect has a positive effect for all founder population sizes (figure 4.2d,h). In summary, the larger is the offspring-number variation, the more beneficial is the Allee effect's influence on genetic diversity.

The differences between offspring-number models in the population genetic consequences of the Allee effect (represented by the solid lines in figure 4.2a–h) result from two ways in which offspring-number variation influences genetic diversity: directly by influencing the strength of genetic drift for any given population-size trajectory, and indirectly by influencing the population dynamics of successful populations and thereby also the strength of genetic drift they experience. To disentangle the contribution of these two mechanisms, we first examine the direct genetic effect of offspring-number variation that results from its influence on the strength of genetic drift. For this, we generated a modified version for each of the binomial, Poisson-Poisson, or Poisson-geometric model (dashed lines in figure 4.2). We first simulated the population dynamics forward in time from the original model. Backwards in time, however, we ignored this family-size information and let lineages draw their ancestors independently, uniformly, and with replacement from the parent generation as in the Poisson model. In the case of the binomial model, where the modified model has stronger genetic drift than the original model, both populations with and without Allee effect maintain on average less genetic variation in the modified than in the original model (figure 4.2a). The Allee effect leads to a stronger reduction in genetic diversity in the mod-

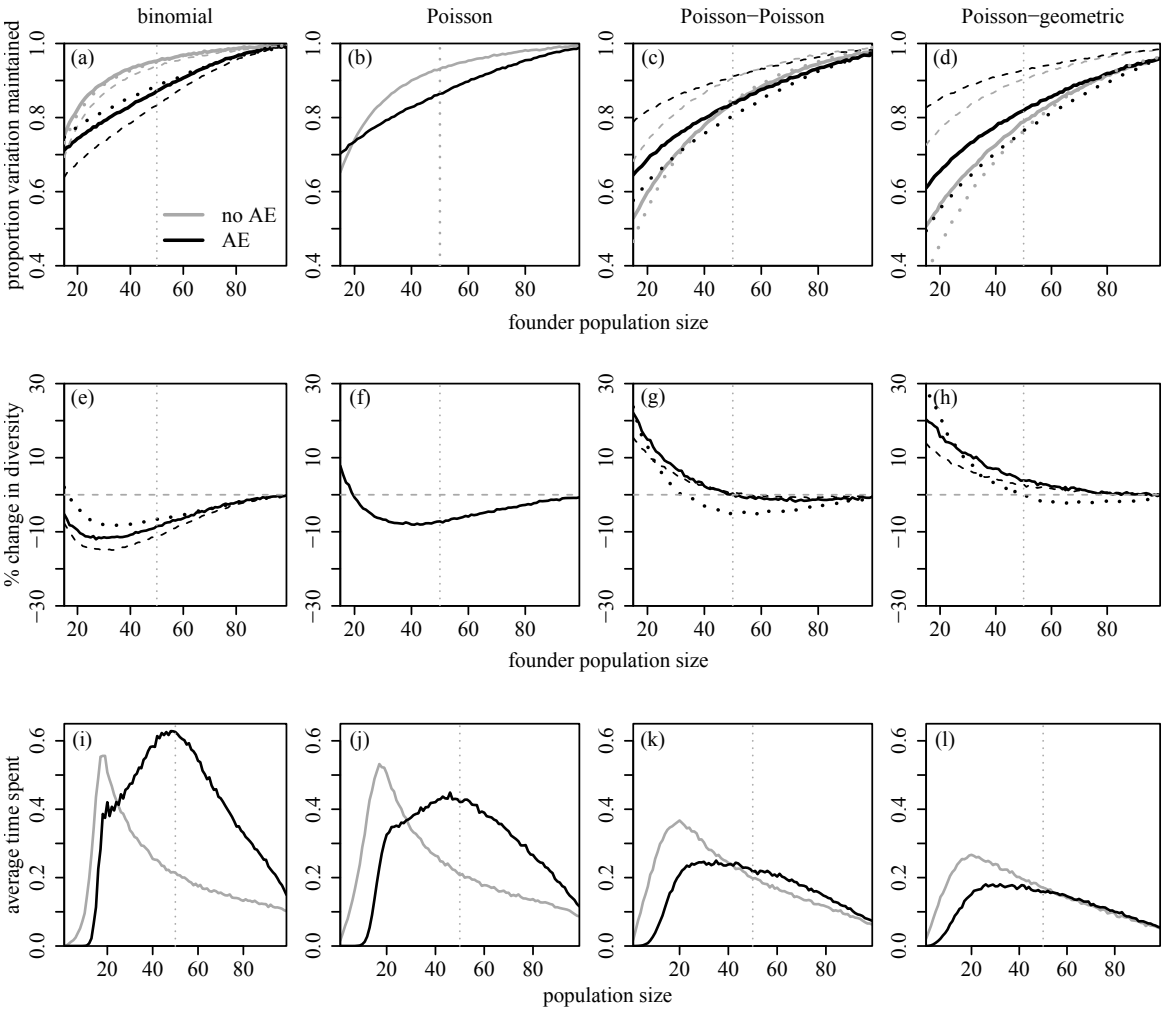

Figure 4.2: Consequences of the Allee effect for the population genetics and dynamics of successful populations under the binomial (1st column; a,e,i), Poisson (2nd column; b,f,j), Poisson-Poisson (3rd column; c,g,k) and Poisson-geometric model (4th column; d,h,l). Upper row: proportion of genetic variation maintained with Allee effect (AE, black lines) or without (no AE, grey lines). Middle row: per-cent change in genetic diversity in Allee-effect populations compared to those without Allee effect (see equation (4.6)). Dashed lines in the upper two rows represent populations whose size trajectories were simulated from the respective offspring-number model, but where the genealogies were simulated assuming the Poisson model. Dotted lines show the results for the respective effective-size rescaled Poisson model. Lower row: average number of generations spent by successful populations at each of the population sizes from 1 to $z-1$ before reaching population size z, either with Allee effect (black lines) or without (grey lines). The founder population size for the plots in the lower row was 15. Note that in the case of the rescaled Poisson model, the values on the x-axis correspond to the founder population sizes before rescaling. Dotted vertical lines indicate the critical size of Allee-effect populations in the original model. Every point in (a–l) represents the average over 20,000 simulations. For the proportion of variation maintained, the maximum standard error of the mean was 0.0019. Parameters in the original model: $k_1 = 1000, k_0 = 10,000, z = 100, r = 0.1$.

ified model than in the original model (figure 4.2e). The opposite pattern holds for the Poisson-Poisson and Poisson-geometric model where the modified model has weaker genetic drift than the original model. Populations in the modified model versions maintain a larger proportion of genetic variation (figure 4.2c,d), and the relative positive influence of the Allee effect is weaker (figure 4.2g,h).

Next, we consider the population dynamics of successful populations with and without Allee effect under the different offspring-number models. For this, we plotted the average number of generations that successful populations starting at population size 15 spend at each population size between 1 and $z - 1$ before reaching the target state z (lower row in figure 4.2). As variability increases (going from left to right) both kinds of successful populations spend fewer generations in total, i.e. reach the target population size faster, but again populations with and without Allee effect respond differently to increasing variability. If we first focus on the offspring-number models with intermediate variation (figure 4.2j,k) we observe that successful Allee-effect populations spend less time at small population sizes but more time at large population sizes than successful populations without Allee effect. This indicates that successful Allee-effect populations experience a speed-up in population growth at small sizes but are then slowed down at larger population sizes. If we now compare the results for the various offspring-number models, we observe that with increasing variability the speed-up effect becomes stronger and takes place over a larger range of population sizes, whereas the slow-down effect becomes weaker and finally disappears.

When rescaling the population-size parameters in the Poisson model to match one of the other offspring-number models, the resulting Poisson model behaves more similarly to the approximated model than does the original Poisson model, but the fit is not perfect (dotted lines in the upper two rows in figure 4.2). In general, the model versions without Allee effect are better approximated by the rescaled Poisson models than the model versions with Allee effect. Although the proportion of variation maintained in the rescaled model is close in magnitude to the one in the target model, the rescaled model often differ in its predictions as to the genetic consequences of the Allee effect. Rescaled Poisson models always predict the Allee effect to have a positive effect for small founder population sizes and a negative effect for larger founder population sizes, although for the binomial and Poisson-geometric model the effect is always negative or positive, respectively (figure 4.2e,h).

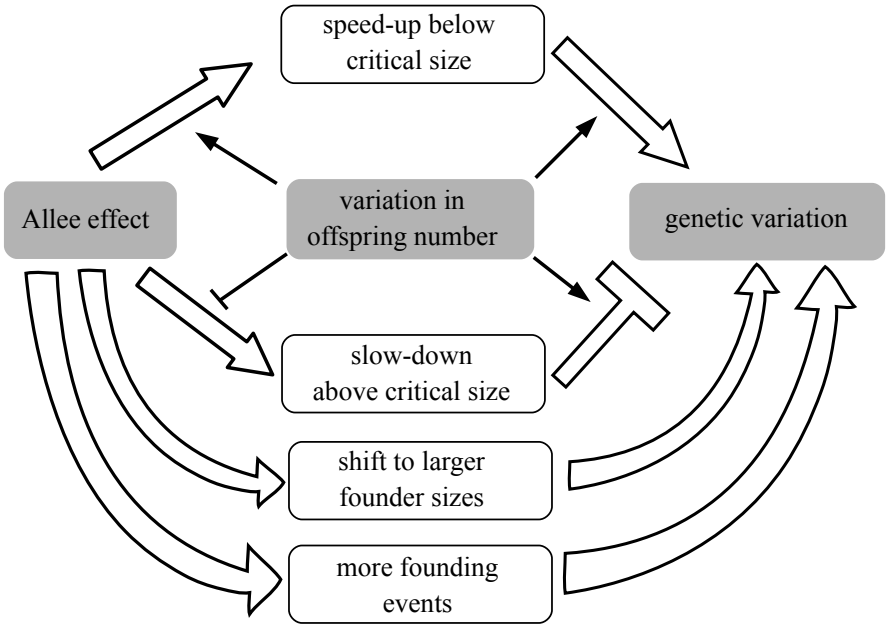

Figure 4.3: Overview over the various mechanisms by which the Allee effect influences the amount of genetic variation in successful introduced populations. Arrows represent positive effects while lines with bars represent inhibitory effects. Variation in offspring number enhances the speed-up of population growth caused by the Allee effect, but prevents the Allee-effect from causing a strong slow-down in population growth above the critical size. Variation in offspring number also magnifies the genetic consequences of both a speed-up and a slow-down in population growth.

Discussion

Our results indicate that offspring-number variation plays a key role for the genetic consequences of the Allee effect. We can understand a large part of the differences between offspring-number models if we consider how many generations successful populations spend in different population-size regions before reaching the target population size. In Wittmann *et al.* (2013c), we found that with Poisson-distributed offspring numbers successful Allee-effect populations spend less time at small population sizes than populations without Allee effect. Apparently, small Allee-effect populations can only avoid extinction by growing very quickly (speed-up in figure 4.3). We also found, however, that Allee effect-populations spend on average more time at large population sizes than populations without Allee effect (slow-down in figure 4.3). Consequently, under the Poisson model the Allee effect had either a positive or a negative effect on levels of genetic diversity depending on the founder population size.

An increase in offspring-number variation leads to more variable population dynamics, which on the one hand lets successful populations escape even faster from the range of small population sizes than under the Poisson model. On the other hand, a large variation also prevents successful Allee-effect populations from spending much time near or above the critical population size because those that do still have a high risk of going extinct even at such high population sizes. Therefore, an increase in variability reinforces the speed-up effect but mitigates the slow-down effect and thus increases the range of founder population sizes for which the genetic consequences of the Allee effect are positive (see figure 4.2,4.3). In that sense, variation in family sizes plays a similar role as variation in founder population size and in the number of introduction events (see figure 4.3), two factors that were examined in Wittmann *et al.* (2013c) and, in the case of founder population size, also by Kramer & Sarnelle (2008). Variation in these aspects also leads to a positive influence of the Allee effect on diversity because by conditioning on success we let the Allee effect select the outliers of the respective distributions, and it is those outliers (particularly large founder sizes, exceptionally many introduction events) that lead to a large amount of genetic diversity.

Apart from its indirect but strong influence via the population dynamics, variation in offspring number also has a direct influence on genetic diversity by determining the strength of drift for a given population-size trajectory. Our comparisons between models with the same population dynamics but a different strength of drift suggest that an increase in the strength of genetic drift amplifies the per-cent change in diversity of Allee-effect populations compared to populations without Allee effect. We suggest this is the case because the stronger genetic drift is, the more genetic variation is lost or gained if it takes one generation more or less to reach the target population size. Thus, by reinforcing both the positive effect of a speed-up on genetic variation and the negative effect of a slow-down (figure 4.3), an increase in variation increases the magnitude of the net influence of the Allee effect on genetic variation.

We have now established that for a given set of parameter values, the population genetic consequences of the Allee effect differ strongly between offspring-number models. Nevertheless, we would still be able to use the Poisson model for all practical purposes if for any given set of parameters in one of the other offspring models we could find a set of effective parameters in the Poisson model that would yield similar results. The most obvious way to do this is to replace the population size parameters in the Poisson model by the corresponding effective population sizes, i.e. the population sizes in the Poisson model at which genetic drift is as strong as it is in the target model at the original population size parameter. However, our results (see figure 4.2) indicate that the effective-size rescaled Poisson mod-

els cannot fully reproduce the results of the various offspring-number models. In particular, the population dynamics of successful populations remain qualitatively different from those under the target model.

Apart from the population-size parameters, the Poisson model has an additional parameter that we could adjust, the growth parameter r. In Wittmann *et al.* (2013c), we considered different values of r in the Poisson model. Small values of r led to qualitatively similar results as we have seen here in models with a larger offspring-number variation and larger values of r. The reason appears to be that the population dynamics of successful populations depend not so much on the absolute magnitude of the average population growth rate (the deterministic forces) or of the associated variation (the stochastic forces), but on the relative magnitude of deterministic and stochastic forces. Indeed, a theorem from stochastic differential equations states that if we multiply the infinitesimal mean and the infinitesimal variance of a process by the same constant ρ, we get a process that behaves the same, but is sped up by a factor ρ (Durrett, 1996, Theorem 6.1 on p. 207). Intuitively, we can make a model more deterministic either by increasing the growth parameter or by decreasing the variance. This suggests that we can qualitatively match the population dynamics of any given offspring-number model if we choose r appropriately. Furthermore, we can match the strength of genetic drift if we rescale the population-size parameters appropriately. One could therefore suppose that by adjusting both the population-size parameters and the growth parameter in the Poisson model, we might be able to match both the population dynamic and the genetic aspects of the other offspring-number models. In Appendix 4.2, however, we show that this can only be possible if the equilibrium strength of genetic drift c equals $\mathbf{Var}(N_{t+1}|N_t)/\mathbf{E}(N_{t+1}|N_t)$. This is not the case for the offspring-number models we examine in this study (see table 4.1), neither with nor without Allee effect. However, our results suggest that the Allee effect enhances the mismatch between the effective-size rescaled Poisson models and their target models.

Overall, our results suggest that if we study populations that had been small initially but successfully overcame an Allee effect, microscopic properties such as the variation in offspring number can play a large role, although they may not influence the average unconditional population dynamics. Thus the common practice of first building a deterministic model and then adding some noise to make it stochastic may not produce meaningful results. As emphasised by Black & McKane (2012), stochastic population dynamic models should be constructed in a bottom-up way starting with modelling the relevant processes at the individual level and then deriving the resulting population dynamics in a second step. This means, we have to gather detailed biological knowledge about a species of interest be-

fore being able to predict the population genetic consequences of the Allee effect or other phenomena involving the stochastic dynamics of small populations. In this study, we have combined offspring-number models with a separate phenomenological model for the Allee effect. This helped us to compare the population-genetic consequences of the Allee effect among the different offspring-number model. A next step to make our model more mechanistic could be to let both the Allee effect and offspring-number variation emerge from a detailed mechanistic model for individual reproductive success.

In this study and in Wittmann *et al.* (2013c), we have focused on how the Allee effect impacts levels of neutral genetic diversity. Additional complications will arise if we take into account aspects of genetic variation that are required for the adaptation of an introduced population to its new environment. This need for adaptation can give rise to an Allee effect in itself because larger founding populations will have a larger probability to harbour the alleles that are advantageous in the new environment. We are currently investigating how this genetic Allee effect interacts with mate-finding or other ecological Allee effects and with the phenomena discussed in this paper.

Acknowledgements

We thank Peter Pfaffelhuber for pointing us to an interesting result on rescaling diffusion processes. MJW is grateful for a scholarship from the Studienstiftung des deutschen Volkes.

Appendix 4.1 Details of the genealogy simulation

To simulate a genealogy, we start with the $n_s = 10$ individuals sampled at generation T_z and then trace their ancestry backward in time generation by generation until we arrive at the most recent common ancestor of all the genetic material in the sample. At time $t+1$, for example, we may have n_a individuals that carry genetic material ancestral to the sample. To trace the ancestry back to generation t, we make use of the family-size vector $\mathbf{F}_{t+1}$ stored during the forward-in-time simulation stage. We first assign each of the n_a individuals to a family by letting them successively pick one of the families in proportion to their sizes, i.e. the entries f_i of the family-size vector. After a family has been picked, the corresponding entry in the family-size vector is reduced by 1 to avoid that the same individual is picked twice.

After all n_a individuals have been assigned, all families that have been chosen at least once draw parents in generation t. Here, the models differ. In the Poisson-Poisson and Poisson-geometric model, each family picks two parents independently, with replacement, and uniformly over all individuals in the parent generation. That is, parents can be picked by several families, and they can even be picked twice by the same family, corresponding to selfing. In the binomial model, on the other hand, parents are chosen without replacement. Each parent can only be picked by one family and selfing is not possible. From there on, everything works as described in Wittmann *et al.* (2013c): The two genome copies of each ancestral individual split and each of the two parents (or possibly the same individual in the case of selfing) receives one genome copy. A coalescent event happens if the same genome copy in a parent receives genetic material from several children.

At time 0, i.e. at the time of the introduction, all remaining ancestors are transferred (backwards in time) to the source population, which is of large but finite size k_0 at all times. We assume that the mechanism of pair formation and the distribution of the number of offspring per pair are the same in the source population and in the newly founded population. Therefore, we still need to take into account family-size information to simulate the genealogy backward in time. Since we did not generate the required family-size vectors during the forward-in-time simulation stage, we now simulate them ad-hoc at every backward-in-time step. We do this by sampling from the distribution of family sizes (Poisson or geometric with mean 2, or binomial with $n = 4, p = 1/2$) until we get a total number of k_0 individuals (truncating the last family if required). Apart from this modification, the simulation algorithm is identical to the one used in the first stage of the simulation. When the simulation arrives at a point where all n_a ancestors carry ancestral material at only one genome copy, it

usually takes a long time until lineages are again combined into the same individual. Under these circumstances, we therefore use a more efficient approximative simulation mode: We draw the number of generations until two ancestors are merged into the same individual from a geometric distribution with parameter

$$p_{merge} = c \cdot \frac{\binom{n_a}{2}}{k_0}, \tag{4.7}$$

where c is the strength of genetic drift compared to that under the Poisson model (see table 4.1). Whenever such an event happens, two randomly chosen ancestors are merged. Here we need to distinguish between two possible outcomes, both of of which occur with probability 1/2. In the first case, there is a coalescent event and we now have $n_a - 1$ ancestors, each still with ancestral material at only one genome copy. We therefore continue in the efficient simulation model. In the second case, there is no coalescent event and the resulting individual has ancestral material at both genome copies. Therefore, we switch back the other exact simulation mode. In this way, we switch back and forth between the two simulation modes until the most recent common ancestor of all the sampled genetic material has been found. For each simulation run, we store the average pairwise time to the most recent common ancestor of pairs of gene copies in the sample.

We will now take a closer look at the genealogical implications of the different offspring number models. Thereby, we will derive the values for the relative strength of genetic drift c given in table 4.1 and justify the approximation in (4.7). Equation (4.7) is valid as long as we can neglect events in which at least three ancestors are merged into the same individual in a single generation (multiple mergers) and events in which several pairs of ancestors are merged in a single generation (simultaneous mergers). In the following, we will show that we can indeed neglect such events for large source population sizes k_0 because the per-generation probability of multiple and simultaneous mergers is $\mathcal{O}(1/k_0^2)$ whereas single mergers occur with probability $\mathcal{O}(1/k_0)$. Here $f(k_0) = \mathcal{O}(g(k_0))$ means that there exist positive constants M and k^* such that

$$|f(k_0)| \leq M \cdot |g(k_0)| \tag{4.8}$$

for all $k_0 > k^*$ (Knuth, 1997, p. 107). In other words, as k_0 goes to infinity, an expression $\mathcal{O}(1/k_0^n)$ goes to zero at least as fast as $1/k_0^n$.

To derive the probabilities of multiple and simultaneous mergers, we first introduce some notation. Let P be a random variable representing the number of pairs with at least one

offspring individual, and O_l the number of offspring of the lth of them, with $l \in \{1, \ldots, P\}$. Because we assume a fixed size k_0 of the source population and because we truncate the last family if necessary to achieve this (see above), the O_l are not exactly independent and identically distributed. However, in all cases we are considering, k_0 is very large relative to typical offspring numbers O_l. To facilitate our argument, we therefore approximate the O_l by independent and identically distributed Y_l with $l \in \{1, \ldots, P\}$ taking values in $\mathbb{N}$. Further, let Z_l be the number of lineages in the next generation tracing back to family l.

In the following computations, we will need the fact that the first three moments of the Y_l ($\mathbf{E}[Y_l]$, $\mathbf{E}[Y_l^2]$, and $\mathbf{E}[Y_l^3]$) are finite. These are the moments of the number of offspring per pair, conditioned on the fact that there is at least one offspring. Let X denote a random variable with the original distribution of the number of offspring per pair, i.e. the one allowing also for families of size 0. Then the moments of the Y_l can be computed from the moments of X with the help of Bayes' formula:

$$\begin{aligned}\mathbf{E}[Y_l^m] &= \sum_{k=1}^{\infty} k^m \cdot \mathrm{Pr}(X = k | X \geq 1) = \sum_{k=1}^{\infty} k^m \cdot \frac{\mathrm{Pr}(X \geq 1 | X = k) \cdot \mathrm{Pr}(X = k)}{Pr(X \geq 1)} \\ &= \sum_{k=1}^{\infty} k^m \cdot \frac{\mathrm{Pr}(X = k)}{1 - \mathrm{Pr}(X = 0)} = \frac{\mathbf{E}[X^m]}{1 - \mathrm{Pr}(X = 0)}. \end{aligned} \tag{4.9}$$

These computations lead to finite constants for the first three moments of the Y_l in the binomial, Poisson-Poisson, and Poisson-geometric model.

Since we switch to the accurate simulation mode whenever two lineages are combined into the same individual, we can assume here that the lineages at time $t + 1$ are in different individuals. Thus, the number of lineages is equal to the number of ancestors n_a and is at most twice the sample size (in our case at most 20), but usually much lower since many coalescent events already happen in the newly founded population. Of course, each ancestor has two parents, but since ancestors here carry genetic material ancestral to the sample only at one genome copy, only one of their two parents is also an ancestor and the other one can be neglected.

A multiple merger can occur in three, not necessarily mutually exclusive, ways (figure 4.4a–c). First we will consider the case where there is at least one family that at least three

lineages trace back to (figure 4.4a). The probability of such an event is

$$\begin{aligned}
\Pr\left(\max(Z_l) \geq 3\right) &\leq \mathbf{E}\left[\Pr\left(\bigcup_{l=1}^{P}(Z_l \geq 3)\middle| P\right)\right] \\
&\leq \mathbf{E}\left[\sum_{l=1}^{P}\Pr(Z_l \geq 3)\right] \\
&\leq \mathbf{E}[P]\cdot\Pr(Z_1 \geq 3) \\
&\leq k_0 \cdot \Pr(Z_1 \geq 3) \\
&\leq k_0 \cdot \binom{n_a}{3} \cdot \left(\sum_{i=1}^{\infty}\Pr(Y_1 = i)\cdot\frac{Y_1}{k_0}\cdot\frac{Y_1-1}{k_0-1}\cdot\frac{Y_1-2}{k_0-2}\right) \\
&\leq k_0 \cdot \binom{n_a}{3} \cdot \frac{\mathbf{E}\left[Y_1^3\right]}{k_0^3} = \mathcal{O}\left(\frac{1}{k_0^2}\right).
\end{aligned} \tag{4.10}$$

Here, we used the fact that $P \leq k_0$, which is true because we take P to be the number of families with at least one member. Note that when lineages trace back to the same family, they still need to draw the same parent individual in order to be merged. For the three lineages here, this probability is 1/4 if the family did not arise from selfing, and 1 if it did. For simplicity, we take 1 as an upper bound for this probability here and for similar probabilities in the following inequalities.

Under the binomial model, the above case is the only way in which multiple mergers can occur. Under the Poisson-Poisson and Poisson-geometric model, however, parent individuals can participate in several pairs and therefore potentially contribute to more than one family. Therefore, we additionally have to take into account the possibility that lineages trace back to different families but then choose the same parent individual (figure 4.4b,c). One possibility (figure 4.4b) is that there is exactly one family that at least two lineages trace back to (event E_1), that two lineages in this family draw the same parent individual (event E_2), and that there is at least one lineage outside the family that draws the same parent (event E_3). Using an argument analogous to that in (4.10), we obtain

$$\Pr(E_1) \leq \Pr\left(\max(Z_l) \geq 2\right) \leq \binom{n_a}{2} \cdot \frac{\mathbf{E}[Y_1^2]}{k_0}. \tag{4.11}$$

Furthermore, $\Pr(E_2) \leq 1$ and

$$\Pr(E_3) \leq (n_a - 2) \cdot \frac{1}{k_0} \leq n_a \cdot \frac{1}{k_0}. \tag{4.12}$$

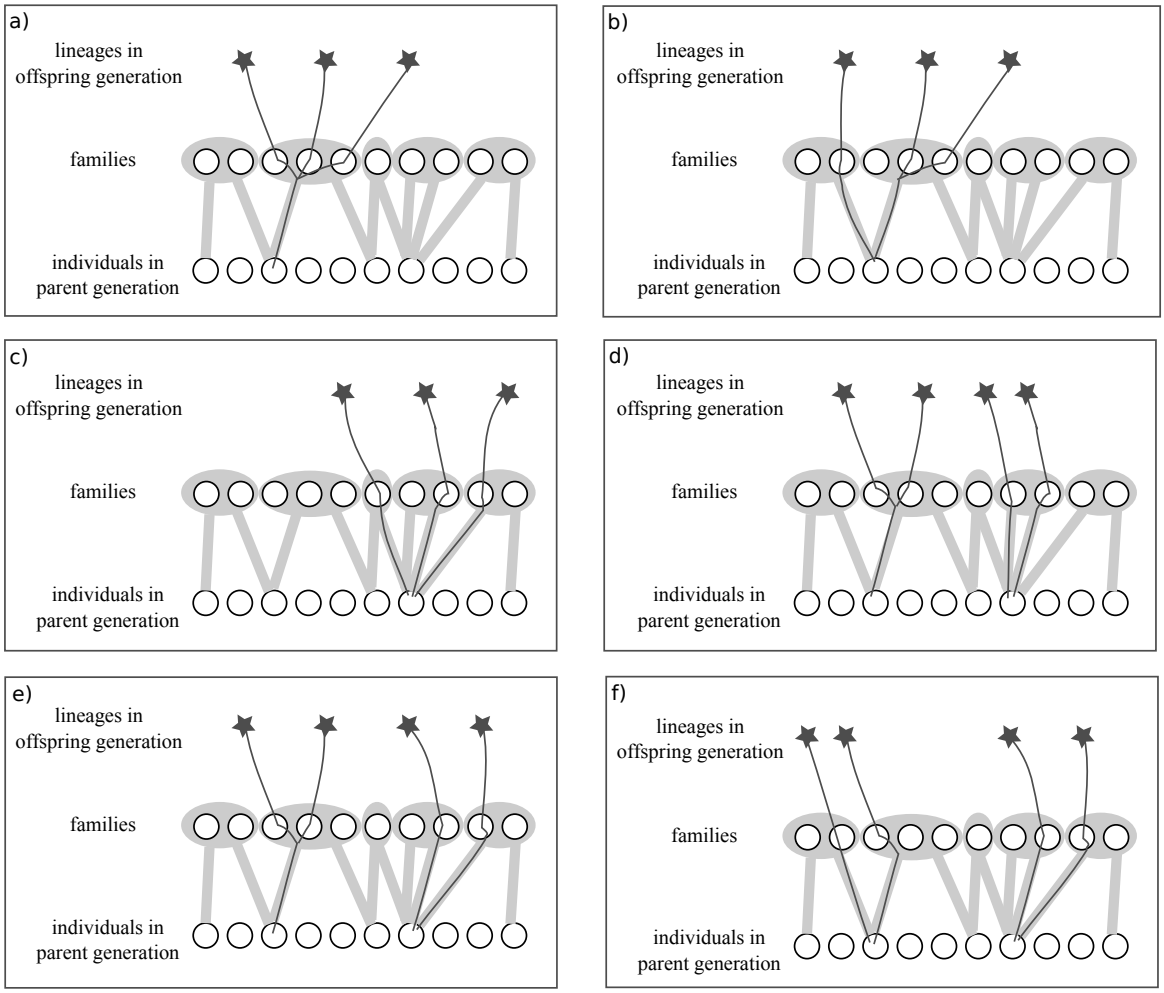

Figure 4.4: Illustration of the various ways in which multiple and simultaneous mergers can arise. Here, the case of the Poisson-Poisson or Poisson-geometric model is depicted, where selfing is possible and individuals in the parent generation can contribute to multiple families.

Here we use the fact that families choose their parents independently and uniformly over all k_0 individuals in the parent generation, such that lineages in different families have a probability of $1/k_0$ of drawing the same parent. Combining the last three inequalities, we can conclude

$$\Pr(E_1 \cap E_2 \cap E_3) \leq \binom{n_a}{2} \cdot \frac{\mathbf{E}[Y_1^2]}{k_0^2} \cdot n_a = \mathcal{O}\left(\frac{1}{k_0^2}\right). \tag{4.13}$$

Finally, the probability that lineages from at least three different families choose the same parent (figure 4.4c) is bounded by

$$\binom{n_a}{3} \frac{1}{k_0^2} = \mathcal{O}\left(\frac{1}{k_0^2}\right). \tag{4.14}$$

For a simultaneous merger to occur, there must be at least two mergers of at least two lineages each. This can happen in three (not necessarily mutually exclusive) ways (figure 4.4d-f). To compute bounds for the corresponding probabilities, we again take 1 as an upper bound for all probabilities that lineages in the same family choose the same parent. First, a simultaneous merger can occur if there are at least two families, each with at least two lineages tracing back to them (event M_1, figure 4.4d). This occurs with probability

$$\begin{aligned}
\Pr(M_1) &\leq \mathbf{E}\Big[\Pr\left(\exists\, l, m \in \{1, \ldots, P\} \text{ s.t. } Z_l \geq 2 \text{ and } Z_m \geq 2 \big| P\right)\Big] \\
&\leq \mathbf{E}\left[\sum_{l,m \leq P,\, l \neq m} \Pr(Z_l \geq 2, Z_m \geq 2)\right] \\
&\leq \mathbf{E}\left[\binom{P}{2}\right] \cdot \Pr(Z_1 \geq 2, Z_2 \geq 2) \qquad (4.15) \\
&\leq \binom{k_0}{2}\left(\sum_{i=1}^{\infty}\sum_{j=1}^{\infty} \Pr(Y_1 = i) \cdot \Pr(Y_2 = j) \cdot \binom{n_a}{2}^2 \cdot \frac{Y_1}{k_0} \cdot \frac{Y_1 - 1}{k_0 - 1} \cdot \frac{Y_2}{k_0 - 2} \cdot \frac{Y_2 - 1}{k_0 - 3}\right) \\
&\leq \frac{1}{2} \cdot \binom{n_a}{2}^2 \cdot \frac{\mathbf{E}[Y_1^2] \cdot \mathbf{E}[Y_2^2]}{(k_0 - 2)(k_0 - 3)} = \mathcal{O}\left(\frac{1}{k_0^2}\right).
\end{aligned}$$

Second, there can be a simultaneous merger if there is one family with at least two lineages tracing back to it and two lineages that merge separately without being in the family (event M_2, figure 4.4e). Using the bound for the probability of exactly one merger of two individuals from (4.11), we obtain

$$\Pr(M_2) \leq \Pr(E_1) \cdot \binom{n_a}{2} \frac{1}{k_0} \leq \binom{n_a}{2}^2 \cdot \frac{\mathbf{E}\left[Y_1^2\right]}{k_0^2} = \mathcal{O}\left(\frac{1}{k_0^2}\right). \tag{4.16}$$

Finally, we can have two pairs of lineages both merging without being in the same family (event M_3, figure 4.4f). This occurs with probability

$$\Pr(M_3) \leq \binom{n_a}{2}^2 \frac{1}{k_0^2} = \mathcal{O}\left(\frac{1}{k_0^2}\right). \tag{4.17}$$

The different possibilities for multiple and simultaneous mergers are not mutually exclusive (for example there could be one triple merger and two double mergers), but since all of them have probability $\mathcal{O}\left(1/k_0^2\right)$, the probability of their union is also $\mathcal{O}\left(1/k_0^2\right)$. With these results, we can write

$$\Pr(\text{1 merger}) = \binom{n_a}{2}\Pr(A) + \mathcal{O}\left(\frac{1}{k_0^2}\right), \tag{4.18}$$

where A denotes the event that a specific pair of lineages merges into the same individual in the previous generation.

$$\begin{aligned}\Pr(A) &= \Pr(\text{same family}) \cdot \Pr(A|\text{same family}) \\ &\quad + \Big(1 - \Pr(\text{same family})\Big) \cdot \Pr(A|\text{different families})\end{aligned} \tag{4.19}$$

For the binomial model,

$$\Pr(A|\text{same family}) = \frac{1}{2} \tag{4.20}$$

and

$$\Pr(A|\text{different families}) = 0. \tag{4.21}$$

For the other models,

$$\Pr(A|\text{same family}) = \left(1 - \frac{1}{k_0}\right) \cdot \frac{1}{2} + \frac{1}{k_0} \cdot 1 = \frac{1}{2} + \frac{1}{2k_0}, \tag{4.22}$$

thereby accounting for the possibility of selfing, and

$$\Pr(A|\text{different families}) = \frac{1}{k_0}. \tag{4.23}$$

For all models, we have

$$\Pr(\text{same family}) = \frac{\mathbf{E}[S]}{k_0} = \frac{\mathbf{E}[X^*] - 1}{k_0}, \tag{4.24}$$

where $\mathbf{E}[S]$ is the expected number of siblings of a sampled individual and $\mathbf{E}[X^*]$ is the size-biased expectation of the number of offspring per family. Using equation 4 in Arratia & Goldstein (2010), we obtain

$$\mathbf{E}[X^*] = \frac{\mathbf{Var}[X]}{\mathbf{E}[X]} + \mathbf{E}[X], \tag{4.25}$$

which is 2.5 for the binomial model, 3 for the Poisson-Poisson model, and 5 for the Poisson-geometric model.

Substituting (4.20), (4.21), and (4.24) into (4.19) and then into (4.18), we obtain for the binomial model:

$$\Pr(\text{1 merger}) = \binom{n_a}{2} \cdot \frac{\mathbf{E}[X^*] - 1}{2k_0} + \mathcal{O}\left(\frac{1}{k_0^2}\right). \tag{4.26}$$

Analogously, substituting (4.22), (4.23), and (4.24) into (4.19) and then into (4.18), we obtain for the other models:

$$\begin{aligned}\Pr(\text{1 merger}) &= \binom{n_a}{2} \cdot \left[\frac{\mathbf{E}[X^*] - 1}{k_0} \cdot \left(\frac{1}{2} + \frac{1}{2k_0}\right) + \left(1 - \frac{\mathbf{E}[X^*] - 1}{k_0}\right) \cdot \frac{1}{k_0}\right] + \mathcal{O}\left(\frac{1}{k_0^2}\right) \\ &= \binom{n_a}{2} \cdot \left[\frac{\mathbf{E}[X^*] - 1}{2k_0} + \frac{1}{k_0}\right] + \mathcal{O}\left(\frac{1}{k_0^2}\right). \end{aligned} \tag{4.27}$$

For the approximation in (4.7), we neglect terms of order $1/k_0^2$ and thus the relative strength of genetic drift in (4.7) is given by

$$c = \frac{\mathbf{E}[X^*] - 1}{2} + \begin{cases} 0 & \text{for the binomial model} \\ 1 & \text{otherwise} \end{cases}. \tag{4.28}$$

Evaluating this expression for the various models yields the values in table 4.1.

Appendix 4.2 Rescaling the Poisson model

In this section, we argue that in general it is not possible to rescale the Poisson model such that it gives reasonable approximations to one of the other offspring-number models with respect to both population dynamics and genetics, even if we both linearly rescale the population-size parameters and change the growth parameter. Given an offspring-number model M with critical population size a, carrying capacity k_1, target population size z, founder population size N_0, growth parameter r, $\nu = \mathbf{Var}[N_{t+1}|N_t]/\mathbf{E}(N_{t+1}|N_t)$, and relative strength of genetic drift c, we will attempt to determine scaling parameters s and ρ such that a Poisson model M' with parameters $a' = s \cdot a, k_1' = s \cdot k_1, z' = s \cdot z, N_0' = s \cdot N_0$, and $r' = \rho \cdot r$ approximates the original model.

In our argument, we will be guided by the following theorem on a time change in diffusion processes (Durrett, 1996, Theorem 6.1 on p. 207): If we speed up a diffusion process by a factor ρ, we obtain the same process as when we multiply both its infinitesimal mean and its infinitesimal variance by a factor ρ. In this study, we do not consider diffusion processes, but processes in discrete time and with a discrete state space. Furthermore, we cannot manipulate mean and variance independently. Therefore, the theorem cannot hold exactly for the models in this study. Nevertheless, it yields good approximations to the population dynamics under the the binomial, Poisson-Poisson, and Poisson-geometric model (figure 4.5). Specifically, a Poisson model M' with $r' = \rho \cdot r$ runs ρ times as fast but otherwise exhibits approximately the same population dynamics as a model M'' with growth parameter r and $\nu = \mathbf{Var}[N_{t+1}|N_t]/\mathbf{E}(N_{t+1}|N_t) = 1/\rho$, given that all other parameters are the same, i.e. $a'' = a', k_1'' = k_1', z'' = z'$ and $N_0'' = N_0'$. However, due to the difference in time scale, the genetic drift experienced by populations under the two models may be very different.

To determine whether the offspring-number model M can be approximated by a Poisson model M', we will check whether it is possible to simultaneously fulfil two conditions, one on the population dynamics and one on the genetic aspect of the models. These two conditions are not sufficient to ensure that the models behave the same in every respect, but they appear necessary. If we can show that it is not possible to fulfil them simultaneously, not even in the unconditioned model, then the population dynamics and/or genetics of successful populations should be different under the two models.

First, we will specify a condition required to match the population dynamics. Since the success or failure of a population and other qualitative features of the population dynamics do not depend on the time scaling and since it is easier to compare models with the same growth parameter, we will use the model M'' instead of model M' here. To match the

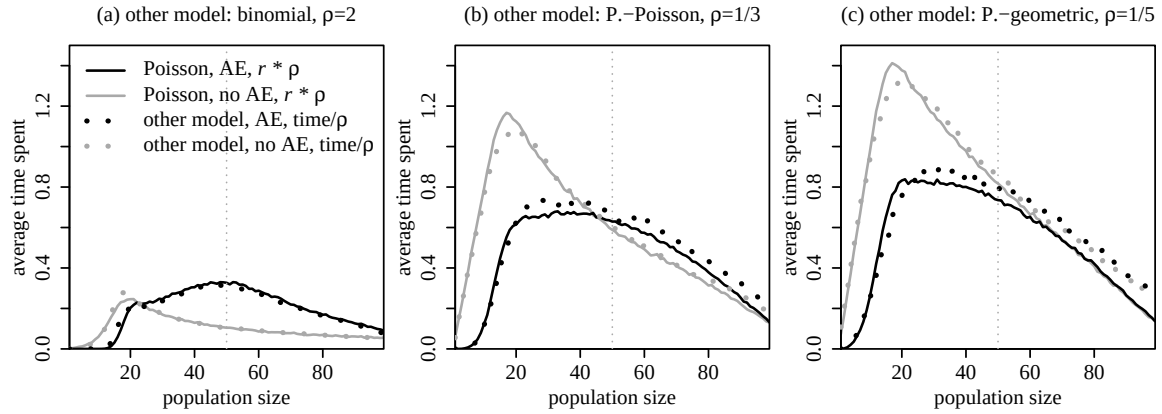

Figure 4.5: Comparison of Poisson models with growth parameter $r' = 0.1 \cdot \rho$ to other offspring-number models, the binomial model (a), the Poisson-Poisson model (b), and the Poisson-geometric model (c), all with growth parameter $r'' = 0.1$. Each subplot shows the average number of generations that successful populations with Allee effect (AE) or without (no AE) under the various models spend at different population sizes from 1 to $z-1$ before reaching the target population size z. In each case, we set $\rho = 1/\nu$ (see table 4.1, and using $\nu \approx 1/2$ for the binomial model) and divided the times spent at the different population sizes under the respective other offspring model by ρ to account for the change in time scale. The dotted vertical line indicates the critical size a of Allee-effect populations, here $a = 50$. $k_1 = 1000, z = 100$.

relative strength of stochastic vs. deterministic forces in the population dynamics, we will require that the standard deviation of the population size in the next generation relative to the corresponding expected value is equal in both models for corresponding population sizes $n'' = s \cdot n$:

$$\frac{\sqrt{\mathbf{Var}[N''_{t+1}|N''_t = n'']}}{\mathbf{E}[N''_{t+1}|N''_t = n'']} = \frac{\sqrt{\mathbf{Var}[N_{t+1}|N_t = n]}}{\mathbf{E}[N_{t+1}|N_t = n]}. \tag{4.29}$$

Given the properties of the model M'' and M stated above, this is equivalent to

$$\frac{\sqrt{\frac{1}{\rho} \cdot \mathbf{E}[N''_{t+1}|N''_t = n'']}}{\mathbf{E}[N''_{t+1}|N''_t = n'']} = \frac{\sqrt{\nu \cdot \mathbf{E}[N_{t+1}|N_t = n]}}{\mathbf{E}[N_{t+1}|N_t = n]}. \tag{4.30}$$

$$\Leftrightarrow \frac{1}{\sqrt{\rho \cdot \mathbf{E}[N''_{t+1}|N''_t = n'']}} = \frac{\sqrt{\nu}}{\sqrt{\mathbf{E}[N_{t+1}|N_t = n]}}. \tag{4.31}$$

$$\Leftrightarrow \frac{1}{\sqrt{\rho \cdot n'' \cdot \phi\left(\frac{n''}{k_1''}\right)}} = \frac{\sqrt{\nu}}{\sqrt{n \cdot \phi\left(\frac{n}{k_1}\right)}}, \tag{4.32}$$

where

$$\phi(x) = e^{r\cdot(1-x)\cdot\left(1-\frac{a}{k_1\cdot x}\right)} = e^{r\cdot(1-x)\cdot\left(1-\frac{a''}{k_1''\cdot x}\right)} \tag{4.33}$$

is the expected per-capita number of surviving offspring in a population whose current size is a fraction x of the carrying capacity (see equation (4.1)). Since $n''/k_1'' = n/k_1$, (4.32) reduces to

$$\frac{1}{\nu} = \frac{\rho \cdot n''}{n} = \rho \cdot s. \tag{4.34}$$

This is our first condition.

Second, both models should have the same strength of genetic drift at corresponding population sizes n and n'. Specifically, we require that the heterozygosity maintained over a corresponding time span is equal in both models:

$$\left(1 - \frac{1}{n'}\right)^{1/\rho} = \left(1 - \frac{c}{n}\right), \tag{4.35}$$

which corresponds approximately to the condition

$$\frac{1}{\rho \cdot n'} = \frac{c}{n} \tag{4.36}$$

as long as n and n' are not too small. Here, we need the exponent $1/\rho$ because—as we have seen above and in figure 4.5—multiplying the growth parameter by a factor ρ effectively speeds up the process by the same factor such that there is less time for genetic drift to act. Using $n' = s \cdot n$, (4.36) simplifies to

$$\frac{1}{c} = \rho \cdot s. \tag{4.37}$$

This is our second condition.

Combining (4.34) and (4.37) shows that the two conditions can only be fulfilled simultaneously if $\nu = c$, which is not the case for the offspring-number models we consider in

this study (see table 4.1). The mismatch between ν and c in our models is related to the way in which the diploid individuals form pairs to sexually reproduce. In a haploid and asexual model, in which individuals independently produce identically distributed numbers of offspring, $\mathbf{Var}[N_{t+1}|N_t] = N_t \cdot \mathbf{Var}[X]$ and $\mathbf{E}[N_{t+1}|N_t] = N_t \cdot \mathbf{E}[X]$, where X is a random variable representing the number of offspring produced by a single individual. In analogy to (4.28), we can then quantify the strength of genetic drift as

$$c = \mathbf{E}[X^*]-1 = \frac{\mathbf{Var}[X]}{\mathbf{E}[X]}+\mathbf{E}[X]-1 = \frac{\mathbf{Var}[N_{t+1}|N_t]}{\mathbf{E}[N_{t+1}|N_t]}+\mathbf{E}[X]-1 = \nu+\mathbf{E}[X]-1. \quad (4.38)$$

This shows that in equilibrium, i.e. for $\mathbf{E}[X] = 1$, there would be no mismatch between c and ν in such a haploid model. In other situations, however, especially if we condition on the success of a small Allee-effect population, there could still be a mismatch. Furthermore, as discussed above, conditions (4.34) and (4.37) may not be sufficient to ensure that two processes behave similarly. Especially if we condition on an unlikely event, the higher moments characterising the tail of the offspring-number distribution may be important and they are not necessarily matched even if mean and variance are. We therefore suggest that the strong differences among offspring-number distributions in the genetic consequences of the Allee effect can only in special cases be resolved by rescaling the parameters of the Poisson model.

General Discussion

In this section, we will revisit the main findings from Chapters 1 to 4 in the light of the four overarching questions outlined in the General Introduction: a) How do factors that influence the average per-capita growth rate influence the population's movement through the invasion process? b) What is the role of variability in population-size trajectories? c) What are the consequences for patterns of genetic diversity, and d) to what extent do they lead to eco-genetic feedbacks? In this synthesis, the concept of *easy* and *difficult* stages in the invasion process (see Introduction and Glossary) will play an important role. Furthermore, I will discuss the merits of stochastic models in invasion biology, and close by pointing out major challenges for future research.

Role of factors influencing the average per-capita growth rate

As we have seen in the General Introduction, many abiotic and biotic factors can potentially influence the average per-capita growth rate and thereby shape the invasion process. The focus in this dissertation has been on two factors: interspecific competition with a resident species (Wittmann *et al.*, 2013a,b, Chapters 1 and 2) and the strong demographic Allee effect (Wittmann *et al.*, 2013b,c,d, Chapters 2–4, see Glossary). Several further factors are briefly considered, for example the introduced species' fecundity and its introduction rate (see Chapter 1). From an ecological perspective at least, these two factors as well as the demographic Allee effect play an unambiguous role in the invasion process. An increase in fecundity or introduction rate increases the average per-capita growth rate throughout the invasion process and thus accelerates the population's progress. By contrast, a demographic Allee effect reduces the average per-capita growth rate, especially at small population sizes, and thus leads to an overall more difficult invasion process.

The role of competition intensity is more complex. In Chapter 1, we have observed that

the expected time until the introduced population reaches the end of the invasion process (the point at which the native species goes extinct) is minimized at an intermediate competition intensity (see Fig. 1 in Chapter 1). This finding may be astonishing at first, but begins to become clear if we recall that competition intensity has opposing effects at different stages in the invasion process (see General Introduction). Increasing the intensity of competition with the native species renders the first stage more difficult, but simplifies the second stage. In other words, for weak interspecific competition (weaker than the minimizing competition intensity, see Fig. I.2 A) the introduced population can establish readily, but then coexistence is relatively stable and it takes a long time to exclude the native species. For competition intensities above the minimizing value, on the other hand (see Fig. I.2 B), the introduced population has difficulty establishing in the first place; it may go extinct again and again, before it finally overcomes the unstable point halfway through the invasion process and then quickly excludes the native species from the system.

The competition coefficient (our measure for competition intensity, see Glossary) that minimizes the expected persistence time of the native species is close to one, i.e. the point where interspecific competition and intraspecific competition are equally strong. This result gives insight into the relative importance of easy and difficult stages in the invasion process. For essentially all values of the competition coefficient, the invasion process contains both an easy and a difficult stage. Just at a competition coefficient of 1.0, the average per-capita growth rate (not accounting for introductions) is zero at all points and thus there are neither easy nor difficult stages. This path of least resistance appears to be close to optimal from the perspective of the introduced population, suggesting that the detrimental effect of difficult stages overwhelms any positive effect that the presence of an easy stage may have.

Based on this reasoning, we might expect the minimizing competition coefficient to be exactly 1.0. This is not the case, however. Chapter 1 revealed that the minimizing competition coefficient is slightly below 1.0 for small introduction rates and slightly above 1.0 for large introduction rates. To understand this phenomenon, let us take the perspective of an introduced population and imagine that we strive to rapidly complete the invasion process. Before we can even start, we have to wait on average 1/(introduction rate) time units for the first individual(s) to arrive. If this small population goes extinct by chance, which can be quite likely, we have to wait for another introduction event. In a scenario with a small introduction rate and hence long average waiting times, such an extinction would substantially delay our progress. Starting from the invasion process at competition coefficient 1.0, we can reduce the probability of losing precious colonizing individuals to extinction by making the first stage in the invasion process slightly easy. Of course, we will then need slightly more

time at a later stage to complete the invasion process. This trade-off causes the minimizing competition coefficient to be below 1.0 for small introduction rates. If introduction events are very common, on the other hand, intermittent extinctions do not cause large delays, and introduction events even make the first stage of the process slightly easy in themselves. Hence, the minimizing competition coefficient is slightly larger than 1.0 to also facilitate the second stage of the invasion process. It is revealing that the minimizing competition coefficient crosses 1.0 at the same introduction rate at which the extinction of the introduced population in the approximating diffusion process becomes an entrance boundary. That is, for higher introduction rates, the process can start at the point where the introduced population is absent, but can never get back there. Thus, delays due to temporary extinctions appear to be negligible in this parameter region.

Two general conclusions emerge from these results. First, due to the dominating influence of difficult stages in the invasion process, understanding what factors make introduced species grow particularly fast may be less important than understanding what determines the presence and magnitude of barriers. To better understand difficult phases, we should embrace the challenging task of gathering information on invasion failures (see also Zenni & Nuñez, 2013). The second conclusion is that we should not neglect the introduction regime itself as a shaping force of the invasion process.

Role of factors influencing variability

Although stochasticity plays a role in all parts of this dissertation, two projects have focused in particular on how different levels of variability influence invasion success: We have considered variability stemming from the temporal distribution of introduced individuals ("introduction stochasticity") in Chapter 2 (Wittmann *et al.*, 2013b), and offspring-number variation in Chapter 4 (Wittmann *et al.*, 2013d). In both cases, variability strongly and qualitatively changes the way introduced populations move through the invasion process. As we have already pointed out in the General Introduction, a certain amount of variability is absolutely required for a population to overcome difficult stages, whereas the movement through easy stages can also be understood in a deterministic framework. The results in Chapters 2 and 4 provide further evidence for fundamental differences in the role of variability between difficult and easy stages. In Chapter 2, we have compared introduction regimes with the same average number of individuals introduced per time unit, but with a different temporal distribution. We found that in completely easy invasion processes, the invasion proceeded faster with a small propagule size and a large propagule frequency,

i.e. many small introductions leading to an even temporal distribution and small variability. In difficult invasion processes, by contrast, introduction regimes with a large propagule size and therefore small propagule frequency (uneven temporal distribution, high variability) led to a faster invasion. Similarly in Chapter 4, Allee-effect populations with a large offspring-number variation overcame the difficult stage below the critical population size more rapidly than populations with little offspring-number variation. In summary, variability is detrimental to invasion success in easy stages, but advantageous in difficult stages. Mixed invasion processes with both a difficult and an easy stage behaved essentially like difficult processes; also here variability was advantageous. This finding is in line with our previous observation on the dominating role of difficult stages in the invasion process.

To further quantify the effect of variability, let us consider a diffusion process with infinitesimal mean $r \cdot x$, representing density-independent population growth or decline, and infinitesimal variance $\alpha \cdot x$, representing demographic stochasticity. The population size x tends to increase if $r > 0$ (easy invasion process) and tends to decline if $r < 0$ (difficult invasion process). Using standard techniques from the theory of stochastic processes (see e.g. Karlin & Taylor, 1981, Ch. 15), Dennis (2002) derived first-passage probabilities for this diffusion process. Using his results, we can write the probability that a population with initial size N_0 reaches size z before going extinct as

$$\Pr(T_z < T_0 | N_0 = n) = \frac{1 - \exp\left(-2 \cdot \frac{r}{\sigma^2} \cdot n\right)}{1 - \exp\left(-2 \cdot \frac{r}{\sigma^2} \cdot z\right)}. \tag{D.1}$$

The plots of equation D.1 confirm the opposing effects of increasing variance in difficult vs. easy scenarios (Fig. D.1). Furthermore, the success probability evidently depends only on the ratio r/σ^2. This result suggests that it is the relative strength of deterministic and stochastic forces that determines the progress of the introduced population through the invasion process. This is quite intuitive: To overcome large barriers in the invasion process, i.e. stages with a strongly negative average per-capita growth rate, large deviations from the average population dynamics are required, whereas small barriers may be overcome already by small deviations.

In Chapter 2 (Wittmann *et al.*, 2013b), we identified several published data sets on intentional or experimental introductions that include information on invasion success as well as on propagule size and propagule frequency. Using generalized linear models for invasion success as a function of propagule size and propagule frequency (and thus indirectly on variability), as well as their product (the average inflow of individuals), we classified the underlying scenarios as difficult or easy. Although strong independent evidence was not

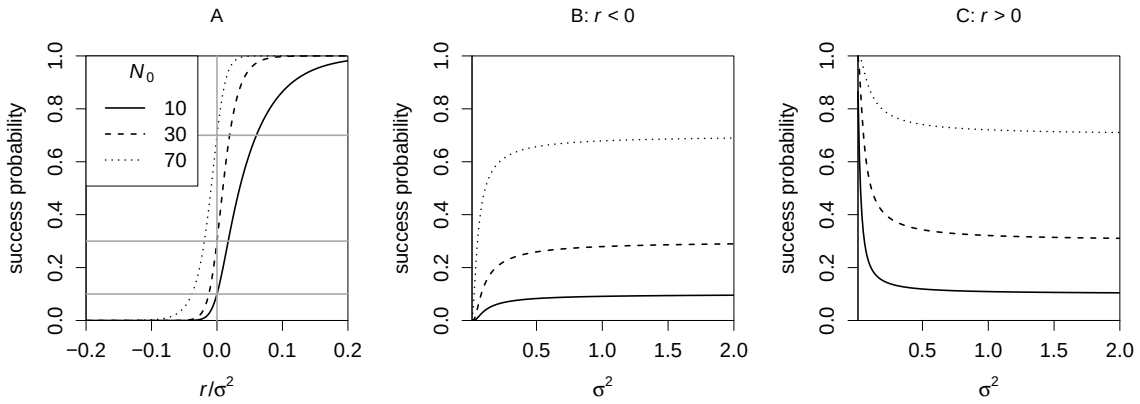

Figure D.1: Success probability ($\Pr(T_z < T_0)$, here $z = 100$) in a diffusion model for density-independent population growth or decline and demographic stochasticity. A) The success probability depends just on the relative magnitude of deterministic and stochastic forces (r/σ^2). For $r/\sigma^2 = 0$, the deterministic forces vanish and the success probability is N_0/z (indicated by gray lines). B) Under a difficult scenario, here with $r = -0.001$, the success probability increases with increasing variance, i.e. increasing σ^2. C) The opposite is true under an easy scenario, here with $r = 0.001$.

available, these classifications made sense in the light of the species' biology and the average invasion success. Asexually reproducing water fleas (*Daphnia magna*) experimentally introduced into containers with suitable medium (data from Drake *et al.*, 2005) fell into the easy scenario class. They also had a very high establishment probability. The same was true for six among seven considered dung beetle species introduced to Australia (data from Tyndale-Biscoe, 1996). These species were introduced in very high numbers to patches with abundant resources. On the other hand, biocontrol introductions of parasitoid insects (data from Hopper & Roush, 1993) were classified as difficult based on the role of propagule size and frequency. This classification was consistent with lower overall success probabilities and the potential for a mate finding Allee effect (Hopper & Roush, 1993).

Of course, the temporal distribution of introduced individuals and offspring-number variation are only two among many possible sources of variability. More generally, Sæther *et al.* (2004, 2005) suggest that the amount of variability in bird population dynamics depends on the position of the species' life history in the "slow-fast continuum". Species with a "fast" life history, i.e. those with short generation time, fast maturation, and large clutch sizes are predicted to have a higher demographic variance than "slow" species with long generation time, high age at maturity, and small clutch sizes.

Although we observed many parallels in how various sources of variability influence

invasion success, in general it can be relevant which mechanism is causing variability. Environmental stochasticity, for instance, differs from demographic stochasticity and introduction stochasticity in that its importance does not cease with increasing population size. For example, a sudden episode of favorable climate can cause all individuals in a population to produce an exceptionally high number of offspring in a given year, leading to a sudden population-size boom. Due to its importance in large populations, environmental stochasticity might be particularly important for the completion of difficult stages later in the invasion process (as for example the second stage in Fig. I.2 A).

As in many other fields of science, research in invasion biology is often focused on determining averages, for example the average number of individuals arriving at a certain location per time unit (propagule pressure) or the average fecundity of individuals in a population. Such measurements are certainly important (as we have seen in the first section of this General Discussion). However, populations with the same average behavior may differ widely in their propensity to produce the extreme events required to overcome difficult stages in the invasion process. This finding suggests that we should devote more attention to quantifying variability when trying to predict or understand invasion success in the field.

Genetic consequences

The fact that introduced populations lose genetic variation through drift in every generation, and that this loss is more severe in small than in large populations links the population dynamics and the population genetics of an introduced species. To understand the population genetic consequences of invasion trajectories, we therefore have to consider how much time the population of interest spends in different population-size ranges. In Chapters 3 and 4 (Wittmann *et al.*, 2013c,d), we have done this for populations with and without a strong demographic Allee effect and with various amounts of offspring-number variation. We focused on single introduction events, and since it is not meaningful to assess genetic diversity in extinct populations, we sampled only those populations that reached a certain high target population z before going extinct (successful populations). Our quantity of interest was the proportion of genetic variation from the source population that was maintained by successful populations upon reaching size z.

Since a demographic Allee effect by definition implies a reduction in average per-capita growth rate, we might expect that Allee-effect populations upon reaching size z have spent more time at small population sizes, experienced more genetic drift, and thus maintained less genetic variation than populations without Allee effect. This is indeed what we ob-

served, but only for the offspring-number model with the least variation. For offspring-number models with intermediate variation, this slow-down was still present at population sizes around and above the critical population size. But counterintuitively, successful Allee-effect populations spent on average less, not more time at very small population sizes compared to populations without Allee effect. Because of this speed-up at small population sizes and the slow-down at large population sizes, successful Allee-effect populations maintained on average more genetic diversity if the founder population size was small compared to the critical population size, but less genetic diversity if the founder population size was similar to or larger than the critical population size. For the offspring-number models with the most variation, the speed-up effect dominated: Allee-effect populations grew faster than populations without Allee effect at all population sizes, and thus maintained on average more genetic diversity across the range of possible founder population size. In other words, with increasing offspring-number variation, the trajectories of successful Allee-effect populations strayed further and further from the average population dynamics. Interestingly, the role of offspring-number variation was much more substantial in our models than it is in standard population genetic models. We could show in Chapter 4 (Wittmann *et al.*, 2013d) that, unlike in these standard models, it is not possible to resolve the differences between our various offspring-number models by simply rescaling the parameters of a baseline model.

In Chapter 3 (Wittmann *et al.*, 2013c), we conducted a simulation study to explore the opportunities for parameter estimation resulting from the population genetic consequences of the Allee effect. Specifically, we have attempted to estimate the critical population size from pseudo-observed genetic data. We found that it is indeed possible to estimate the critical population size, but only if genetic data from sufficiently many independent populations are available. Given the large role of stochasticity in small introduced populations, it is not surprising that independent replicates are required to determine the parameters of the invasion process. In our simulation study, we assumed all other model parameters to be known. This is of course rather unrealistic. Since the population genetic consequences of the Allee effect appear very context-dependent—the magnitude and even the direction of the effect depends on offspring-number variation—we have to caution that estimating the critical population size from genetic data is only possible if detailed knowledge about the species' biology is available.

Eco-genetic feedbacks

Also to understand the eco-genetic feedback in the impact of an introduced species on a native competitor (Chapter 1, Wittmann *et al.*, 2013a), it has been helpful to first consider the population-size regions in which the species of interest—in this case the native species—spends most of its time. We found that for small competition coefficients, the coexistence of native and introduced species is relatively stable such that both populations remain at intermediate size for a long time. For large competition coefficients, where the more common species has an advantage, on the other hand, the native species spends most of the time before its extinction at high population sizes, essentially making up the entire community. Thus, we expect a stronger reduction in genetic diversity and evolutionary potential at small rather than large competition coefficients. This is indeed what we observed. The eco-genetic feedback, i.e. the reduction in persistence time in the eco-genetic compared to the ecological model, was strongest at small competition coefficients. This led to a shift of the minimizing competition coefficient towards smaller values.

Eco-genetic feedbacks are expected also for Allee-effect populations: a small population that declines due to the Allee effect can experience a loss of genetic diversity, reducing its evolutionary potential and thus leading to further population decline. Another way to see this extinction vortex is as the interaction of two Allee effects, an ecological Allee effect (e.g. due to mate limitation) and a genetic Allee effect. The latter can be defined as a genetically-mediated reduction in fitness at small population sizes (Berec *et al.*, 2007). A special example for a genetic Allee effect is that with increasing founder population size it becomes more probable that some of the founding individuals carry alleles that will be advantageous in the new environment. In her Bachelor thesis, Hanna Stuis (Stuis, 2013) has begun to quantify the interactions between such a genetic Allee effect and a mate finding Allee effect—a project that we will continue to pursue.

Merits of stochastic models

In my opinion, one of the main merits of stochastic modeling in invasion biology is to let us constructively address questions that we would have to give up on otherwise. In fact, many authors have a very pessimistic view on the predictability of invasions. They stress the idiosyncrasy or context-dependency of invasions, meaning that every invasion is different and influenced by a large number of unpredictable historical events. For example, it seems that several species of woody plants that had been cultivated in Berlin and Branden-

burg for decades or even centuries could only spread outside cultivation after World War II bombs had opened up suitable sites in urban environments (Kowarik, 1995). This is a rather extreme example of a historical event; in other cases invasions may be facilitated or stopped by events that are difficult to predict but may still occur on a regular basis. My point here is that simply stating that invasions are context-dependent is not very productive. Since it is not feasible to delve into all the details of the processes involved—from the biochemical reactions inside introduced individuals to societal and economic developments—research usually stops at this point.

With the help of stochastic models, however, we can do better. Even if we never reach the "Holy Grail of invasion biology" (Enserink, 1999), i.e. reliable prediction of a specific population's fate, we may get valuable insights into the statistical properties of the invasion process. Ideally, stochastic models can allow us to estimate the probability that a population will successfully establish or the expected time until it reaches a certain point in the invasion process. Like other types of mathematical models, stochastic models range from very simple, but analytically tractable models to complex models incorporating extensive biological detail for a particular species of interest. The models in this dissertation tend to the former side of this spectrum. In each case, we included one or two key factors influencing the average per-capita growth rate and one or two important sources of variability. Thereby we gained novel insights into the factors that shape invasion success, impacts on resident species, as well as evolutionary and genetic aspects of biological invasions. Some of the most interesting mechanisms we discovered would not even be possible in a deterministic world but depend on stochasticity in a fundamental way.

Outlook

Based on the insights that have emerged from this dissertation, I see three important challenges for future theoretical work in invasion biology.

Large deviations

As I have argued in different parts of this dissertation, invasion biology is a field where it is crucial to understand and quantify unlikely events, namely the rare events in which a very small population grows against all expectations, and overcomes various barriers to form a large and wide-spread population, with potential impacts on native ecosystems. We need to understand better how frequent such deviations from the average population dynamics are

and which ecological and evolutionary factors affect them. It is not sufficient to know the mean and the variance of outcomes, but we need to characterize the tail of the underlying distribution, the extreme events. One mathematical approach that promises such insights is the theory of large deviations (see e.g. den Hollander, 2008).

Genealogies under stochastic demography

An analytical approach to the ecology of rare events would be even more useful if at the same time it could also provide insights into the population genetics and evolution of the population. One step in this direction would be to derive the stochastic process conditioned on invasion success. Such a conditioned process represents a concrete mathematical structure that we can analyze and simulate from. In Chapter 3, this technique revealed how much time successful populations spend on average in various population-size regions. From this, we can qualitatively understand how much genetic drift such populations experience. To actually characterize the genealogies of such populations, we first simulated a population-size trajectory forward in time from the conditioned stochastic process. For each trajectory, we then simulated sample genealogies backward in time. Compared to simulating from the original process and discarding unsuccessful runs, or even worse jointly simulating the population dynamics and the whole genetically structure of the population forward in time, our "forward-backward" approach is an advance, especially in terms of computational efficiency. However, it still does not allow us to take advantage of many of the elegant features of coalescent theory. For example, it appears impossible to derive a formula for the expected time to the most recent common ancestor of two sampled lineages. To solve this problem, we need a joint model for demography and genealogies of successful populations. Branching processes are a promising candidate since they essentially include all the genealogical information from the population. If we had a way to analyze sample genealogies in stochastically varying populations, this would of course not only be of interest for studying the genetics of introduced populations, but could be used to elucidate the footprints of various ecological processes and interactions.

Eco-evolutionary dynamics

The eco-genetic feedbacks described in the General Introduction and investigated in Chapter 1 can be seen as an example for eco-evolutionary dynamics. This term refers to situations in which evolutionary processes are fast enough to influence population dynamics, which in turn influence selection pressures and thus feed back on evolutionary processes.

Interestingly, some of the clearest examples for rapid evolution involve introduced species, possibly because introduced species are often exposed to strong directional selection in their new environment (Thompson, 1998). For instance, populations of *Drosophila subobscura* introduced to the Americas in the late 1970s have since then evolved latitudinal clines in wing size similar to those observed in the Old World (Huey *et al.*, 2005). Such evolutionary change in a focal population can have ecological consequences at different levels of biological organization: it can influence the population's own demography, the population dynamics of interacting species, or even affect ecosystem processes (Pelletier *et al.*, 2009).

Despite the importance of eco-evolutionary dynamics in biological invasions, tractable models are lacking. At first sight, the task of building an eco-evolutionary model—either stochastic or deterministic—does not appear to be too difficult. Classical models in ecology keep track of the size of a population while similar models in population genetics represent the number of copies of an allele of interest. Although analytical formulas are available only in special cases, we can always numerically compute quantities such as first-passage probabilities or expected times. Thus, one might suppose that combining an ecological with a population genetic model will be similarly straightforward. Indeed, it is often easy to write down formulas for the transition rates (as we have done for the eco-genetic model in Chapter 1). However, the fact that such models necessarily have at least two dimensions, one for the population dynamics and at least one for the genetic configuration of the population, makes it challenging to work with them. Even numerical solutions may be unfeasible due to the enormous size of the state space. In the simple two-alleles model in Chapter 1, for example, a community size of K resulted in a number of possible states of the eco-genetic model that is of the order of K^2 , and thus a transition rate matrix whose number of entries is of the order of K^4. There is an urgent need for more tractable models that include both an ecological and a genetic/evolutionary dimension. The theory of multi-dimensional diffusions (see e.g. Durrett, 2008, Ch. 8) might be one promising direction to explore.

Conclusions

Using a stochastic modeling approach, we have explored in this dissertation how various ecological factors influence the invasion success of introduced populations, their genetic diversity, and their impacts on interacting native species. Some of the factors we considered, for example competition and the Allee effect, act by shaping the introduced population's average growth rate. Others, for example offspring-number variation and the temporal distribution of introduced individuals, determine the variability of population-size trajectories. Overall, we can conclude that the expected time until an invasion process is completed depends mainly on the presence and severity of difficult stages, i.e. stages with a negative average growth rate. If there are difficult stages, it matters little whether or not there are also easy stages with a positive average growth rate and how fast the population grows in these stages. Thus, difficult stages tend to dominate the features of the invasion process. A second important conclusion is that the relative magnitude of deterministic and stochastic forces has reverse effects in easy vs. difficult stages of the invasion process. During easy stages, the population progresses faster if deterministic forces are strong relative to stochasticity. During difficult stages, however, a large amount of stochasticity relative to deterministic forces promotes invasion success. The relative strength of deterministic and stochastic forces also influences *how*, i.e. on which population-size trajectories, the population moves through difficult and easy stages, provided that it does. The population-size trajectory, in turn, determines how much time the population spends in various population-size ranges and thereby the strength of genetic drift it is exposed to. In several chapters of this dissertation, we have seen that this causal chain can lead to—sometimes counterintuitive—results for genetic variation and evolutionary potential of the introduced population as well as its native competitor. In summary, stochasticity does not just lead to fluctuations around some mean outcome, but can fundamentally affect even the qualitative behavior of a biological system. As illustrated by the above suggestions for future research, the theory of stochastic processes has many aspects that remain to be discovered for invasion biology. For these future endeavors, the scientific community needs curious and fearless ecologists and mathematicians, or—even better—collaborations between the two.

Glossary of important concepts with references to key publications

additive genetic variance the part of genetic variance in quantitative traits that natural selection can act on

Approximate Bayesian Computation (ABC) simulation-based method for parameter inference, approximates the posterior distribution of parameters of interest through a comparison of simulated and observed summary statistics (see Beaumont, 2010; Csilléry *et al.*, 2010)

biological control attempt to reduce the population size of an undesired species, e.g. an invasive species or an agricultural pest, by the introduction of natural enemies (see e.g. Fauvergue *et al.*, 2012)

birth-death process a Markov process in which transitions are only possible between neighboring states, e.g. population dynamics with one birth or one death event at a time (see Karlin & Taylor, 1975, p. 131-150)

bottleneck loss of genetic diversity due to a sudden reduction in population size (Nei *et al.*, 1975)

branching process Markov model for density-independent population dynamics; in each generation every all individuals independently produce an identically distributed number of offspring (see Karlin & Taylor, 1975, Ch. 8)

critical population size parameter of models with strong demographic Allee effect; the average per-capita growth rate is negative below the critical population size and positive above

coalescent theory modeling framework in population genetics: the genealogy of a sample from the population is constructed by following the ancestral lineages backwards in time (Wakeley, 2009)

competition coefficient strength of interspecific competition relative to intraspecific competition

demographic Allee effect positive relationship between population size and average per-capita growth rate at small population sizes (Stephens *et al.*, 1999)

demographic stochasticity fluctuations in population size due to randomness in the number of birth and death events and in sex ratio (Simberloff, 2009)

density dependence phenomenon that the average per-capita growth rate changes with population density (or size)

difficult stage stage in the invasion process in which average per-capita growth rate is negative (Chapter 2 of this dissertation)

diffusion process Markov model in continuous time whose trajectories are almost always continuous (Karlin & Taylor, 1981)

easy stage stage in the invasion process in which average per-capita growth rate is positive (Chapter 2 of this dissertation)

eco-evolutionary dynamics ecological and evolutionary processes happening on the same time scale and mutually influencing each other, thus leading to a closed feedback loop (see e.g. Post & Palkovacs, 2009)

eco-genetic feedback consequences of changes in population size for patterns of genetic diversity and the resulting feedback on population dynamics, for example through a change in evolutionary potential (Chapter 1 of this dissertation)

effective population size the size of an idealized population (usually a Wright-Fisher population) experiencing the same strength of genetic drift and sharing other properties of genetic samples with the population of interest (Charlesworth, 2009)

environmental stochasticity fluctuations in demographic parameters such as birth and death rate due to changes in the environment (Engen *et al.*, 1998)

extinction vortex synergistic feedback between different processes that can lead to a reduction in population size; can speed up extinctions (Gilpin & Soulé, 1986)

invasion process series of stages and transitions describing the progress from the arrival of an introduced population to potentially becoming a large wide-spread population (Blackburn *et al.*, 2011)

invasion success used here as a general term for success at any stage in the invasion process

invasive species controversial concept that can stand either for introduced species in general, or only for those that are abundant and widespread, or only for those that have negative impacts; I therefore avoid it in this dissertation

joint site frequency spectrum (JSFS) generalization of the site frequency spectrum to a situation where n_1 lineages are sampled from one population and n_2 lineages from a second population: array containing the numbers of derived mutations that appear i times in population 1 and j times in population 2 for $i \in \{1, \ldots, n_1\}$ and $j \in \{1, \ldots, n_2\}$ (Tellier *et al.*, 2011)

lag time a time period during which an introduced population grows more slowly than it does later (Crooks, 2005)

likelihood mathematical function describing how the probability of observed data depends on model parameters

Markov model stochastic model whose behavior at any point in time only depends on the current state of the system and not on its history

Markov process stochastic process in continuous time, usually with a discrete state space, characterized by the rates at which different types of events occur, e.g. birth and death events (see Karlin & Taylor, 1975, Ch. 4)

per-capita growth rate per-individual rate of population growth; in the case of non-overlapping generations it can be approximated as $(N_{t+1}/N_t) - 1$, where N_t is the population size in generation t

propagule frequency the frequency at which introduction events happen, i.e. the average number of introduction events per time unit

propagule pressure quantifies the inflow of individuals of an introduced species to a certain location; often defined as a measure with two components: propagule size and propagule frequency (Lockwood *et al.*, 2005)

propagule size the (average) number of individuals per introduction event (Lockwood *et al.*, 2005)

site frequency spectrum vector with the numbers of derived mutations that appear $1, \ldots, n$ times in a sample of size n (Wakeley, 2009, p. 102-106)

strong demographic Allee effect a demographic Allee effect in which the average per-capita growth rate is negative at small population sizes (Taylor & Hastings, 2005)

weak demographic Allee effect a demographic Allee effect in which the average per-capita growth rate is reduced but still positive at small population sizes (Taylor & Hastings, 2005)

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