Understanding how evolution affects the spatial dynamics of interacting species

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Selective pressures on organisms are constantly changing due to multiple factors, such as climate change and the introduction of exotic species. Empirical evidence shows that interspecific interactions and evolution can have important effects on species distributions, independently or simultaneously. We are thus in need of models capable of describing the effects of both these factors, in order to better understand the drivers of species geographical distributions in a changing world.

In this thesis we explored the effects of adaptation to environmental gradients under two different interaction scenarios: in a monospecific context and for a two-species predator-prey community. In a third time, we studied the effects of adaptation to pathogens in an invasion scenario, where three species interact: a native population, and an exotic competitor co-introduced with a pathogen. The studied models reveal the effects of adaptation and interaction through eco-evolutionary propagation speeds that are closely linked to the species' adaptation potentials. We discuss implications of these results for description and interpretation of currently utilized Species Distribution Models.

Keywords: quantitative genetics, boom and bust dynamics, PDE models, spatial heterogeneity, environmental cline, reaction-diffusion equations, theoretical ecology, evolutionary ecology

RÉSUMÉ

Understanding how evolution affects the spatial dynamics of interacting species

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Les pressions sélectives sur les organismes étant en perpétuel changement, en conséquence de multiples facteurs tels que le changement climatique et l'introduction d'espèces exotiques, il est indispensable d'avoir une meilleur compréhension des dynamiques déterminant les distributions spatiales des espèces. L'évidence empirique montre que les interactions inter-spécifiques et l'évolution peuvent avoir des effets importants sur la distribution des espèces, individuellement ou simultanément. Il est donc nécessaire de disposer de modèles capables de décrire l'effet de ces facteurs afin de mieux comprendre les déterminants des distributions géographiques des espèces dans un monde qui change.

Au cours de cette thèse, nous avons étudié les effets de l'adaptation à des gradients environnementaux dans deux scénarios d'interaction: dans un scénario monospécifique et dans le cas d'une communauté à deux espèces dont une proie et un prédateur. Dans une troisième partie, nous avons étudié les effets de l'adaptation à des pathogènes dans un scénario d'invasion où trois espèces interagissent : une population native et un compétiteur exotique co-introduit avec un pathogène. Les modèles étudiées révèlent les effets de l'adaptation et de l'interaction à travers des vitesses de propagation eco-évolutives qui dépendent fortément des capacités d'adaptation des espèces considerées. Nous discutons des implications de ces résultats pour la description et l'interprétation des modèles de distribution d'espèces (SDMs) utilisés actuellement.

Mots-clés : génétique quantitative, dynamique boom-and-bust, modèles d'EDPs, hétérogénéité spatiale, cline environnemental, équations de réaction-diffusion, écologie théorique, écologie évolutive

CHAPTER I

Introduction

It has now become of critical importance to understand and treat the effects of global changes. On the one hand, these changes are a new source of evolutive pressures on existing species which can have direct effects on some ecosystems (Daufresne et al. 2009); while on the other hand, they can have indirect effects, such as the impact the geographical distribution of some species (Parmesan and Yohe 2003) and altering the way some species interact (Tylianakis et al. 2008). It is undoubtedly important to understand the consequences climate changes can have on known species, but there are also important effects that appear as interactions of those factors. The main motivation of this work is to understand these simultaneous effects given by evolutive pressures and interspecific interactions on the spatial dynamics of species, in order to have a better understanding of their dynamics and thus have a better idea of our expectations and predictive capacities faced to ongoing global changes.

The purpose of this chapter is to introduce the concepts, phenomena and basic ideas that motivated this work. I begin by reviewing problems that are intimately linked to the spatial structure of ecological populations and communities, and then define the concepts and historical approaches used to understand the influence of space on the distribution of species. This view has changed from considering species distributions as being purely determined by local abiotic factors, to being modified

by different interspecific interactions and dispersal processes. Evolution can also play an important role in the spatial structure of existing populations at relatively large spatial scales, be it either given by adaptation to the environment or to different interacting species in a community. I then move on to theoretical models that have been used to understand the spatial dynamics of known populations. These models have progressively become more specific in the problems they treat, so as to being able to include different relevant factors. I review the ways in which several important biological factors, such as interactions and evolution, can be included in these models. I thus set up a framework to study problems in spatial biology taking simultaneously into account the structure of space, the impact of interspecific interactions and different types of adaptation. We finish by contextualizing the works in this thesis based on this framework.

1.1 A few words on current challenges in understanding species distributions

The world has always been in constant change, but humans have played an important role in the changes the world has experienced in the last decades. Amongst the most severe manifestations of anthropogenic global change, fragmentation, climate change and the introduction of invasive species are some well-known examples. While these three phenomena have different direct causes, they share a common consequence: they are intrinsically related to species distribution patterns. Parmesan and Yohe (2003) observe that a large number of species have shifted their geographic ranges in directions predicted by climate change; while Foley (2005) indicates that use of land for crops, pasture or plantations, and the expansion of urban areas, have brought along considerable losses in biodiversity (see also Murphy and Romanuk (2014) for an estimation of biodiversity losses due to human disturbances).

Landscape fragmentation is understood as a reduction in accessibility to available space where a large expanse of habitat is transformed into a smaller patches of smaller areas isolated from each other (as defined by Wilcove et al. 1986). This is directly linked to the use of land for cities and agriculture (Foley 2005). There are two ways in which fragmentation can affect the distribution of species: by changing the quality and availability of resources, for example as a consequence of contamination by pollutants or the use of land for human-driven activities such as agriculture or urbanization; and by directly impeding the access to suitable localities (i.e. source patches sensu Pulliam (1988)). The first factor has an impact on local population fitness, while the second one affects its dispersal.

Climate change is associated to a steady increase in global temperatures in the last decade. Rosenzweig et al. (2008) have shown that most of the observed changes in physical and biological systems are due to anthropogenic climate change. Species are expected to follow the direction of climate change so as to track their niche requirements (sensu Hutchinson (1957), refer to the box on Ecological Niches for the definition), and thus climate change would shift their geographic distributions. Some species have been observed to respond this way, with species inhabiting restricted ranges being the most affected ones and even being driven to extinction in some cases (Parmesan and Yohe 2003; Parmesan 2006). Nevertheless, sometimes the range shifts do not occur in the expected direction, and climate change can have an impact on the way species interact, making the task of prediction more difficult.

The introduction of exotic species is mostly a human-mediated phenomenon (Vitousek 1997; White et al. 2006), and sometimes it can have devastating consequences. Exotic species are not a problem by themselves, but when they increase uncontrollably in number invading large proportions of space, their growth impact other species in invaded ecosystems (David et al. 2017). These are known as invasive species. Two important challenges involving invasive species are (i) to predict the extension of

invaded space, and (ii) the way they will interact with local species to which they were never exposed to. Species invasions thus constitute an important problem in the context of spatial ecology (refer to the box on Invasive Species for more details).

Invasive species do not only affect communities through direct interaction with local species (competition, predation, parasitism), but they may also provoke indirect effects through mechanisms such as apparent competition and trophic cascades (White et al. 2006). The effects of these disturbances, thus, may be difficult to predict, since they depend on the affected trophic level (David et al. 2017).

Trophic disturbances or competition with local species are not the only way in which invasive species can locally alter the dynamics of a community, they can also have an important impact on the environment, modifying the availability of resources for this community and creating changes in the habitats. This way, the problem of ecological invasions asks for a framework capable of dealing with multiple factors simultaneously.

Invasive species

Invasive species are species introduced into a new environment that are capable of persist and expand their geographic distribution. As they enter a new environment, they establish new interaction with native species, and they may evolve traits to adapt to newly encountered conditions either biotic or abiotic. Thus the dynamics of invasive species naturally include the components that motivate this thesis: changes in spatial distribution, interspecific interactions and adaptation.

Invasive species are a very interesting topic in ecology because invasion episodes can help us understand several hypotheses about interspecific interactions, community assemblage, population genetics and spatial dynamics.

An invasive species has the peculiarity that, as it has evolved in a different environment, it has not co-evolved with the community where it is introduced.

It can thus produce unexpected changes in these communities, since native species do not have the necessary defenses to counteract them when interactions have negative effects. The results then can be catastrophic.

Invasive species usually experience a series of stages upon introduction describing their growth dynamics, namely: survival in transport, establishment in new areas, lag period and spread (Mack et al. 2000). Usually when an exotic species is introduced and passes through the lag period, it is difficult to determine whether it will become invasive or not. Sometimes after these lag in growth they experience a rapid growth, followed by a sudden decrease. These dynamics have been named boom-and-bust.

The ecology and spatial dynamics of ecological invasions need to be understood carefully in order to successfully predict the possible consequences of introducing new species into communities and spaces of interest, and to take efficient control policies after introduction. Although some researchers suggest that removing introduced species sometimes may be detrimental to native communities (David et al. 2017).

Species are expected to react to adapt to new environments through evolutionary changes. Evolution is usually thought to be a slow process, but in the light of these human-driven changes, some species have been observed to respond accordingly (Reznick et al. 2019). While adaptation to local abiotic conditions is expected, species can also adapt to pressures from other interacting species. The mixture of these factors is important and can have important effects on the spatial dynamics or structure of species, but its impact is not so well understood compared to abiotic pressures.

I have described perturbations as affecting organisms or environment, but there may also exist other indirect effects that are more difficult to observe. Novel selective pressures can push individuals to modify their behavior in ways that are not easy

to predict, either facilitating or hindering persistence (Wong and Candolin 2015). In turn, the feedback loop of individuals with their environment can also be modified as a consequence of these perturbations (e.g. Loeuille and Leibold (2014)).

Although here I have highlighted the impacts of human-mediated perturbations on the environment because of its relevance in current global context, perturbations need not be of anthropogenic origin: repeated episodes of climate change (such as glaciations) and biological invasions occur naturally. The question of understanding the ecological and evolutionary consequences of invasions and climate changes on species and populations is thus of very general scope and is not reduced to understanding the consequences of anthropogenic perturbations.

All of the discussed problems are also interesting because they are directly linked to modifications of biodiversity, to which we assign value (Costanza et al. 1997). Ecosystem services provide implicit benefits, and while we do not fully understand the mechanisms driving their dynamics, it is difficult to predict how big a perturbation an ecosystem can normally endure without changing considerably (Hooper et al. 2005).

Important examples ecological-driven phenomena with direct economic consequences consist of damages to crops, fisheries and epidemics among others; see e.g. Pimentel et al. (2001), Lovell et al. (2006), Simberloff et al. (2013), and Bradshaw et al. (2016). Thus, the anthropogenic disturbances we described have consequences which are important even from areas that are not directly related to ecology.

1.2 Facing the challenges: predicting and managing changes in species distributions

All of these problems have been observed and well studied and we have useful predictive tools that allow us to take action on them, but mechanistic frameworks allowing us to interpret possible outcomes are still needed (see e.g. Mouquet et

al. (2015)). Ecology has dealt with spatial structure problems in different manners along time, each one enlightening our understanding of species distributions.

There are two major classes of drivers thought to have important direct effects on species distributions, which are useful to propose a first approach to understanding the geographic arrangement of species on Earth, namely heterogeneity of environmental features and dispersal processes. The first factor is thought to play a role because species have different needs for survival defining its ecological niches (refer to the box on The Ecological Niches) which can vary a lot among taxa or even between related species, while dispersal limitation explains why some populations remain limited when they are expected to be found elsewhere based on their niche. We review the theoretical importance of both factors here.

The Ecological Niche

There is not a sole definition of Ecological Niche. Several authors have proposed different definitions, depending on the focus of their studies. Here I review some interesting definitions that lead to different ways to interpret the relation between species and their geographic distributions.

- **The niche as a place in the environment**. This first definition was proposed by Grinnell (1917), who defines the niche as the place a species can occupy inside a given environment. This implies that similar environments should harbor similar communities, and that species are thus determined by their environments. Two species occupying the same niche in different localities are called ecological equivalents (see e.g. Harmon et al. (2005) for ecological convergence of different lizard species in the Greater Antilles).

  Following Grinnell's idea, species distributions should be completely determined by the abiotic conditions of the environment, so that a full

description of the planet's biotopes should naturally determine where one could find each species or their equivalents.

- **The niche as the role of a species**. Elton (1927) takes a different view by proposing the niche to be a property of each species and not of the environment. Following Elton, the niche of a species is the role or function it fulfills in a trophic chain of a community. This notion is also termed the functional niche of a species. In this sense, plants, hervibores and carnivores of first and upper orders necessarily fulfill different niches in a community, as they are links in different positions inside this chain.

  For example, Elton 1927 mentions the arctic fox which subsists on eggs of guillemots, and the remains of seals left by polar bears; while the spotted hyena that destroys a large number of ostrich eggs and lives upon the remains of zebras killed by lions. Since they both fulfill a similar role in their respective communities, they occupy the same niche.

  Elton's functional niche does not really establish a way to determine the geographical distribution of a species, but it changes the focus from the environment to the species.

- **The niche as the conditions for persistence**. Hutchinson 1957 refines the view of the niche as a species property, by focusing on the conditions it needs to persist. Suppose there are a series of environmental variables (e.g., temperature, humidity, acidity, etc.) on which the growth rate of a species depends. The niche of a species is then defined as the set of all the combination of values that allows it to persist, i.e., that allow for the growth rate to be non-negative.

  Although this definition is precise, it can be cumbersome to accurately

determine the ecological niche of a given species. The definition implies that one should measure the growth rate of the species of interest for each combination of environmental variables; this could be cumbersome even for bacteria.

It is also important to remark the difference between the **fundamental niche** of a species, as originally defined by Hutchinson, and the realized niche of a species. In nature, one should only find species where they are able to persist, so that they are inside their niche, but their distribution may be disturbed because of interaction with other species or barriers to dispersal. The **realized niche** are the values for the environmental variables where the species can be found in nature, and it is a subset of the fundamental niche as it is disturbed by ecological interactions (Notice, however, that when dispersion is important the abundance may be important even outside the realized niche (Pulliam 1988)).

In spite of its limits, Hutchinson's niche is the theoretical basis for the widely used species distribution models or niche envelope models. Refer to the box on niche models for more details on this.

- **The resource-utilization niche**. Macarthur and Levins 1967 define the ecological niche of a species in a consumer-resource scenario as the utilization curve of such a resource. That is to say, the niche is composed by a feeding center which is the typical consumed resource, and a variability associated to its consumption. Two competing species may consume a similar resource up to a certain limit: when the consumption profiles are too similar (i.e. when there is too much overlap in their utilization curves) they may perish due to competitive exclusion. The definition of the resource-utilization niche gives a way to explicitly find the maximum

degree of similarity of two species so that they can coexist.

An important implication of this theory for spatial ecology is that potentially competing species should be more similar in allopatry than in sympatry, i.e., when they do not share space and are thus not limited by the utilization of the same resources (see e.g. Klawinski et al. 1994).

- **Neutral view of the niche**. A completely different approach is taken by Hubbell in his Neutral Theory (Hubbell 2001, 2005). This theory proposes that inside a community, similar individuals (in the same trophic level) have the same competitive ability. In this sense, the usual definitions of niche do not matter since different species would have the same niches. Neutral Theory explains the differences in species abundances inside a community as being purely determined by randomness, a phenomenon called community drift.

  What is useful about this theory is that it is testable. It predicts certain abundance patterns, which are not determined by competitive abilities.

- **Synthesis: the species and resource niche**. Chase and Leibold (2003) propose another definition of niche in the line of Hutchinson's. They acknowledge the fact that individuals have an impact on the resources they utilize, which can be important in some cases. The niche is defined thus as the joint description of the environmental conditions that allow a species to have a positive growth rate along with the set of per capita effects of that species on these environmental conditions.

1.2.1 The heterogeneity of environmental variables as a spatial determinant of species distributions

Environmental heterogeneities exist since not every place on Earth is the same but also because of the changes induced by human activities (as exposed in the previous section). Different species will have very different requirements survival and reproduction, depending on the environment where they can be found. The degree to which environmental heterogeneities affect the distribution of species can be understood through the different proposed definitions of ecological niche (cf. box on Ecological Niches).

A first approach to spatial ecology is motivated by Grinnell's definition of the ecological niche (Grinnell 1917). The niche is a property of the environment, understood as the places the species can occupy. In other words, a community is the result of the abiotic conditions in a particular environment. The study of spatial ecology is thus simplified to a description of the many different "habitat types" one can find on Earth, called biomes (see, e.g., Woodward et al. 2004).

However, one can find different communities in similar environments, so that Grinnell's niche is too rough of an approximation. Hutchinson's definition of niche tackles this problem by defining the ecological niche as a characteristic of a species rather than the environment.

The relevance of this idea of the niche is that it provides a useful and explicit way to define the environmental characteristics allowing for a species to persist. Thus, the distribution of a species is given by the environments that allow for all the conditions allowing the persistence of such a species. This is a powerful idea that has motivated the development of species distribution models (SDMs), which are very useful despite their limitations.

While SDMs provide a good way to summarize the direct determinants of a species geographical distribution, sometimes additional understanding on the indirect factors

may be needed to correctly interpret the results upon estimating the niche of a particular species (David et al. 2017, refer also to the box on Niche Models for an example). SDMs also neglect evolution, so they may not be suited to predicting distributions on long time scales or for quickly adapting species. These factors are thus interesting to study.

Niche models

Niche models are a way to explicitly model and approximate the niche of a given species so that its geographical distribution can be predicted. They are largely based on Hutchinson's definition of the ecological niche of a species, so that niches are understood as a characteristic for each species, and they deal with the conditions allowing for this species to persist.

Niche models are based on localized geographic measurements of a certain species presence/absence data, and environmental variables such as temperature, precipitations, humidity, etc. The presence or absence of such a species is then modeled as a response variable for the environmental ones, so that niche models can be understood as statistic correlative models.

The justification behind such approach depends on a series of hypotheses, which link the measured variables to the theoretical definition of niche. Namely:

- Spatial niche equilibrium: the species has filled all of the geographic locations where its niche can be found.
- Evolutive niche equilibrium: the species is well-adapted to every set of conditions where it can be found.

The major problem in niche modeling is that these two hypotheses are not necessarily met in nature. Spatial niche equilibrium implies every species has had the time to move everywhere in space where its fundamental niche can be found. This is thus the equivalent to assuming that it is not limited by dis-

persal. Another limitation is that it neglects possible interspecific interactions having a negative impact on geographical distribution, i.e., there may be areas where a species could persist, but it cannot be found there because of competitive exclusion, or presence of a fierce predator, etc. The second hypothesis is evolutionary: it assumes that the fundamental niche is fixed in time; in other words species are not expected to evolve, and their realized niche would correspond to the fundamental niche (refer to (Guisan and Thuiller 2005) for an introduction to SDMs).

In spite of their limitations, niche models seem to be good to predict possible areas for introduction of foreign populations. It has been observed, however, that in some cases of invasion some species have shifted their realized niche with respect to their native locations, and the differences are not easily attributable to ecological factors (thus a different realized niche inside the species fundamental niche) or to evolutionary factors (thus a shift in fundamental niche). An example is given by Broennimann et al. (2007).

In illustrative example showing the potential limits of climatic envelope models is given by the modeling of the cane toad's niche (*Rhinella marina*). This is an invasive species in northern Australia that is natively found in South America. Tingley et al. (2014) approximated the niche of this toad in these two different locations separately and compared the realized distribution on one continent as predicted by the model on the other continent.

They found that when the training set was the native range they failed to predict some locations where it is found as an invader. But by doing the inverse processes, the invasive range data predicted a much larger area in the native continent range. Closer inspection of the ecology of the toad in its native range revealed that its southern range is limited not due to niche restrictions, but to

hybridation with a close species (*Rhinella schneideri*).

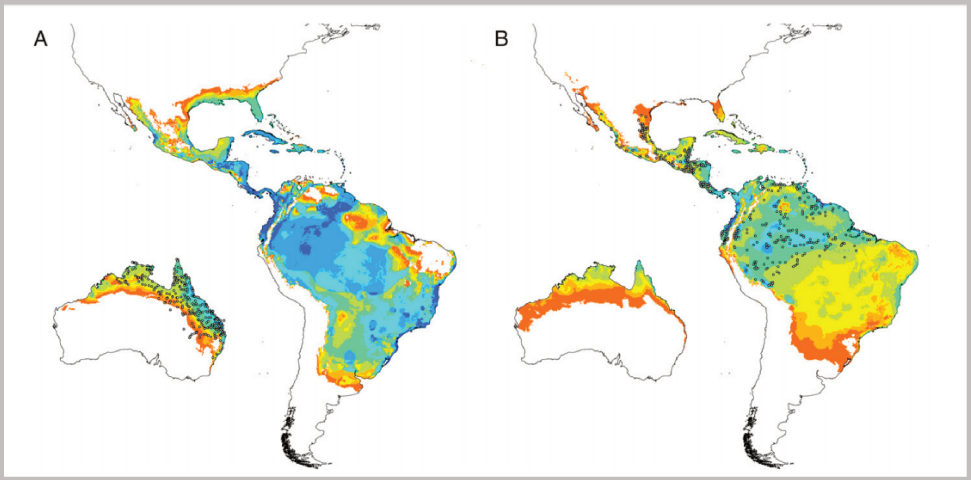

Figure 1.1: Niche of *Rhinella marina*. The image at the left shows the geographical extension of this species' niche when calibrated in its invasive range (Australia), overestimating its realized geographical range. The image at the left shows the extension of the niche in space when calibrated in its native range (South America), underestimating its realized invasive range.

1.2.2 Dispersal processes

An important framework to understand the way populations are placed along space is metapopulation theory, initially proposed by Levins (1969). This view treats space not as a continuum defined by its heterogeneities, but rather as a collection of patches where different populations of a same species can survive.

Levins (1969) proposed that there are two variables controlling the dynamics of interconnected patches, which are migration m and extinction rate e. An important conclusion of his model is that the proportion of occupied patches will be positive whenever the overall migration rate is bigger than the extinction rate of individuals,

and the occupied area (i.e., the proportion of patches) occupied by the species at equilibrium is $p^* = 1 - e/m$. Notice that the proportion of inhabited patches is an increasing function of m, i.e., the stronger the dispersal, the more inhabited patches there will be. This equilibrium is valid only when $m > e$ so that the overall migration rate must be bigger than the extinction rate.

An interesting feature of this model is that the extinction rate of species has been measured empirically, and it has been found that this rate decreases with increasing area of habitat patches, and that colonization rates decrease with isolation (Hanski and Gilpin 1991). This implies that bigger patches are expected to have more species than small ones, and the same pattern should be observed for better connected patches (MacArthur and Wilson 2001).

Dispersal is interesting because it allows individuals to expand their ranges, but it implies they can potentially travel to zones where they are not well-adapted to, incurring in fitness costs. This phenomenon was studied by Pulliam (1988) from a theoretical point of view: he studied the effects of dispersal on population dynamics in a patch system. An important conclusion he found is that patches where population are able to survive and produce a positive net amount of individuals, called sources, may have less individuals than patches where the opposite is true, i.e., where population density decreases in the absence of dispersal, dubbed sinks. This is a possibility when sources have limited space with breeding sites decreasing in quality as they become crowded, so that sinks are comparatively better.

An important observation for Hutchinson's definition of ecological niche is that Pulliam's study implies that, when dispersal is important, some species may be found outside their fundamental niche. In other words, species may be found in sinks (which by definition are outside the fundamental niche) sustained only by sources. This has important implications for niche envelope models, because they are based on species presence and absence data to estimate the fundamental or realized niche of the species

in question.

1.3 Effects of evolution on spatial distributions

Adaptation to the environment is another important factor to be taken into account when studying the geographical distribution of species. If the environment is a source of selective pressures on different species, populations may become adapted to local conditions when natural selection dominates other evolutionary forces. As individuals encounter new conditions affecting their fitnesses, populations should be able to adapt, which may help them to expand their ranges.

An important implication of this fact in relation with Hutchinson's definition of niche is that it allows niche to change over space and time, as adaptation allows persistence under new environmental conditions, thus expanding the niche with time.

Gene flow is caused by the fact that individuals in a population that has adapted to local conditions migrate to neighboring populations where environmental conditions are different (for example, mountaintop-adapted angiosperms dispersing down the slope towards valleys and nearby plains). These immigrants provide genes that are not optimal for this new population, thus bringing maladaptation to its progeny. In other words, this population's adaptation to local conditions becomes diminished due to dispersal.

In this sense, immigrants coming from central populations towards a species' range edge populations may keep the latter from adapting to local conditions and thus hamper colonization of new areas beyond the edge. This phenomenon would explain range edge limits as an equilibrium between local adaptation and gene flow (Pease et al. (1989), García-Ramos and Kirkpatrick (1997), Kirkpatrick and Barton (1997), and Alleaume-Benharira et al. (2006); see also Case and Taper (2000) and Norberg et al. (2012) for multispecies extensions).

Gene flow can thus genetically link populations that are placed along an environ-

mental cline, also causing them to exhibit phenotypical clines whose relation to the environmental one is given by dispersal. For example, populations of drosophila in South America have been observed to be adapted to local temperatures, establishing a genetic cline along space (Balanya 2006).

Conversely, the lack of genetic flow between populations of the same species may cause them to differ as time passes, as a consequence of adaptation to local conditions. This is a proposed explanation for speciation.

These examples illustrate that even when gene flow is not an important factor limiting the spatial range of species, it can have non-negligible consequences on the phenotypic or genetic structure of a species along space.

While the equilibrium between local adaptation and maladapted gene flow is a possible explanation for the limited range of a single species constrained by environmental factors, this approach still fails to include the effects interspecific interactions can have.

1.4 Effects of interactions on spatial distributions

The theoretical elements presented in the previous sections can help understand why we can find species where they are, explaining the relationship to the different abiotic components of their habitats and variations in abundance due to dispersal and intraspecific interactions. However, they do not consider interactions with different species.

Interactions are understood as the effect of one species on itself (intraspecific interaction) or on another species (interspecific interaction), usually measured through effects on fitness or abundance. Ecological interactions can be generally classified among different categories which include competition, predation, mutualism, etc. The definition of interactions suggests that the spatial distributions of species may be modified due to the presence of another one. The typical one-species approaches

usually link distribution exclusively to abiotic factors and thus overlook interactions. Intuitively, these interactions are expected to affect species distributions, because, for example, specialist predators cannot survive without their prey, so their distribution must necessarily be linked. There are efforts to include co-occurrence of species at the geographic spatial scale, but they are mostly phenomenological (Ovaskainen et al. 2016).

Competition between two species is understood as a mutual negative impact. Theoretically, when one species is able to use resources more efficiently than the other one, it can drive the other one to extinction, a phenomenon known as competitive exclusion. Some communities of Andean birds are thought to be limited through competition (Terborgh and Weske 1975), thus revealing the importance of this interaction in species distributions.

In theory, species that are exclusively predatory cannot survive without their preys, so that it would be impossible for a predator to survive in an area where preys are absent (unless other processes such as dispersal from nearby populations can explain it), even if abiotic conditions are not so restraining as to restrict their fitness. This is thus another factor important to be considered when studying and explaining species presence and abundance patterns.

Another important interspecific interaction to consider is mutualism, where two species can profit from the presence of one another. Typical examples of mutualism include plant-pollinator relations, where one species, the pollinator, carries the pollen of a plant from one plant to another and thus increasing its fitness by actively dispersing its genetic material. The pollinator is benefited as it uses pollen as a source of food. This interaction can have positive effects on agriculture. See Ollerton (2017) for a review on pollinator diversity.

This list is, of course, not exhaustive. Other kinds of interspecific interactions include commensalism, herbivory, parasitism, etc. They are not less interesting to

study than the already mentioned ones, but they differ only in some details and they can explain abundance differences in a similar way.

Striking examples of complex interspecific interactions modifying local abundances of species, even to the point of extinction, are given by invasion scenarios. Consider for example the cases of the invasion of the Asian ladybird in Europe and North America (Vilcinskas et al. 2013) and the red squirrel in the United Kingdom (Darby et al. 2014). First studies of these invasions explained the success of the foreigners in terms of their competitive abilities, however it has been discovered that both these invasions are atypical due to the fact foreign species carried pathogens that were lethal to natives while not affecting them. This case exemplifies the great importance of considering interspecific interactions when studying spatial distributions.

The following box presents four cases where evolution and interactions simultaneously influence the characteristics of the involved species.

Examples of evolution and interactions influencing Spatial Distributions

There are some well-documented cases where abiotic conditions alone are not enough to explain the observed distribution of species, and interspecific interactions or local adaptations on their own are not satisfying. There is thus a mixed effect of local adaptation and interactions. Here we present four examples, which are an important motivation for the ideas developed in this thesis.

1. The case of the invasion by the **cane toad** in Australia. There are several interesting factors playing a role here. On the one hand, toads have been observed to evolve morphological characteristics allowing them to disperse faster, with better dispersers being important composers of the invasion front. On other part, they have an influence on species that predate on them, such as black snakes, selecting them for smaller heads and longer bodies (B. L. Phillips and R. Shine 2004; Ben L Phillips and Richard Shine 2006; Phillips et al. 2006; Urban et al. 2007).

2. The **brown argus butterfly**, which inhabits Great Britain, was thought to have reached its equilibrium north range, and it was usually understood to be a specialist feeding as larvae on rockrose (*Helianthemum mummularium*). After the 1980s, this butterfly was seen to have extended its northern rage, with northern populations feeding on Geranicaeae. The alteration of the interactions in this case are thought to be a consequence of warm summers, which allow butterflies to lay more eggs. The increased egg-production allowed for the selection of butterflies laying eggs on Geranicaeae, altering the previosly known interspecific interactions (Pateman et al. 2012).

3. The **Australian soapberry bug** (*Leptocoris tagalicus*) used to feed on the native 'woolly rambutan' (*Alectryon tomentosus*). This insect feeds by probing its beak into the fruits this plant produced, and sucking on its seeds. The invasive balloon vine (*Cardiospermum grandiflorum*) produced fruit with a bigger distance between the capsule wall and its seeds, selecting for insects with bigger beaks (Carroll et al. 2005).

4. Another study examined the coevolution between **garter snakes** (*Thamnophis sirtalis*) and toxic newts of the genus *Taricha* by measuring tetrodoxin (TTX) resistance. The level of resistance to TTX in garter snakes varies with presence of these toxic newts, which present diverse degrees of toxicity depending on their particular species. This study found evidence for a mosaic of variation in TTX resistance, which is consistent with a mosaic of hotsposts and coldpsots of evolutionary response by snakes. They found patterns of clinally varying resistance to the toxin, with hotspots coinciding with zone where toxicity is more pronounced, hence stronger selection, and coldspots coinciding with zones where selec-

tion is suspected to be absent, as no known toxic news have been found. A possible explanation for the clines joining these zones is gene flow (Brodie et al. 2002).

1.5 Theoretical approaches to spatial ecology

We have already presented the conceptual framework detailing why the simultaneous consideration of interspecific interactions and evolution can have an important effect on the spatial distributions of species. Now we turn to theoretical tools that have helped us understand the importance of space on ecology.

We distinguish two types of spatial models: those who are spatially implicit and those who are spatially explicit. The importance of the former ones is that they shed light on important concepts such as dispersal and fragmentation while being simple, but they do not provide explicit descriptions of species area distributions. They do however offer partial information such as quantity of space (proportion of patches) that are occupied. The latter ones deal with these problems, and here we present some examples capable of dealing with the factors we have described. This thesis follows the formalism of these spatially explicit models.

1.5.1 Spatially implicit models

Spatially implicit models used to understand the composition and spatial distribution of species usually distinguish monospecific and plurispecific cases. When referring to the distribution of a single species and the way different population of such a species are linked through dispersal, we speak about **metapopulations**; but when studying the distribution of several species structured along several communities, we speak of **metacommunities**.

1.5.1.1 Metapopulations

Consider a series of patches, i.e., space locations that are accessible from one another but that at a certain distance so that individuals can not move freely between them, only occasionally. The measure of connectance between patches is usually called dispersal in this context, and it is related to the probability of individuals crossing from one patch to another. Metapopulation theory studies the proportion of patches that a single species can occupy, as a function of its dispersal capabilities and death rates.

The importance of patch models is that they recognize the importance of dispersal as a new driver of species persistence, and it can be a trait subject to natural selection. Holt (1985) proposed a model to study this question for two patches of different quality, and concluded that dispersal should be selected against. However, Pulliam (1988) proposed a framework accounting for immigration, emigration, births and deaths of local populations, and density dependent fitnesses at the interior of each patch. In his model, there is an additional fitness gain by dispersing from one patch to another whenever good quality nesting sites are scarce. He concluded dispersal is favored in these circumstances.

Although simple and descriptive, there are some drawbacks to these patch occupancy models. For example, they do not consider transition dynamics, so that, in the absence of dispersal, populations are always in ecological equilibrium. They do not consider space explicitly either, so that patches are treated equally as long as dispersal between them is the same. There is not an obvious measure of distance between patches.

This last point is important, as today's ecology problems ask for approaches that are explicit, or that let us at least obtain a concrete approximation for distribution areas. The abstract approach to a species dispersal does not let us study dispersion as a phenotypic trait of a species either.

1.5.1.2 Metacommunities

Metacommunity theory treats the problem initially posed by Gause, who proposed that in a community where two species compete fiercely, one is bound to disappear due to competitive exclusion (Gause 1934). In nature, however, several communities seem to be composed of similar species feeding on similar resources, which puts the competitive exclusion principle into question. Metacommunity theory posits that two such species can coexist even if one is competitively stronger, whenever space has an important effect on its presence. Thus, if the strongest competitor is a bad disperser, the weaker competitors can survive in a patch provided they arrived there first and immigration is not strong.

While this subject had been treated in previous works, Levin (1974) proposed a clear model that accounts for patch occupancy by two different species with different dispersal rates. In his framework he analizes the case where both species have the same diffusion and similar competition rates.

Levin (1974) shows that there is a big importance of the influence of space on the structure of ecological communities, since he found that for dispersing populations, Gause's competitive exclusion principle may be violated, as species coexistence is maintained through dispersal. Moreover, he showed that dispersal also has an impact on species abundances, with bigger dispersals increasing a species' density on neighboring patches. Here we only described patch dynamics, but other paradigms are possible (see e.g. Leibold et al. (2004)).

This result puts a new question on the table, as the mechanism suggested by dispersal to maintain diversity is through dispersal from source patches. In other words, there are patches that produce a surplus of individuals which then migrate to neighboring patches, and their existence if supported this way in spite of competitive decreases in their fitness. If dispersal suddenly stopped (for example, if patches became disconnected), then these local populations would perish, as the dynamics of

single patches follow typical models where competitive exclusion holds. The question then is if dispersal is evolutionarily selected for (see Mouquet and Loreau (2003)).

1.5.2 Spatially explicit models without evolution

Regarding continuous space, models that treat space explicitly are usually based in the hypothesis that individuals move randomly following a random walk without a particular preference. The relation between this random walk and partial differential equations is made explicit by Skellam (1951). The main assumption is that individuals move in discrete time steps moving a fixed length from their position, which is chosen randomly around them. For a one-dimension walk, this means choosing randomly to make the step to the left or to the right; for two-dimension walks, this means choosing a direction in the circle at random.

The derivation presented by Skellam (1951) can be summarized as follows. Suppose we denote the probability of finding an individual in position x at time t by $n(t, x)$. Denoting the typical step length by ε and the time step by ω, then the dynamics of this probability are given by

$$\frac{\partial n}{\partial t} = \frac{1}{2}\frac{\varepsilon^2}{\omega}\frac{\partial^2 n}{\partial x^2}, \tag{1.1}$$

which means that for the scaling to make sense, it is necessary that ε^2/ω tends to a constant. Usually, ε is taken proportional to $\sqrt{\omega}$ so that $\varepsilon = \sigma\sqrt{\omega}$, where σ is called the diffusion coefficient in analogy with equations in statistical physics.

The variable n does not necessarily need ot be understood as the probability of finding an individual in space, it can also represent the proportion of a big population lying in a certain location at a particular time. While (1.1) models the dynamics of p for a population that only moves through space, general local effects $f(t, x)$ can be

introduced through the equation

$$\frac{\partial n}{\partial t} = \frac{1}{2}\sigma^2 \frac{\partial^2 n}{\partial x^2} + f(t, x), \tag{1.2}$$

which models the fact that individuals can experiment different processes at local scales: reproduction, death, migrations, interactions, environmental effects, etc.

Fisher (1937) presented a model for a population undergoing logistic growth, corresponding to local reproductions and population density limitation due to intraspecific competition, follows the dynamics given by the equation

$$\frac{\partial n}{\partial t} = \frac{1}{2}\sigma^2 \frac{\partial^2 n}{\partial x^2} + rn(1 - \alpha n). \tag{1.3}$$

This equation has two equilibria, given by the constant solutions $n = 0$ and $n = 1$, corresponding to the states where population is totally extincted or when it has reached a maximum density everywhere.

An interesting characteristic of this equation are the properties of its non-constant solutions, which are called traveling waves. The specific nature of the solutions depends on the initial condition, but when $n(0, x)$ is 1 for negative x and 0 for positive x, then the solution converges quickly to a function joining 1 and 0 traveling to the right at a constant speed, which we dub c_F. Moreover, $c_F = \sigma\sqrt{2r}$. Figure 1.2 shows an example of its dynamics.

This traveling wave nature of the solution was first attested by Fisher (1937) and Kolmogorov et al. (1937). Fisher's motivation was to study the propagation of an advantageous gene in a population, but his results can be reinterpreted in terms of the propagation of a population. Kolmogorov et al. (1937) showed that the same conclusion holds for a more general fitness function f satisfying certain hypothesis.

What is useful about Fisher's equation is that it predicts a constant invasion speed, which is something that is easily testable from field data sets. For example,

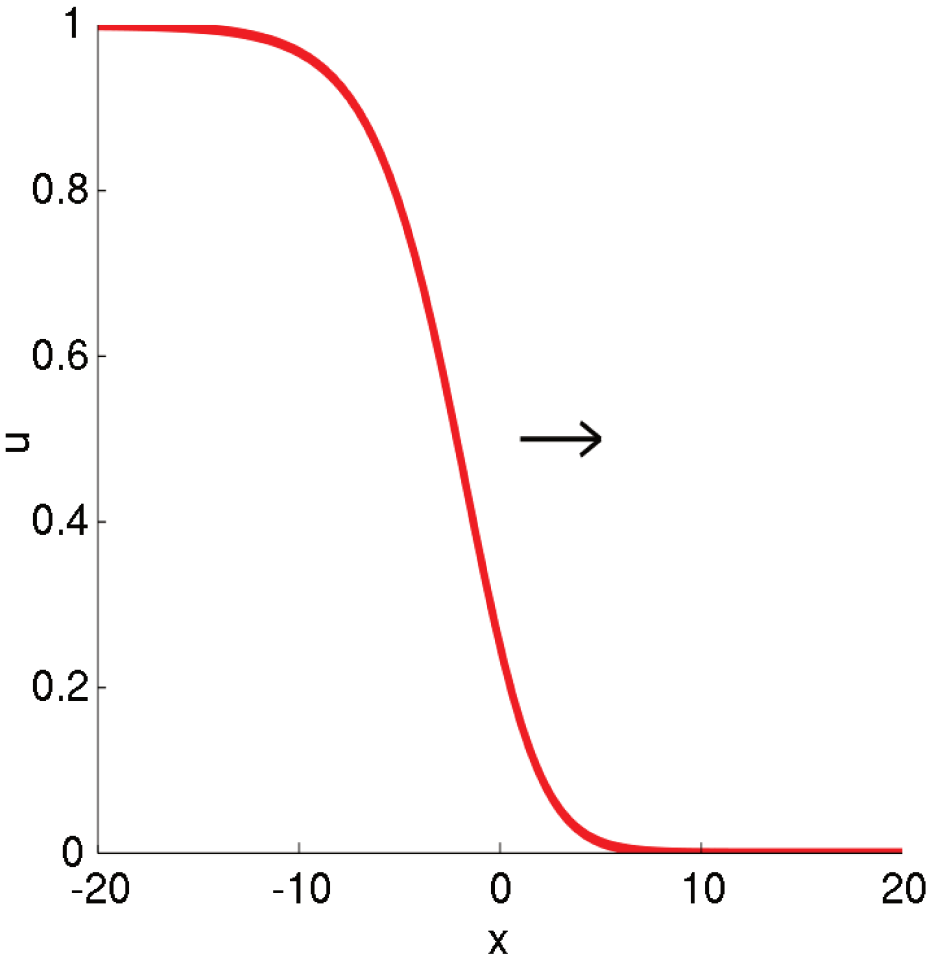

Figure 1.2: The solution of Fisher's equation (1.3) behaves like a front joining the equilibrium states $n = 0$ and $n = \alpha^{-1}$ at a constant speed $c_F = \sigma\sqrt{2r}$; here, $\alpha = 1$.

Bosch et al. (1992) estimate the values of growth rate and dispersal for several invasive species and compare the observed invasion rate to that predicted by Fisher's model. While the estimated values are near the observed velocities to an order of magnitude, the approximation is not very accurate.

This equation has several drawbacks, for example it does not consider heterogeneities in space (in the sense that the diffusion σ, the growth rate r and the carrying capacity K are constant and do not depend on space) and it only considers one type of population dynamics, namely the logistic growth. It is also a monospecific model, meaning that previously discussed effects of interactions are not taken into account.

The diffusion equation (1.2) can be extended in several ways to include interspecific interactions and/or evolution. For example, when two species under competition also experience diffusion due to random displacement in space their dynamics are given by

$$\frac{\partial n_1}{\partial t} = \frac{\sigma_1^2}{2}\frac{\partial^2 n_1}{\partial x^2} + r_1 n_1 \left(1 - \frac{n_1}{K_1}\right) - \alpha_{12} n_1 n_2, \tag{1.4a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\sigma_2^2}{2}\frac{\partial^2 n_2}{\partial x^2} + r_2 n_2 \left(1 - \frac{n_2}{K_2}\right) - \alpha_{12} n_2 n_1. \tag{1.4b}$$

This model can be understood as Lotka and Volterra's competition equations in a spatial setting. This equation can exhibit a variety of solutions depending on the value of the involved parameters and its initial conditions. It is interesting, however, that in some cases the dynamics can be understood through the behavior of the corresponding one species equation (1.3) (see e.g. (Volpert and Petrovskii 2009) for a review). Figure 1.3 shows an example of a solution.

We can similarly study a predator-prey system under diffusion. Its dynamics are

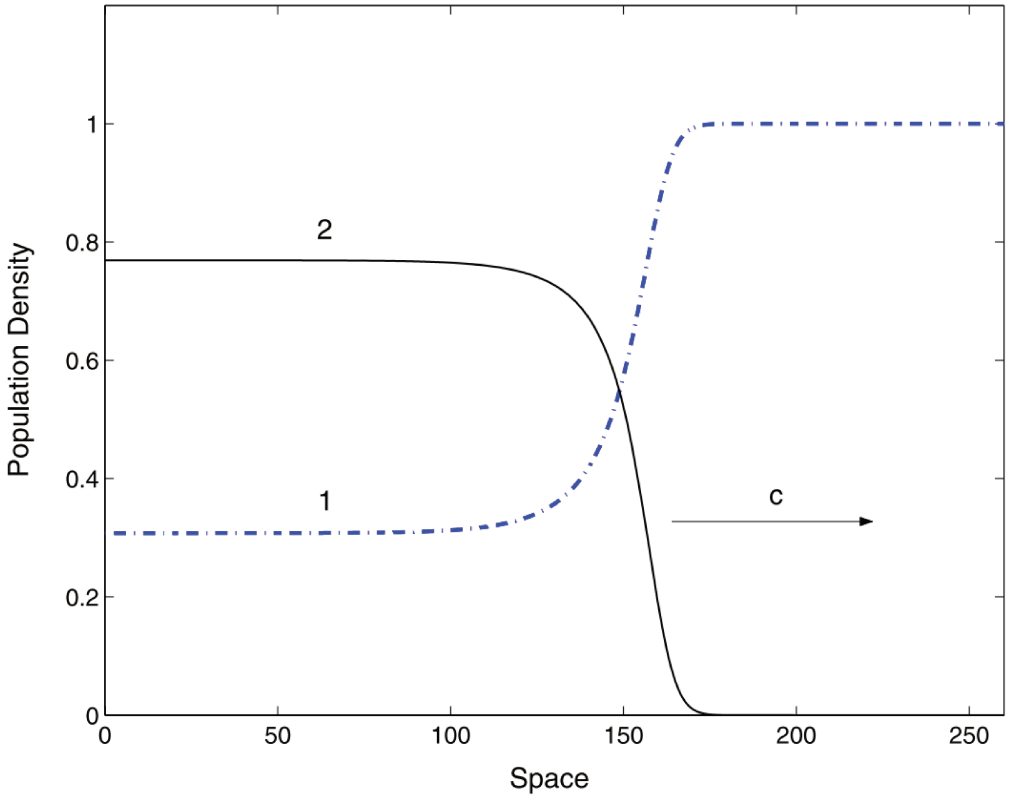

Figure 1.3: The solution of equation (1.4) behaves like a front joining two equilibrium states at a constant speed c. Here both species are allowed to coexist, thus $n_1 > 0$ and $n_2 > 0$ at the left (Image taken from Volpert and Petrovskii (2009)).

given by the equation system:

$$\frac{\partial n}{\partial t} = \frac{\sigma_1^2}{2}\frac{\partial^2 n}{\partial x^2} + r n_1 \left(1 - \frac{n}{K}\right) - \beta n p, \tag{1.5a}$$

$$\frac{\partial p}{\partial t} = \frac{\sigma_2^2}{2}\frac{\partial^2 p}{\partial x^2} + p\left(e\beta n_1 - \alpha p - d\right). \tag{1.5b}$$

Interestingly, the dynamics of this system can also be understood through the dynamics of the one-species system (1.3) and the non-spatial system (i.e., when both the diffusion parameters are null, $\sigma_1 = \sigma_2 = 0$ so that we may ignore the effects of space). Solutions behave like a prey and a predator traveling waves each one propagating at their own speed (S. Dunbar 1983, 1984; Zhang et al. 2016). Figure 1.4 shows an example of a solution.

There are several ways in which these model can be extended to account for different types of interactions modeling food webs with varying complexity. The literature on this topic is vast in ecology and in mathematics. However, all of these equations follow a similar structure: they model separately spatial effects (through diffusion) and local effects (here, the interaction between species). This first approximation is qualitatively good enough for our purposes.

1.5.3 Spatially explicit models with adaptation

As a mode of example Kirkpatrick and Barton's model (Kirkpatrick and Barton 1997) studies the dynamics of population density and mean trait value of a population structured by a phenotypic trait. This model supposes that there is an optimal trait value $\theta(x) = Bx$ such that when the phenotypic trait z of an individual differs from this optimal, there is a penalization in fitness. The dynamics of the model are given

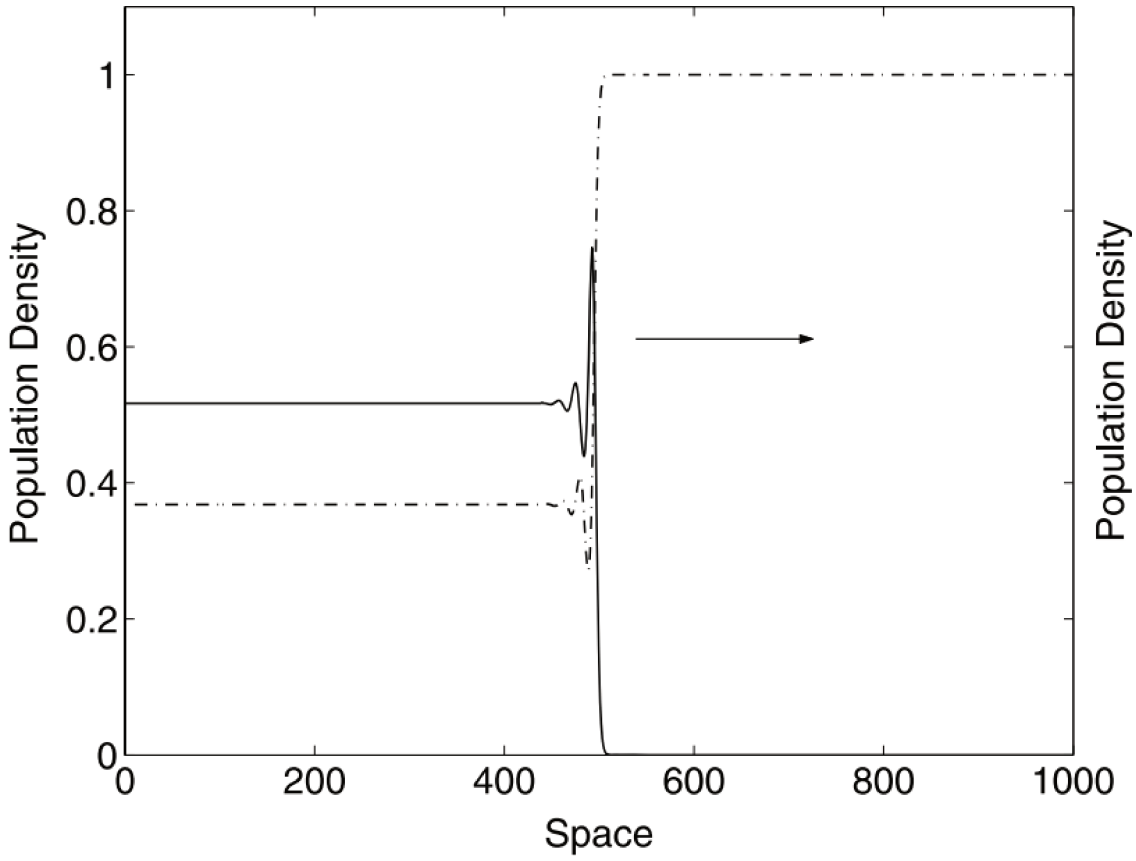

Figure 1.4: The solution of equation (1.5) behaves like a front joining two equilibrium states at a constant speed. Here predator population invades its prey range. The oscillations behind the predator's front depend on the value of the parameters (Image taken from Volpert and Petrovskii (2009)).

by

$$\frac{\partial n}{\partial t} = \frac{\partial^2 n}{\partial x^2} + n\left(1 - n - \frac{1}{2}\left(\bar{z} - Bx\right)^2\right), \tag{1.6a}$$

$$\frac{\partial \bar{z}}{\partial t} = \frac{\partial^2 \bar{z}}{\partial x^2} + 2\frac{\partial \log n}{\partial x}\frac{\partial \bar{z}}{\partial x} - A\left(\bar{z} - Bx\right). \tag{1.6b}$$

Here, the parameter A represents the adaptation potential of the considered population, and B is a measure of the degree of change of the phenotypical cline, so it is a measure of spatial heterogeneity.

Before moving on to the principal results of Kirkpatrick and Barton (1997), observe that equation (1.6b) corresponds to a spatial diffusion term, corrected by population density, plus a local selection term, which drives population towards the optimum trait value. This term is standard in quantitative genetics, and it is directly proportional to the derivative of the absolute fitness with respect to the phenotypic trait (Lande 1976, 1979):

$$\delta z = h^2 \frac{\partial r}{\partial z} = h^2 \frac{\partial r}{\partial z}\left(1 - n - \frac{1}{2}\left(\bar{z} - Bx\right)^2\right) = -2h^2\left(\bar{z} - Bx\right).$$

The main result found by Kirkpatrick and Barton (1997) is that solutions n can be restricted to a limited area, due to an interplay between evolution and dispersal. Figure 1.5 summarizes the possible behavior of solutions.

Kirkpatrick and Barton's model can be extended to account for other types of interactions, following the same logic as for the spatially explicit models without evolution presented in the previous section. In other words, multispecies extensions consider random dispersal in space and interspecific interactions and the effects of phenotypic traits are modeled through the local fitness term.

Case and Taper (2000) proposed a two-species model accounting for interspecific competition, following the lines of Kirkpatrick and Barton (1997). That is to say, they

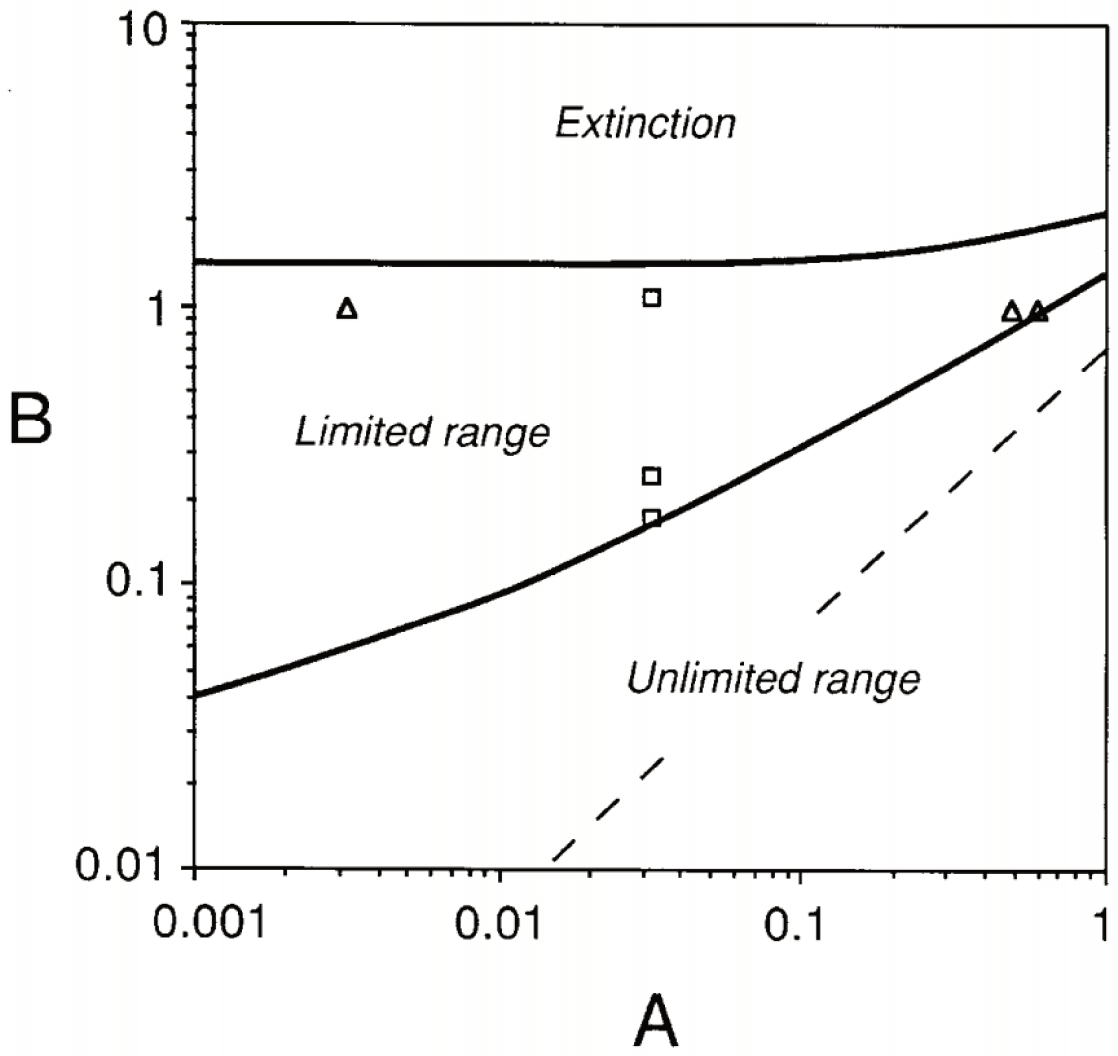

Figure 1.5: Solutions of Kirkpatrick and Barton's equations depend on the value of the adaptation potential A and the degree of spatial heterogeneity B. Populations may become extinct, persist in a limited range or persist in an unlimited space (Image taken from Kirkpatrick and Barton (1997)).

consider two competing species placed along an environmental cline corresponding to an optimal phenotypic value, and the species fitnesses are affected by the difference of their respective mean trait with respect to this cline. Their approach also considers that the more similar the species are, the more pronounced competition is. In this sense, the phenotypic trait can be also considered as a measure of similarity, and the competition strength depends on how similar the niche (sensu Macarthur and Levins (1967)) of both species is.

Two important observations about this model are in order. The first one is that it extends Kirkpatrick and Barton's model, in that when one of the species is removed from the system, the original equation system is recovered. The second one is that when one of the species could have had an unlimited range as predicted by the monospecific model (1.6), the second species can limit its geographic area. This is not merely due to competition, as Case and Taper show that at the range limit the phenotypic trait $\bar{z}_i$ grows away from its optimum. This limitation phenomenon is thus due to both competition and evolutionary factors.

Holt et al. (2011) proposed a different extension of Kirkpatrick and Barton's model, but accounting for predation. In their framework, prey are placed along an environmental cline, but predators are not subject to selective pressures. This is possible if predator adaptation is very fast compared to the prey's, so that fitness costs are negligible in the considered temporal scale. An interesting result of this study is that predators may have important effects on prey at an edge population by altering their gene flow. By predating on immigatring individuals, predation reduced maladapted gene flow from central populations, allowing a possibly limited-range population to expand its range. This example highlights the importance of considering both interactions and adaptation when studying geographical distributions, as this interaction that has negative effects on fitness is nevertheless positive in terms of space.

An interesting case is when the phenotypic trait z is correlated to the optimal temperature for the population's survival, so that $\theta(x)$ is, for example, mean temperature at a certain location. The main interest of this case is that it is suited to study the effects of climate change on species distributions.

For example, Pease et al. (1989) studied the dynamics of a one-species model similar to Kirkpatrick and Barton (1997)'s model but where population followed exponential growth and the environmental cline changed through time (and thus, also the optimal phenotype), modeling the effects of climate change. They found that the population followed the direction of the cline through time with a lag, corresponding to the fact that the spatial location where fitness is maximum shifts through time. They also found that the effective range of the population is larger the bigger dispersal is.

The article by Norberg et al. (2012) uses a similar equation system to study the effects of interspecific interactions and evolution on species distribution under a climate change scenario. They found that when neglecting interactions or when neglecting adaptation, species survival may be overestimated under such a scenario, highlighting the fact that both need to be considered simultaneously if we wish to have a better understanding of the determinants of species distributions. They also indicate that even long after climate change has stopped, some species can become extinct, a phenomenon they dubbed evolutionary debt.

To summarize, the principal message of this section is to show how versatile a PDE formalism can be to propose and describe the effects of different phenomena in nature. Diffusion does not necessarily always model a random movement of individuals, but it can account for grouping or degrouping effects, correlations in movement, escape from dangers, heterogeneity in space, etc. At the same time, the local dynamics can account for various effects such as population limiting, Allee effects, intraspecific competition, interspecific interactions, etc; it can even add selection on important

phenotypic characteristics as shown above.This is only a framework flexible enough to propose a description of a lot of different phenomena and plausible explanations. Utimately the choice of the model should rely on the level of accuracy that is wished for and on the temporal and spatial scales to be studied.

1.6 Organization and general idea of this thesis

This thesis is a logical proposal in the context presented above in that it is an effort towards modeling the spatial distribution of interacting species, accounting for important evolutive effects. Our purpose is to provide a framework flexible enough to study the problem of spatial distribution in an explicit manner, so as to be able to understand and predict these distributions and to provide mechanistic explanations.

Chapter II consists in a reanalysis of Kikpatrick and Barton's model, where we take an alternative approach to the one they considered originally. Kirkpatrick and Barton (1997) where focused in the conditions on the adaptation potential of a species and the degree of spatial heterogeneity that allowed or restricted a population to have limited or unlimited ranges or drive it to extinction. Here I followed an approach inspired in the dynamics of Fisher's model: when focused on a geographical range limit, the solution of the equation is a traveling wave, with a speed that depends on the value of adaptation potential and environmental heterogeneity. This provides a way to understand the propagation speed of an invading species as en eco-evolutionary indicator.

Chapter III moves on to consider a two-species predator-prey model, extending the model by Kirkpatrick and Barton (1997). Both species are subject to selection on an environmental cline, and so they propagate following their respective evolutionary invasion speed.

Finally, I apply a similar formalism to a community inspired by boom-and-bust dynamics observed in ecological invasions. Here, I consider a different mode of adap-

tation, allowing the native species to adapt to exotic predators, showing the versatility of the chosen approach. This constitutes Chapter IV.

Every chapter constitutes an article on which I worked during the duration of the thesis, and are collaborations with my advisors. The article in Chapter II has already been submitted for publication, the one in Chapter III awaits minor corrections before submission, and the one presented in Chapter IV is still a work in progress, but we have some satisfactory preliminary results.

For more clarity on the cited literature per article, every chapter has its own bibliography at the end. At the very end of the thesis I included a list joining the totality of the cited works.

The next section presents a summary of these works, before fully detailing them in each chapter.

1.7 Work Summary

1.7.1 Evolutionary invasion speeds and invasion mechanisms

Current global changes make it urgent to understand the eco-evolutionary determinants on species' ranges. A deep understanding of the way populations can help us derive efficient and useful conservation and control policies. This is also useful for understanding the impacts on biodiversity, as species modify their ranges and thus their interaction with other species.

Usual models used to describe distributions are only based on measurements of climatic variables, which do not provide a mechanism explaining them. These models do not consider adaptation to environment either, which has been observed to be an important determinant on geographic distributions, even at short timescales.

We take Kirkpatrick and Barton's model as a starting point, which is a monospecific model accounting for adaptation to environment and spatial heterogeneity. It

assumes that individuals are characterized by a phenotypical trait and that there is a cline for optimal values of this phenotype in space, so that deviation from the optimal implies a fitness penalty. If we denote the space by x, then the optimal cline is given by $\theta(x) = Bx$. Denoting the density of individuals by $n(t, x)$ and the mean trait of the considered population by $z(t, x)$, the dynaics are then given by the equation system:

$$\frac{\partial n}{\partial t} = \frac{\partial^2 n}{\partial x^2} + n\left(1 - n - \frac{1}{2}\left(\bar{z} - Bx\right)^2\right), \tag{1.7a}$$

$$\frac{\partial \bar{z}}{\partial t} = \frac{\partial^2 \bar{z}}{\partial x^2} + 2\frac{\partial \log n}{\partial x}\frac{\partial \bar{z}}{\partial x} - A\left(\bar{z} - Bx\right). \tag{1.7b}$$

This model depends on only two parameters, which are the degree of spatial heterogeneity, B, and the adaptation potential of the population, A.

The first equation assumes that population density follows a logistic growth, and that penalization for deviation from the optimum has a quadratic form. The second equation models the dynamics of the trait, and it accounts for random movement of individuals, asymmetric gene flow and local adaptation, driving the mean trait $\bar{z}$ towards the optimum Bx at a rate A.

It has already been shown that migration and adaptation potential A have antagonistic effects, depending on the degree of spatial heterogeneity B.

- When A is big compared to B, the population invades the whole spaces and persists in an unlimited range.

- When A has an intermediate value, the population can persist in a limited range area. This situation marks an equilibrium between local adaptation and maladapted gene flow in the boundary populations.

- If the adaptation potential is small compared to spatial heterogeneity, then the population becomes extinct.

We can reanalyze this equation system in terms of propagation speeds: when looking at a geographic front, it may advance or retract depending on the value of the parameters. We call $c_{KB}(A, B)$ the speed of advancement or retraction of this front.

We show that when the adaptation potential is big, $A \to \infty$, the equation system (1.7) becomes the Fisher-KPP equation:

$$\frac{\partial n}{\partial t} = \frac{\partial^2 n}{\partial x^2} + n\,(1 - n) \tag{1.8}$$

Its solutions behave like traveling waves with a minimum admissible speed $c_F = 2$.

On the other hand, we can derive a limit $A \to 0$ when $D = \frac{B}{\sqrt{2A}}$ is constant (as done in Mirrahimi and Raoul, 2013). In this limit, solutions have a speed that equals

$$c_D = \frac{\sqrt{A}}{20}\left(7\left(\frac{2}{D} - 9D\right) + 3\sqrt{\left(\frac{2}{D} - 9D\right)^2 + 40}\right). \tag{1.9}$$

We measured the traveling wave speed for the system (1.7) and we compared it to the speed of the two limit cases presented above. This provides a criterion to define what it means for a species to have high or low adaptation potentials, in terms of its traveling wave speed. When the traveling wave speed $c_{KB}(A, B)$ is near c_F then we can say that the population has a big adaptation potential, whereas $c_{KB}(A, B)$ near c_D means the population's adaptation potential is small. The results are summarized in figure 1.6.

We find that propagation speed is increasing as a function of A, and decreasing as a function of B. Moreover, if we exclude the extinction regime (i.e., the region above the red line in Figure 1.6), we see that a big part of the graph is well approximated by the formulas for the speed presented above. In other words, speed needs to be assessed numerically only for the zone outside the hatched areas, which we dub the

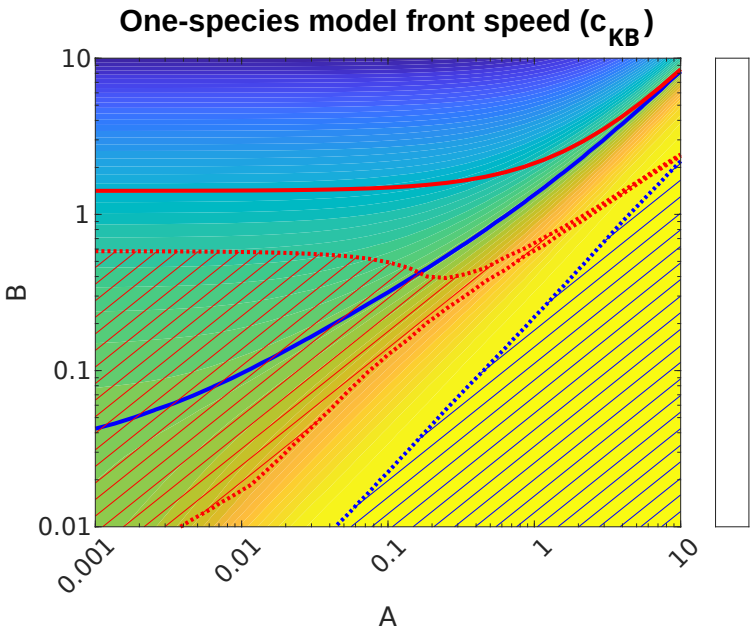

Figure 1.6: Approximated speeds for Kirkpatrick and Barton's one-species model along with several important lines showing some regime changes in the original system (1.7). The color gradient shows the speed value for different (A, B) parameters (see color legend on the right-hand side of the plot). The thick red line shows the regime change between extinction and limited ranges, as approximated by Kirkpatrick and Barton 1997. The thick blue line corresponds to the regime change between limited and unlimited range, which is also the zero-level line for the invasion speed (also in Kirkpatrick and Barton 1997, Figure 2). The blue hatched area is the zone in the parameter space where the difference between propagation speed in Kirkpatrick and Barton's model and Fisher-KPP's model is at most 0.1, marking the *strong adaptation* regime; the red hatched area is where propagation speed in Kirkpatrick and Barton's model is well approximated (i.e. the difference is at most 0.1) by the formula by Mirrahimi and Raoul 2013, i.e., given by (1.9), marking the *weak adaptation* regime.

medium adaptation zone.

1.7.2 Effects of predation on evolutionary invasion speeds and species distributions

We would also like to understand the way interspecific interactions modify the way species and populations establish themselves along space. Usual models used to describe distributions do not always consider interspecific interactions, and even if they do, they do not consider possible fast adaptations.

The model by Kirkpatrick and Barton (1997) is a good starting point for understanding one-species distributions along a linear gradient, but it ignores interspecific interactions. Although there have been studies considering competing species along an environmental gradient Case and Taper (2000) and Norberg et al. (2012), the predation case, for simultaneously adapting prey and predator, has not yet been studied.

There is evidence of interactions between community and evolutionary context in empirical studies, highlighting the importance of simultaneously considering interactions and adaptation.

Here, we propose a spatially explicit model accounting for predator-prey interactions and adaptation to environment along an optimal phenotypic cline.

We consider two interacting prey and predator populations distributed along one-dimensional space, parameterized by the variable $-\infty < x < \infty$, with dynamics taking place over time $t \geq 0$. We suppose that populations are structured by a phenotypic variable $-\infty < z < \infty$ and that, for each population, there is an optimal phenotypic trait value $\theta_1(x) = b_1 x$ (for prey) and $\theta_2(x) = b_2 x$ (for predators).

Denoting by $n_1(t, x)$ and $n_2(t, x)$ the local density of prey and predators, respectively, and by $\bar{z}_1(t, x)$ and $\bar{z}_2(t, x)$ their respective mean trait value, then the dynamics

of the system are given by:

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \frac{1}{2}\left(b_1 x - \bar{z}_1\right)^2 - \beta n_2\right) \tag{1.10a}$$

$$\frac{\partial n_2}{\partial t} = \delta\frac{\partial^2 n_2}{\partial x^2} + r n_2\left(n_1 - d - n_2 - \frac{1}{2}\left(b_2 x - \bar{z}_2\right)^2\right) \tag{1.10b}$$

$$\frac{\partial \bar{z}_1}{\partial t} = \frac{\partial^2 \bar{z}_1}{\partial x^2} + 2\frac{\partial \log n_1}{\partial x}\frac{\partial \bar{z}_1}{\partial x} - a_1\left(\bar{z}_1 - b_1 x\right) \tag{1.10c}$$

$$\frac{\partial \bar{z}_2}{\partial t} = \delta\frac{\partial^2 \bar{z}_2}{\partial x^2} + 2\delta\frac{\partial \log n_2}{\partial x}\frac{\partial \bar{z}_2}{\partial x} - r a_2\left(\bar{z}_2 - b_2 x\right). \tag{1.10d}$$

Like the one-species system, the first two equations model population density dynamics and they account for interspecific interactions, logistic growth, and a quadratic penalization for deviation from the optimum. The last two equations model the trait dynamics of prey ($\bar{z}_1$) and predators ($\bar{z}_2$), which take into account diffusion of individuals, asymmetric gene flow and local adaptation. The equations for predators have to be corrected for different diffusion rates (δ) and growth rate (r) relative to the prey's.

The equation also depends on predation rate (β), predator death rate (r) and different adaptation potentials of prey (a_1) and predators (a_2)

The equation has three equilibrium states, which are

$$E_0 : (n_1, n_2) = (0, 0), \quad E_1 : (n_1, n_2) = (1, 0), \quad E_2 : (n_1, n_2) = \left(\frac{1 + \beta d}{1 + \beta}, \frac{1 - d}{1 + \beta}\right), \tag{1.11}$$

corresponding, respectively, to: extinction of both populations, extinction of predators and coexistence of both populations.

The solutions of system (1.10) behave as traveling waves joining one or two of the three described equilibria at possibly different speeds, depending on the value of the parameters and initial conditions.

- A solution joining equilibria E_0 and E_1 corresponds to prey invading an environment where predators are absent. The speed of the traveling wave solution

associated to such a front is called the *intrinsic prey speed*, which we dub c_1^I. It can be approximated by $c_1 = c_{KB}(a_1, b_1)$.

- A solution joining equilibria E_1 and E_2 corresponds to predators invading an environment where their prey are present everywhere, and they are thus unrestricted. The front speed of such a solution is called the *intrinsic predator speed*, which we dub c_2^I.

- A solution joining equilibria E_0 and E_2 may do so directly or through an additional transition (passing through E_1). The speeds associated to the prey and predator fronts are called *prey joint speed*, c_1^J, and *predator joint speed*, c_2^J, respectively.

We found that the intrinsic predator speed can be approximated by the formula

$$c_2^I \approx \sqrt{\delta r\,(1-d)}\, c_{KB}\left(\frac{a_2}{\sqrt{1-d}}, \frac{b_2}{1-d}\sqrt{\frac{\delta}{r}}\right). \tag{1.12}$$

The accuracy of this approximation was attested numerically and the results are summarized in Figure 1.7.

We studied solutions initially joining E_0 and E_2 where the intrinsic speed of prey is positive, i.e., $c_1^I > 0$. We found that there are three important types of solutions, which can be understood in terms of the previously defined intrinsic and joint speeds:

I. *Local extinctions of predators*, which can result in total extinction or persistence in a limited area. These are characterized by negative traveling speeds $c_2^J < 0$ and total extinction and localized persistence cannot be distinguished based on speeds alone. We also found that $c_1^J = c_1^I$, i.e., the intrinsic and joint speed of prey coincide.

II. Predator invasion with a *growing gap* between predator and prey's front tips. Propagation speeds in this range satisfy the relation $0 < c_2^J < c_1^J$. We also found

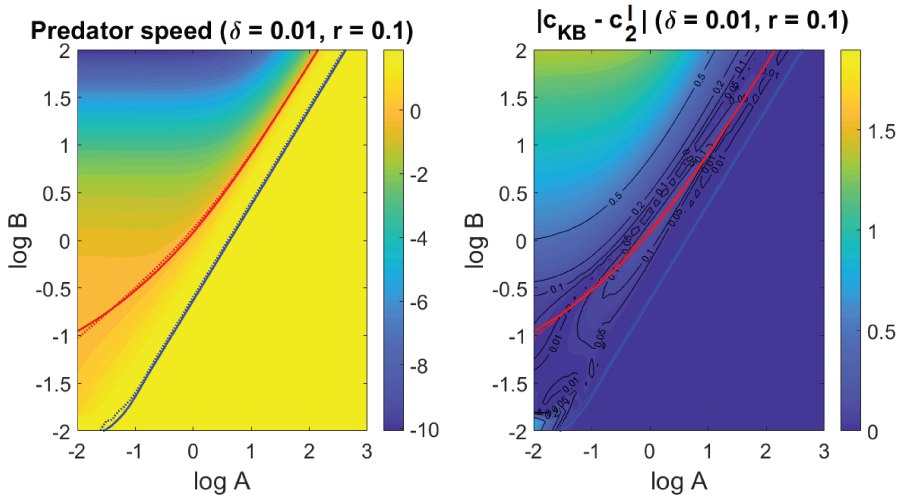

Figure 1.7: At the left, the independent predator speeds for a variety of parameters $A = \dfrac{a_2}{\sqrt{1-d}}$ and $B = \dfrac{b_2}{1-d}\sqrt{\dfrac{\delta}{r}}$. At the right, the absolute difference between the independent predator speeds and their approximation when using an equivalent one-species model. Blue color lines correspond to infinite adaptation limit, while the red ones correspond to the zero-speed limit.

that intrinsic speed coincide with the joint speeds.

III. *Simultaneous invasion* of both prey and predators. For parameters in this range, prey and predator invade the whole space at the same speed $c_1^J = c_2^J$. We also found that $c_1^J \leq c_1^I$.

We summarize our principal results in Figure 1.8, where we relate joint propagation speeds to propagation regimes.

When the relative predator diffusion δ is big, we also observed a spillover phenomenon at the prey's front tip, where predator's are able to invade space before their prey arrive. As a result, prey local density at the tip is decreased, causing its joint invasion speed to be lower than its intrinsic speed. We can thus summarize the principal results as follows:

- Whenever $c_2^I < c_1^I$ predator and prey edges do not meet, so that there is not interference at the front tips and each species advances at its own intrinsic speed.

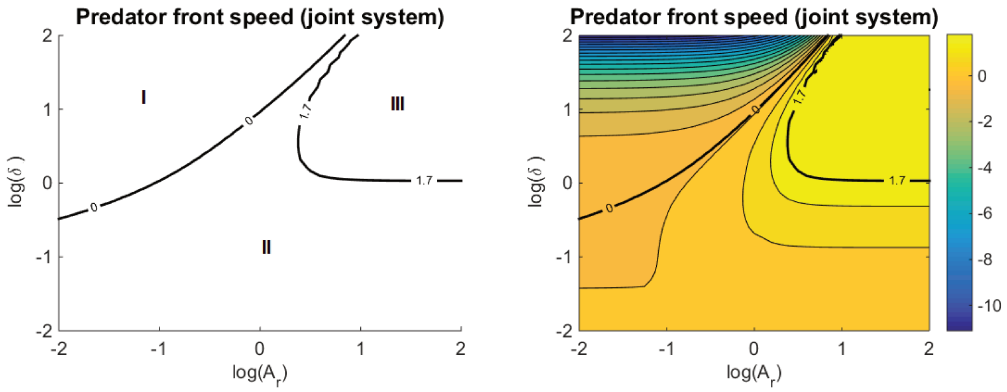

Figure 1.8: Phase graphic determining three different behaviors of the model depending on two parameters, relative adaptation potential $A_r = \frac{a_2}{a_1}$ and relative diffusion δ. The rest of the parameters, inclusing a_1, is left fixed. The zones correspond to local extinctions of the predators (zone I), local invasion of the predator population with a growing gap between the front tips (zone II) and simultaneous invasion of both populations (zone III). For all of the simulations, the intrinsic prey speed is $c_1^I = 1.7$.

If $c_2^I < 0$ predators go extinct (outcome I), otherwise propagation of both species will occur, with a growing gap (outcome II). In terms of geographic distribution, this second case leads to a decrease in the overlap of the two distributions in time.

- When $c_2^I > c_1^I$ we get a simultaneously invading predator-prey front (outcome III). Whether predators interfere with prey speed or not is impossible to tell apart based on intrinsic speeds alone. Prey speeds are decreased when diffusion causes a non-negligible density of predators to be present at the prey's front tip (predator spillover). When outcome III occurs, the geographic distribution of both species eventually perfectly overlap and this overlap is maintained in time.

1.7.3 Pathogen-aided invasions and adaptation to pathogens

Biological invasions are mainly a human-driven phenomenon that can permanently modify known ecosystems and their functioning. While several ecologic invasions have been observed, many hypothesis have been proposed to explain why invaders have

such a success in previously unknown environments Amsellem et al. 2017. Here we explore the hypothesis that invaders may be aided by their accompanying pathogens. There are two well-known examples of such invasions: the Asian ladybird Vilcinskas et al. 2013 and the red squirrel Darby et al. 2014.

Usual models used to study the dynamics of invasions take care of space in a simplified manner or do not take possible adaptation to foreigners into account. Here we propose a view that models space explicitly, and that considers the phenotype of the native species to be the degree of resistant to the foreign pathogen.

Thus, we will model one-dimensional space through the variable x, time as $t \geq 0$, and we will observe the native population density $n_1(t,x)$, foreign population or invaders density $n_2(t,x)$, pathogen density $p(t,x)$ and mean phenotype value $z(t,x)$. An individual with phenotype $z = 1$ in the native population is as resistant to the pathogen as is an individual from the foreign population, whereas an individual with $z = 0$ suffers an additional infection cost. There is also a cost in fitness to being resistant, so that an individual with trait $z > 0$ will suffer a decrease in fitness equal to κz^2.

When considering the effects of interspecific interactions (competition and predation) in a logistic growth model for all of the involved species, and using standard quantitative genetic laws for the phenotypic trait, the equations governing the system's dynamics are given by

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \alpha_{12} n_2 - \beta p\left(1 + \bar{B}(1-z)^2\right) - \kappa z^2\right) \tag{1.13a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\partial^2 n_2}{\partial x^2} + r n_2\left(1 - n_2 - \alpha_{21} n_1 - \beta' p\right) \tag{1.13b}$$

$$\frac{\partial p}{\partial t} = \delta\frac{\partial^2 p}{\partial x^2} + Rp\left(n_2 - p + \gamma\left(1 + \bar{B}(1-z)^2\right) n_1 - \bar{d}\right) \tag{1.13c}$$

$$\frac{\partial z}{\partial t} = \frac{\partial^2 z}{\partial x^2} + 2\frac{\partial \ln n_1}{\partial x}\frac{\partial z}{\partial x} - A\left(\beta\bar{B}p(z-1) + \kappa z\right). \tag{1.13d}$$

Here the different parameters account for: interspecific competition α_{12}, α_{21}; pre-

dation β, β' and additional relative predation $\bar{B}$; conversion efficiency γ, growth rates r, R; diffusion rate of the predators δ; adaptation potential A; and cost of making defenses κ.

Suppose that R is big enough that the pathogens reach quickly their equilibrium density, so that $p \approx n_2 + \gamma \left(1 + \bar{B}\,(1-z)^2\right) n_1 - d$ is a good approximation. Replacing this in the equations for the competing species gives

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1 \left(1 + \beta d - \kappa z^2 - \left(1 + \beta\gamma \left(1 + \bar{B}\,(1-z)^2\right)\right) n_1 - (\alpha_{12} + \beta) n_2\right) \tag{1.14a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\partial^2 n_2}{\partial x^2} + r n_2 \left(1 + \beta' d - (1+\beta')\, n_2 - \left(\alpha_{21} + \beta'\gamma \left(1 + \bar{B}\,(1-z)^2\right)\right) n_1\right) \tag{1.14b}$$

$$\frac{\partial z}{\partial t} = \frac{\partial^2 z}{\partial x^2} + 2\frac{\partial \ln n_1}{\partial x}\frac{\partial z}{\partial x} - A\left(\beta \bar{B} p (z-1) + \kappa z\right). \tag{1.14c}$$

Notice that the changes in pathogen density cause the densities of the competing species to change accordingly, modifying net growth rates and competition coefficients. This phenomenon is known as apparent competition, and in our particular scenario the intensity of competition changes as the native species becomes adapted, i.e., as z grows away from 0.

Since initially the native population has grown isolated from the introduced pathogens, it is a fair supposition that $z(0, x) = 0$. Moreover, since it has been established there for a longer time, they should be able to use local resources more efficiently. In other words, we expect competitive exclusion of the foreign species to take place when $p = 0$ and $z = 0$, which translates into a condition for the competition coefficients: $\alpha_{21} > 1$ and $\alpha_{12} < 1$.

We are modeling the scenario where the invading population is aided through their pathogens. This translates then as the foreign species being competitively exclusive when $z = 0$ and $p > 0$, which is a condition on the competition coefficients in

equations (1.14):

$$\frac{\alpha_{12}+\beta}{1+\beta d} > \frac{1+\beta'}{1+\beta' d}, \text{ and } \frac{1+\beta\gamma(1+\bar{B})}{1+\beta d} > \frac{\alpha_{21}+\beta'\gamma(1+\bar{B})}{1+\beta' d}. \tag{1.15}$$

Finally, we expect the native population to be competitively exclusive when perfectly resistant ($z = 1$). Again from (1.14) this translates as

$$\frac{\alpha_{12}+\beta}{1+\beta d-\kappa} < \frac{1+\beta'}{1+\beta' d}, \text{ and } \frac{1+\beta\gamma}{1+\beta d-\kappa} < \frac{\alpha_{21}+\beta'\gamma}{1+\beta' d}. \tag{1.16}$$

When both conditions (1.15) and (1.16) are met, solutions of system (1.14) behave as traveling waves with some particularities. As the predator front p advances at a constant speed decreasing the local density of native population n_1, the phenotypic trait $z = 0$ steadily increases to positive values. Behind the predator front follows the foreign population front n_2 which advances at a decreasing speed (due to the change of the value of z), until it becomes negative and this front retracts steadily.

We would like to approximate the time it takes the native population to stop the foreign invasion. Since this is caused by the change of resistance to predators, z, and that predator density between the two fronts (native and foreign population's fronts) is approximately constant, we expect this time to be well approximated by a non-spatial model where p is constant. This model is

$$\frac{dz}{dt} = -A\left(\beta\bar{B}p(z-1)+\kappa z\right).$$

If we denote by $z_\eta = (1-\eta)z_*$ the value of z at a relative distance η from equilibrium, the time until achieving this resistance level is given by

$$t_\eta = -\frac{\ln\eta}{A(\bar{B}\beta p+\kappa)}.$$

We measured the time until foreign population's front reversal in system (4.2) and we compared it to t_η in order to confirm this hypothesis. Preliminary results show that this does not seem to be a bad approximation, although they are still not very clean.

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CHAPTER II

Evolutionary invasion speeds and invasion mechanisms

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Abstract

Confronted with global changes and their potential impacts on biodiversity, an important question is to understand the ecological and evolutionary determinants of species geographic distributions. In order to understand how adaptation in heterogeneous environments constrains such distributions, we analyze how the potential of adaptation along an environmental cline affects the geographic distribution and propagation dynamics (invasion or extinction) of a single species. We re-analyse a model initially proposed by Kirkpatrick and Barton using propagation speed to assess whether species distribution is spatially limited or not.

We found that for big adaptation potentials, the species invades space following Fisher's model, whereas for small adaptation potentials the propagation depends on the evolutionary challenge to overcome. We have explicit approximations for the propagation speeds in both cases. We discuss the utility of these propagation speeds as an eco-evolutionary index based on empirical studies.

Key words: local adaptation; partial differential equation model; quantitative genetics; linear gradient.

2.1 Introduction

In response to current climate changes, many species have been observed to shift their geographic distribution (Parmesan and Yohe 2003). Such changes in the spatial distribution of species may largely alter their co-occurence, thereby affecting the structure of ecological networks (Tylianakis et al. 2008), the functioning of ecosystems and the services they provide. To better understand such consequences, we urgently need to predict how species establish themselves along environmental gradients, but also to understand the mechanisms determining species distributions, so that we can forecast their future changes and thus adapt conservation policies.

To tackle this question, the most common approach relies on the development of niche-based species distribution models (SDMs), which provide predictions of species distributions based on presence/absence data and their association with a given set of environmental variables (refer to Guisan and Zimmermann 2000 for an introduction to SDMs or to Guisan and Thuiller 2005 for a more recent review; see also Thuiller et al. 2003 for a comparison of the performance of some SDMs). SDMs usually assume niche conservatism and range equilibrium, thus failing to include local adaptation. On some occasions, niche models alone may fail to describe the observed distribution of a species, especially in out-of-equilibrium cases such as species invasions. For instance, Broennimann et al. 2007 document a case study in which an invasive species has a different niche in its invasion range, although in this case data does not allow to determine if differences are adaptive (due to a shift in fundamental niche) or ecological (due to another possible realized niche taking place). Understanding such aspects would require the development of models which would simultaneously consider the niche model and a mechanistic approach of eco-evolutionary dynamics (e.g., Bush

et al. 2016).

Although adaptation to local conditions should help a population expand its range, boundary populations may be constrained in their adaptation due to the negative effect of gene flows from more central populations, i.e. genetic swamping. For example, Sanford et al. 2006 and Dawson et al. 2010 observed high migration load in boundary populations of a fiddler crab (*Uca pugnax*) and a volcano barnacle species (*Tetraclita rubescens*), respectively, while showing that individuals from the range limit are able to produce offsprings that would survive past the limit. Adaptation may take place on relatively short timescales: Balanya 2006 has shown that *Drosophila subobscura* at the leading edge of an ongoing invasion are able to adapt to local conditions while establishing a cline of genetic characteristics linked to temperature adaptation, following climatic gradients. Rapid adaptation and genetic swamping are quite general phenomena not restricted to species with short generation time. High gene flow has for instance been suggested to occur in many tree species Kremer et al. 2012, with potentially important effects on genetic variance at edge populations. Such evolutionary constraints may play a critical role in the persistence of tree species and in the variations of their geographic distributions, affecting the future of forest ecosystems under climate change scenarios. These studies underline the crucial need of including local adaptation when studying species distributions, and even more so when the aim is to understand and forecast future distributions under global change.

One monospecific spatially structured model accounting for both local adaptation and migration was presented by Kirkpatrick and Barton 1997. This model explains limited range distribution as an equilibrium between migration and genetic load from maladapted populations. Since there are no known explicit solutions, this model needs to be applied through numerical simulations.

In the present work, we take another look at the model by Kirkpatrick and Barton 1997 to study how adaptation alters the propagation and distribution of a single

species in a linearly varying environment. Our goal is to better understand the propagation dynamics according to adaptation potential for a single species and to derive useful approximations for limit-adaptation cases. We address the question of how adaptation potential affects the geographic dynamics of a single species by providing approximations of species propagation speed under extreme scenarios (very low or very high adaptation potential) and using extensive simulations to understand intermediate scenarios. We link the local adaption and limits to range size to the variation in propagation speeds.

2.2 Kirkpatrick and Barton's model for a single species' range evolution along a linear gradient

The one-species model proposed by Kirkpatrick and Barton 1997 is a spatially explicit model in heterogeneous space accounting simultaneously for migration effects and adaptation. It assumes individuals are characterized by a phenotypic trait and that heterogeneity in space is given by a continuous cline of the optimal value for this phenotype. Individuals whose phenotype deviates from this optimum will suffer a fitness penalty. Although this model assumes that the environmental cline remains fixed in time, it provides a framework to study, for example, the effects of climate change on species distributions (see e.g. Norberg et al. 2012), as it can easily be modified to include a time-varying environment. It is also suitable to study invasion scenarios, linking the characteristics of the environment and those of the introduced population.

The model assumes infinite one-dimensional linear space and considers local population density $n(t, x)$, i.e. the density of individuals at location x at time $t \geq 0$, and the mean phenotypic value of this population, $\bar{z}(t, x)$, at this time and this location. The environmental cline is modeled through the optimal phenotype function

$\theta(x) = Bx$ meaning the optimal value varies linearly through space. After a renormalization of the original variables and parameters in the full system (see Kirkpatrick and Barton 1997 for details), the equations governing density and phenotype dynamics are given by

$$\frac{\partial n}{\partial t} = \frac{\partial^2 n}{\partial x^2} + n\left(1 - n - \frac{1}{2}\left(\bar{z} - Bx\right)^2\right), \tag{2.1a}$$

$$\frac{\partial \bar{z}}{\partial t} = \frac{\partial^2 \bar{z}}{\partial x^2} + 2\frac{\partial \log n}{\partial x}\frac{\partial \bar{z}}{\partial x} - A\left(\bar{z} - Bx\right), \tag{2.1b}$$

where A is a measure of *adaptation potential* of the species (A is proportional to genetic variance) and B is the rate of change of the optimal phenotype through space, also considered to be a measure of *spatial heterogeneity*.

System (2.1) describes the eco-evolutionary dynamics of the species under local adaptation and spatial diffusion. The first term of Equation (2.1a) models the dispersal of the population through a diffusion process. The second term contains the local ecological dynamics, corresponding to the logistic model and a penalizing term that captures local maladaptation. The first term of equation (2.1b) models the diffusion of genes that is linked to the diffusion of individuals, while the second term corrects for asymetries in gene flows (gene flow being more important from large populations to small populations than the other way round). The third term corresponds to the effects of local adaptation due to directional selection, driving the mean phenotype value $\bar{z}$ toward the local optimum Bx at a rate A.

Migration and adaptation potential A have antagonistic effects, whose results vary depending on the spatial heterogeneity B. Depending on A and B, the population may survive in a limited space (for intermediate values of A and B), may invade the whole space (when adaptation is larger than a certain critical value, allowing the population to surmount spatial heterogeneity) or may become extinct (when adaptation is too small with respect to spatial heterogeneity; (Kirkpatrick and Barton

1997)). This result can be partially re-stated in terms of propagation speeds (Fisher 1937), which answer at the same time the question of geographic dynamics of the population: if we consider as initial condition a geographic frontier, i.e., the initial condition is $n(0,x) = 1$ for $x \leq 0$ and 0 otherwise, with the species being perfectly locally adapted ($\bar{z}(0,x) = Bx$) wherever it is present ($n(0,x) = 1$), then the solutions behave like propagating fronts with a characteristic speed. For Kirkpatrick and Barton's one-species model, the direction and magnitude of the advancing front depend on the parameters A and B. Positive speeds mean the front moves towards positive values of x so that the species progressively invade (hereafter *invasion fronts*). On the contrary, negative values mean that the species distribution retracts (either to a limited range or toward the extinction of the species, hereafter *extinction fronts*). We dub $c_{KB}(A,B)$ the speed of the solution of system (2.1) for parameters A and B.

In terms of propagation speeds, species whose borders correspond to invasion fronts are able to continuously adapt to new environments and thus will always be able to invade the whole space. On the contrary, negative speed fronts only mean maladapted gene flow is stronger than adaptation, causing local extinctions that can lead to two outcomes: either the population becomes extinct, or two fronts from different directions collide canceling out maladaptations in the center and allowing the species to survive in a limited space. We cannot distinguish between these two last outcomes based on speed alone, another demographic criterion is needed to do so. Refer to Figure 2.1 for a clearer link between propagation speeds and spatial distribution.

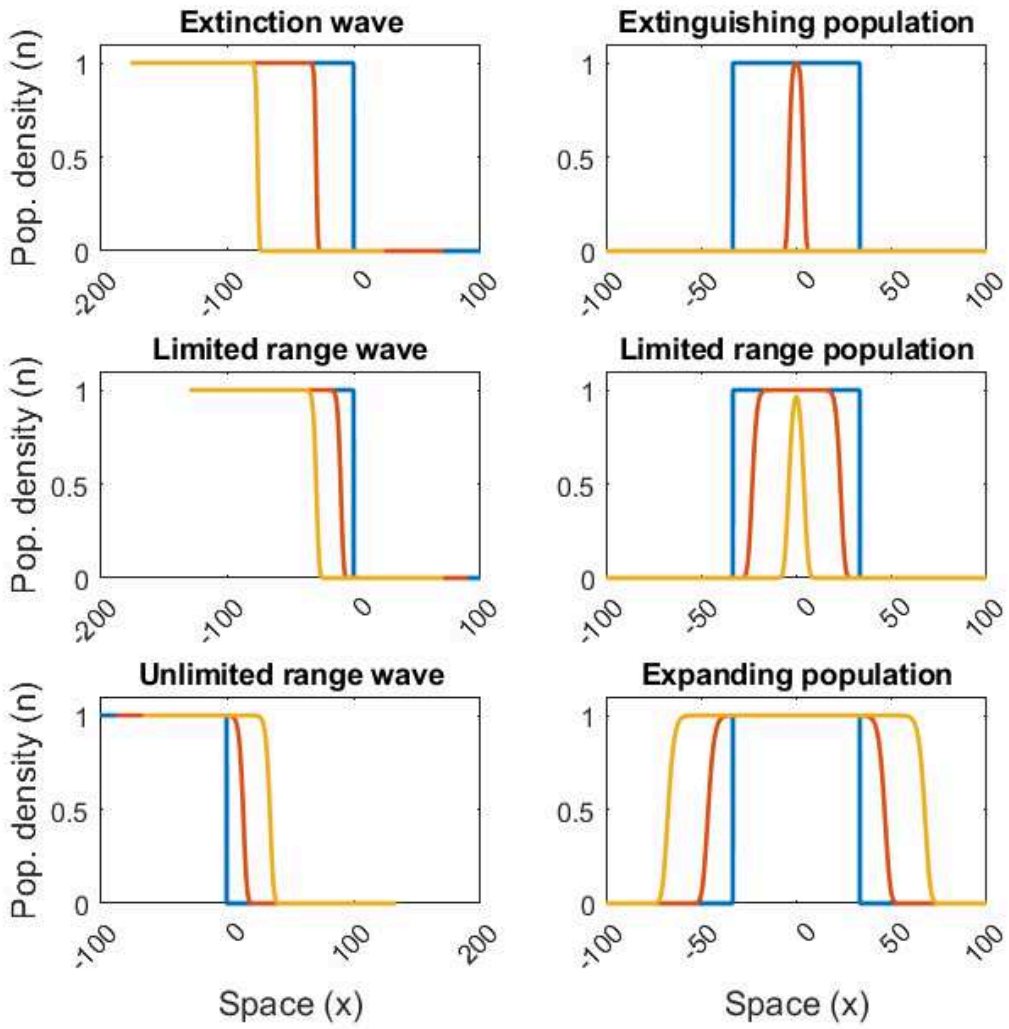

Figure 2.1: Panels showing the relation between a propagation wave and the respective population density distribution. In every panel, color blue indicates the initial condition, color red indicates an intermediate value ($t = 20$) and color yellow a long time ($t = 50$) distribution. The panels on the left column feature the dynamics of a boundary, whereas the panels on the right column feature the dynamic of an initially limited-range population distribution, with the same parameters (A, B) for each row. The first two rows show that a negative propagation speed may drive a population towards extinction (first row) or to a limited range distribution (second row). The third row shows that a positive propagation speed leads to an unlimited range distribution.

2.3 Explicit approximation of propagation speeds under various adaptation scenarios

We investigate the variation of propagation speeds for different values of parameters A and B, with a focus on the two limit cases of infinitely strong adaptation and very weak adaptation potentials. Even though it is unlikely species adapt infinitely fast, the variation in propagation speeds between these two limit cases can tell us when a finite adaptation is strong enough so that it is qualitatively infinite.

One first important result is that when adaptation goes to infinity, $A \to \infty$, the system (2.1) becomes the Fisher-KPP equation (after Fisher 1937 and Kolmogorov et al. 1937, see the Appendix 2.6.1 for details), given by

$$\frac{\partial n}{\partial t} = \frac{\partial^2 n}{\partial x^2} + n\,(1 - n) \tag{2.2}$$

in its non-dimensional form (refer to the appendix for details on this infinite adaptation limit). Its solutions are traveling fronts with a minimal admissible speed of $c_F = 2$ (or, in its dimensional form, $c_F^* = 2\sqrt{r\delta}$ with r corresponding to the intrinsic growth rate of the population and δ a measure of its dispersal), so that for infinite adaptation potential invasion speed is finite and constant. Equation (2.2) has an infinity of solutions for different front speeds $c \geq c_F$, but c_F is the smallest one and the only one with biological meaning.

We can draw two other important conclusions thanks to equation (2.2). First, in an ecological context, the Fisher-KPP equation can only model propagation of species whose adaptation is so fast that they are continuously well-adapted everywhere, since the equation is the same as the system (2.1) neglecting maladaptation (and all the terms involving the phenotypic trait). Second, invasion speeds for the one-species model given by system (2.1) will always be lower than $c_F = 2$, since growth rate in the Fisher-KPP model is always larger than the one of the KB model, because

maladaptation effects can only decrease population fitness (having thus a negative effect on speed). This means that the maximum speed of range expansion is only constrained by the species growth rate and dispersal ability (since it is $c_F^* = 2\sqrt{r\delta}$ in its dimensional form).

At the other extreme of the adaptation gradient, the limit of small adaptations $A \to 0$ needs to be studied more carefully. For $A = 0$, we would have a non-adapting species which cannot invade environments it is not suited to. We consider the term $D = \frac{B}{\sqrt{2A}}$ which we dub the *evolutionary challenge*, since it embodies the spatial heterogeneity to overcome for a given adaptation potential (measured not directly as A, but as $\sqrt{2A}$). We consider species with decreasing adaptation potentials while keeping a constant evolutionary challenge (i.e. $A \to 0$ with D constant). This provides a way to study a small adaptation potential while scaling the environment accordingly. This small adaptation limit has already been studied by Mirrahimi and Raoul 2013 and there is an explicit expression for the propagation speed for such low adaptation scenarios, given by:

$$c_D = \frac{\sqrt{A}}{20}\left(7\left(\frac{2}{D} - 9D\right) + 3\sqrt{\left(\frac{2}{D} - 9D\right)^2 + 40}\right), \tag{2.3}$$

or $c_D^* = c_D\sqrt{r\delta}$ in its dimensional form.

Note that this expression is decreasing in D, meaning that for larger evolutionary challenges the invasion speed will be smaller (refer to Figure 2.2). Also, invasion speed depends on the spatial heterogeneity (i.e., B) only through D. The value $D_{crit} = \frac{2}{3}$ gives an invasion speed of 0, which means that for very small adaptation potentials, when the challenge is larger than D_{crit} the species goes extinct, while when the challenge is smaller they are able to invade at a speed c_D^*. Notice how c_D^* is proportional to $\sqrt{r\delta}$, as is Fisher-KPP's speed, corrected by a factor c_D that depends on the adaptation potential $\sqrt{A}$ and challenge D, which corresponds to a

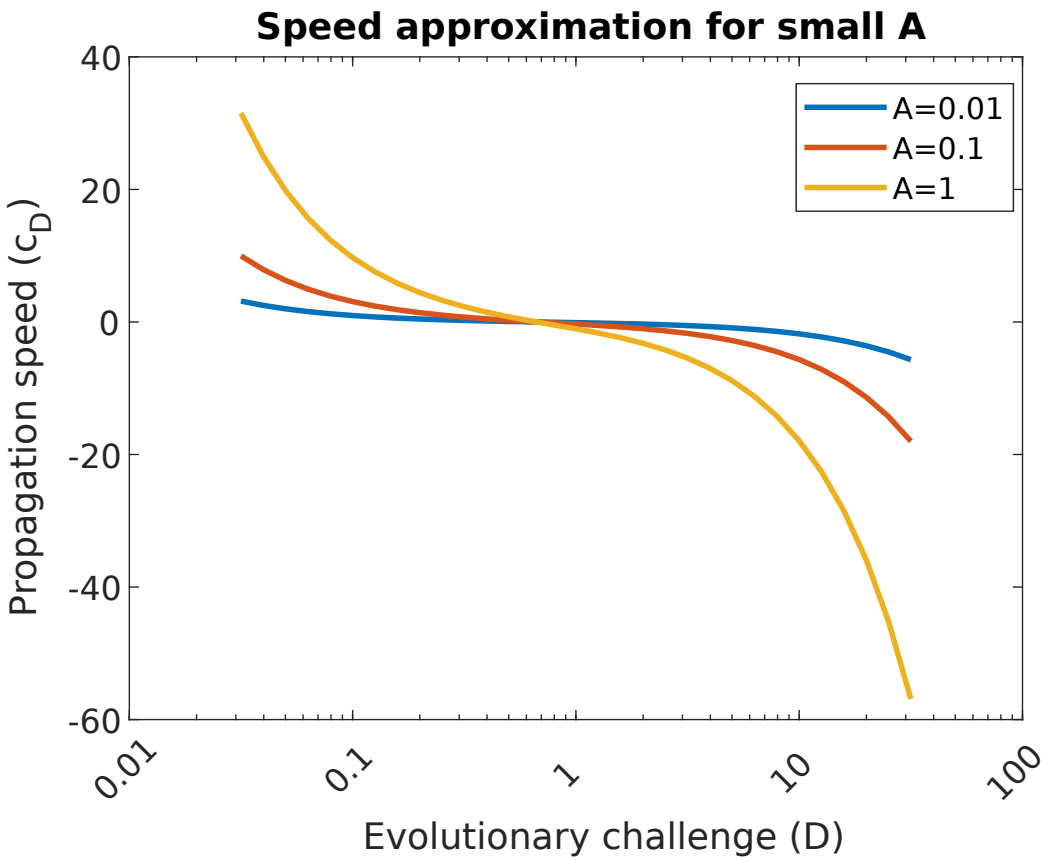

Figure 2.2: Propagation speed c_D as a function of the environmental challenge D, as defined by formula (2.3).

loss in invasion efficiency due to maladaptation effects.

2.4 Relating propagation speeds to adaptation regimes

A natural question is then how these extreme scenarios relate to Kirkpatrick and Barton's one-species model in terms of propagation speeds, which provides a method to concretely determine what strong and weak adaptations mean. To understand this, we numerically approximate the solution of the one-species model for a variety of parameter pairs (A and B) and compare the results to those given by the extreme adaptation limits (Figure 2.3. Refer to the Appendix 2.6.2 for details on the numerical scheme).

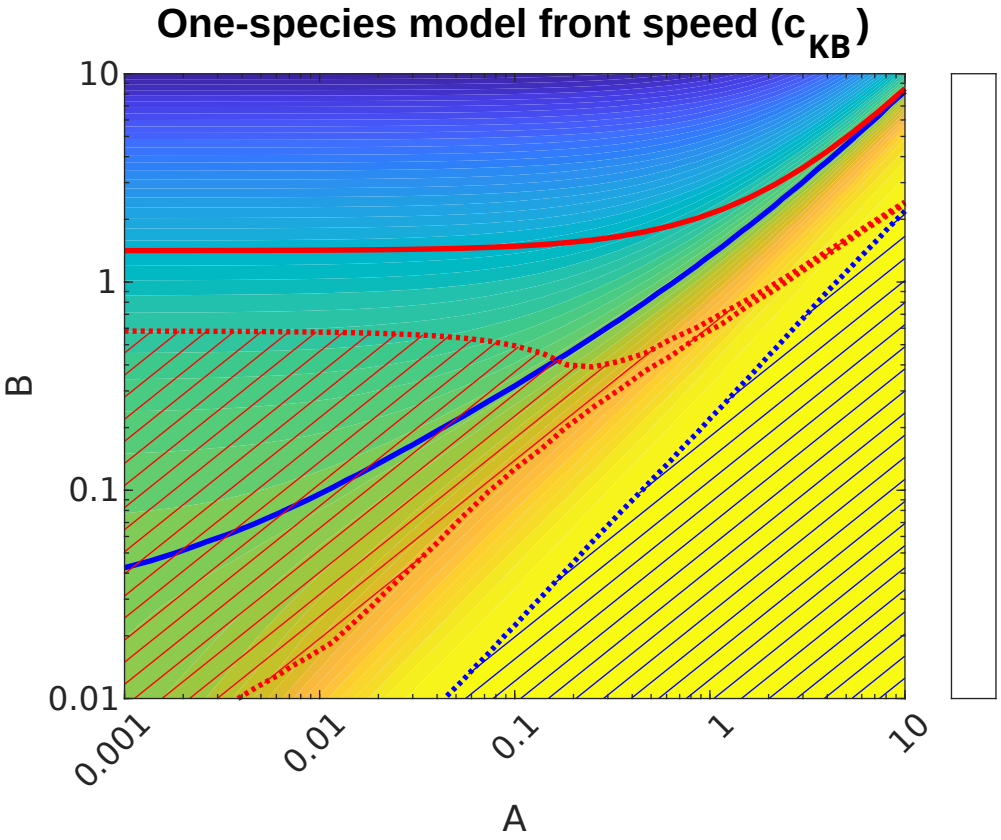

Figure 2.3: Approximated speeds for Kirkpatrick and Barton's one-species model along with several important lines showing some regime changes in the original system (2.1). The color gradient shows the speed value for different (A, B) parameters (see color legend on the right-hand side of the plot). The thick red line shows the regime change between extinction and limited ranges, as approximated by Kirkpatrick and Barton 1997. The thick blue line corresponds to the regime change between limited and unlimited range, which is also the zero-level line for the invasion speed (also in Kirkpatrick and Barton 1997, Figure 2). The blue hatched area is the zone in the parameter space where the difference between propagation speed in Kirkpatrick and Barton's model and Fisher-KPP's model is at most 0.1, marking the *strong adaptation* regime; the red hatched area is where propagation speed in Kirkpatrick and Barton's model is well approximated (i.e. the difference is at most 0.1) by the formula by Mirrahimi and Raoul 2013, i.e., given by (2.3), marking the *weak adaptation* regime.

First, note that propagation speed is increasing as a function of A and decreasing as a function of B, which is intuitive since larger adaptation potential and smaller spatial heterogeneity imply species will invade more easily.

The blue hatched area in Figure 2.3 shows where the difference between the one-species model speed c_{KB} and Fisher-KPP's model speed c_F is at most 0.1. In this sense we can say that the blue dotted line marks *the limit between strong and intermediate adaptation potential*. A simple linear regression lets us approximate this region analytically by the inequality $A \geq 10^{0.65}B$. In other words, whenever adaptation potential surpasses the critical value $A_{\text{crit}} = 10^{0.65}B$ maladaptation effects are negligible, and species invade at a maximal speed, well approximated by the Fisher-KPP model.

The red hatched area in figure 2.3 marks where the speed in the one-species model is close to the small adaptation limit speed given by (2.3) (i.e. the difference between the propagation speed and the speed given by this formula is at most 0.1), so that in this zone adaptation is weak; thus the red dotted lines establish *the limit between weak and intermediate adaptation potential*.

The thick blue line corresponds to the zero-speed line, marking the division between positive and negative speeds. In other words, this line corresponds to the limit between unlimited and limited range which was studied in Kirkpatrick and Barton 1997.

Interestingly, this leaves only a small zone of parameter space that cannot be explicitly approximated. Propagation speeds then need to be assessed numerically since we do not have explicit formulae for them. This parameter space corresponds to the area outside the extinction regime (below the thick red line) that is not hatched, which we dub the *intermediate adaptation potential* zone. We also decided not to consider the speeds in the extinction zone since behavior in this zone is not very interesting, although we were able to measure corresponding extinction speeds.

We can find an explicit approximation for this line (by fitting a two-degree polynomial on the level-line, using MATLAB's methods) which is given by:

$$\log_{10} B_{\text{crit}} = 0.085(\log_{10} A)^2 + 0.707 \log_{10} A + 0.125. \tag{2.4}$$

This means that for a given level of adaptation A, the population can only invade the whole space if the heterogeneity is smaller than B_{crit}, otherwise it will suffer local extinctions, being restricted to a limited range or disappearing altogether. Solving the equation for A, we can interpret this result the other way round: for a given level of spatial heterogeneity B, the population will be able to invade only if its adaptation level is greater than the solution A^*. Unfortunately, the method we used does not provide us with an explanation or an intuition as to why the regime change occurs on this line, but it is nevertheless an improvement of the condition found by Kirkpatrick and Barton 1997, $B_{\text{crit}} = \sqrt{2}A$, or equivalently,

$$\log_{10} B_{\text{crit}} = \log_{10} A + 0.150515. \tag{2.5}$$

2.5 Discussion

Our model highlights how species adaptation can affect species extinctions and their geographic distributions. In this single-species model, explicit propagation speed and conditions of extinction can be obtained for most of the parameter space. These approximations highlight how different mechanisms act when considering low- vs high-adaptation potential.

The single-species adaptation model by Kirkpatrick and Barton 1997 shows various interesting behaviors. Even though there are no known explicit solutions to this system of equations, we were able to relate this model to other works, thereby providing explicit propagation speeds for most of the parameter space (refer to Figure 2.3

and its legend for details). Although the purpose of Kirkpatrick and Barton's one-species model was not to study invasion processes, the usual approach to understand similar models is through the analysis of the speed of propagating fronts (as done in the first articles Fisher 1937 and Kolmogorov et al. 1937 or in literature in general, Skellam 1991, Shigesada and Kawasaki 1997). This corresponds to the speed of ongoing local invasions (positive speed fronts) or local extinctions (negative speed fronts), which is the approach we took here. The original study by Kirkpatrick and Barton 1997 focused on the antagonistic effects of gene flow in a heterogeneous environment to understand the conditions under which a population has a finite geographic range. In order to comment on their results, it is helpful to recall the definitions of their compound parameters: the adaptation potential $A = G/(2V_s r^*)$ is the additive genetic variance of the population G divided by the basic population growth rate r^* and the strength of stabilizing selection (a smaller value of V_s meaning stronger selection); and the spatial heterogeneity $B = b\sigma/(r^*\sqrt{2V_s})$ is proportional to the environmental gradient b and the dispersal rate σ (which makes sense since the more an individual disperses, the more the environment will be different proportionally to b). Although the approach developed by Kirkpatrick and Barton 1997 provided an efficient way to understand the determinants of range boundaries, it only deals with a small part of the parameter space (refer to Figure 2.3). Our results indicate that interesting conclusions, mostly about adaptation potential, can be obtained by looking at propagation speeds in the whole parameter range for A and B. Besides, the speeds of advancement or retraction of these models is interesting because it gives a way to roughly predict the future repartition of the modeled species (i.e. extinction/retraction or expansion of its range).

Our analysis revealed that adaptation potential, measured through parameter A, has a strictly positive effect on invasion speed. This speed is always smaller than that of Fisher's model (Fisher 1937), which neglects spatial heterogeneity. Thus,

adaptation potential not only dictates whether a species can establish itself over space in a limited or unlimited manner, but it also helps overcome spatial heterogeneity, as shown by its invasion speed. For very strong adaptation potentials, the effects of spatial heterogeneity become negligible, with invasion speed being nearly equal to that of Fisher's model. This is what we called the "strong adaptation zone" in Figure 2.3. For small adaptation potentials, the explicit approximation suggests that invasion speed critically depends on the evolutionary challenge $D = B/\sqrt{2A}$ (equation (2.3)). This means that the fate of species with small adaptation potential depends not so much on their adaptation capabilities, but rather on the spatial gradient to overcome given their evolutionary potential. The more challenging an environment is, the slower the invasion speed will be. Some case studies suggest that such constraints do act in nature. Consider for instance the reinvasion of its historic range by the California sea otter (*Enhydra lutris*) and the invasion of the sugar cane toad (*Rhinella marina*) in Australia. In the first case, Lubina and Levin 1988 showed important differences in expansion speeds at the north and south limits of the otters' ranges possibly due to important environmental differences. In the second case, Urban et al. 2008 show that the invasion speed for the cane toad is not constant in time, and that periods of acceleration or decrease may be linked to changes in the environmental clines being invaded. In both cases, it is safe to suppose that the adaptation potential of the species is small, since both invasions started with only a few individuals.

Excluding the zone in Figure 2.3 where the species becomes extinct, the two approximations we used provide good descriptions of speeds in most of the parameter space. Having covered the infinite- and small-adaptation limits, only speeds in intermediate regimes remain to be studied. The explicit expressions for speeds we found make our model highly applicable. These explicit propagation speeds are also interesting because they let us determine where the limit between limited and unlimited range occurs. This limit matches the 0-speed isocline, corresponding to the zones

where either increasing spatial heterogeneity or decreasing adaptation potential lead to species extinction. Limited ranges occur when two extinction fronts from opposite directions meet and maladaptation manages to cancel out in the middle, which is possible before population decreases critically if selection is not too strong. This is another motivation to study the intermediate-adaptation regimes more in depth.

We can draw two other important conclusions from this analysis: knowing a species adaptation potential A and the rate of change of its optimal phenotype over space, B, we can determine whether the species is going to invade space or not and at what speed. As a corollary, knowing the speed of advancement of a species and estimating the degree of spatial heterogeneity B can give an indirect assessment of the species adaptation potential A, which is directly related to its genetic variance. In other words, invasion speed can be used as an eco-evolutionary index allowing us to draw conclusions on genetic characteristics of a population. For instance, the previously cited cases of the sea otter and the cane toad (Lubina and Levin 1988 and Urban et al. 2008, respectively) are ideal cases to which our framework could be applied, for example to determine whether adaptation potential is the same along the different environments, which would explain the difference in invasion speeds only as a consequence of changing environments (i.e., different spatial heterogeneities B) and not due to genetic characteristics of the species in question.

The idea of using ecological emergent properties of a system to approximate evolutionary quantities echoes some approaches from evolutionary demography. In Hiltunen et al. 2014, prey evolution affects the phase diagram of consumer-resource oscillations. The authors propose, based on cycle observations alone, to compute an Evolutionary Dynamics Index quantifying ongoing prey evolution. In our case, having sufficient knowledge of the slope of the optimal niche (B) and of adaptation potential (A) lets us draw predictions on spatial dynamics. Yoshida et al. (2003) showed that it is also possible to infer characteristics of the population genetics based on the nature

of the observed predator-prey cycles.

It would be valuable to use the explicit formula we provide to compare invasion speeds with those observed in nature. Such a work has already been done for numerical approximations of Kirkpatrick and Barton's one-species model. García-Ramos and Rodríguez 2002 explored evolutionary speeds given by this model and compared them to the observed speeds for the expansion of the muskrat (*Ondatra zibethicus*) in Europe. They found empirical expansion speeds to be within the range predicted by the model. However, discrepancies have also been shown between observed expansion speeds and those predicted by the Fisher model (equation (2.2)), as remarked in Grosholz 1996, with speeds being either under- or overestimated. While these discrepancies may be due to an incorrect estimation of ecological parameters, we suggest other possibilities, such as limits due to lagging species adaptation or variation in species interactions.

In order to focus on the role of adaptation, we took a simple approach to ecological dynamics, relying on a simple logistic growth. In the context of species invasion, however, densities are low at the front, so that Allee effects may be commonly encountered. S. V. Petrovskii et al. 2002; S. Petrovskii et al. 2005 showed that for a population model with Allee effects, it is not always possible to observe traveling waves and that various modes of propagation and persistence may be found (for example, patchy invasion). Burton et al. 2010 and Bénichou et al. 2012 also show that expanding fronts usually select for dispersive traits, so that invasion speeds are usually larger than predicted by constant diffusion models.

Changes in species distributions are nowadays commonplace, as species track changes in their environment and due to the accumulation of invasive species transported by human activities. Our results highlight how geographic shifts may rely on different mechanisms when species adaptation happens slowly or fast. Understanding the future of diversity depends on the development of models of co-evolving ecological

networks in heterogeneous space, and the gathering of empirical data documenting simultaneously changes in species trait and distribution.

2.6 Appendices

2.6.1 Infinite adaptation case

We can show that when adaptation potential is high, i.e. when $A \to \infty$, then the solution of the KB equations (system (2.1)) converges to the solution of the Fisher-KPP equation (equation (2.2)). This may seem intuitive, since population density is penalized by the maladaptation term $\frac{1}{2}(z - Bx)^2$, and when population adapts rapidly, this term should become negligible.

We propose that, for fixed values of the cline steepness B, when $A \to \infty$ then the population density for the KB equations, $n_{A,B}$, tends to the solution of the Fisher-KPP equations n_F. We may write this as $n_{\infty,B} = n_F$.

We propose the change of variables $w = z - Bx$ so that equations (2.1a) and (2.1b) are rewritten as

$$\partial_t n = \partial^2_{xx} n + n(1 - n) - \frac{1}{2} n w^2 \tag{2.6a}$$

$$\partial_t w = \partial^2_{xx} w + 2\partial_x \ln n \, (\partial_x w + B) - Aw. \tag{2.6b}$$

If we take equation (2.6b), multiply by w and integrate over x, we obtain (thanks to the integration by parts formula)

$$\partial_t \int w^2 = -\int (\partial_x w)^2 + 2\int w \partial_x \ln n \, (\partial_x w + B) - A\int w^2. \tag{2.7}$$

We wish to find estimates for the second term in the right hand side of this equation. We will assume that $\partial_x \ln n$ is uniformly bounded over A, i.e., for fixed valued of B, $|\partial_x \ln n(t, x)| \leq C$ for every (t, x) with C a constant that does not depend on A.

$$\int w\partial_x \ln n \left(\partial_x w + B\right) = \underbrace{\int w\partial_x \ln n \partial_x w}_{I_1} + B \underbrace{\int w\partial_x \ln n}_{I_2}$$

We can bound the first integral since

$$|I_1| \leq \int |\partial_x \ln n| \, |w\partial_x w| \leq C \left(\int w^2\right)^{\frac{1}{2}} \left(\int (\partial_x w)^2\right)^{\frac{1}{2}},$$

and thanks to Young's inequality we can take some $\varepsilon > 0$ so that

$$|I_1| \leq \frac{C}{2}\left(\varepsilon^{-1}\int w^2 + \varepsilon \int (\partial_x w)^2\right).$$

We need additional assumptions to obtain similar bounds on the integral I_2. For example:

1. If additionally $\partial_x \ln n(t,\cdot) \in L^2(\mathbf{R})$ for every A and the L^2 norms are uniformly bounded over A, say $\int (\partial_x \ln n)^2 \leq C_1$ and C_1 does not depend on A,

$$|I_2| \leq \int\int |w\partial_x \ln n| \leq \frac{1}{2}\left(\int w^2 + \int (\partial_x \ln n)^2\right) \leq \frac{C_1}{2} + \frac{1}{2}\int w^2,$$

   where we used Young's inequality.

2. If additionally $\partial_x \ln n(t,\cdot) \in L^1(\mathbf{R})$ with a uniform bound over A, then we can use Hölder's inequality in the following way

$$\begin{aligned} |I_2| &\leq \int |w\partial_x \ln n| = \int |\partial_x \ln n|^{\frac{1}{2}} |\partial_x \ln n|^{\frac{1}{2}} w \leq \left(\int |\partial_x \ln n|\right)^{\frac{1}{2}} \left(\int |\partial_x \ln n| \, w^2\right)^{\frac{1}{2}} \\ &\leq \sqrt{CC_1}\left(\int w^2\right)^{\frac{1}{2}} \leq \frac{CC_1}{2} + \frac{1}{2}\int w^2. \end{aligned}$$

3. If we do not make additional assumptions on $\partial_x \ln n$ but we suppose for example that we can control the L^1 norm of w by its L^2 norm, and the bound is uniform

over A, we have

$$|I_2| \leq \int |w\partial_x \ln n| \leq C \int |w| \leq CC_w \left(\int w^2 \right)^{\frac{1}{2}} \leq \frac{CC_w}{2} + \frac{1}{2} \int w^2.$$

In any case, we found a bound of the form $|I_2| \leq C_3 + \frac{1}{2} \int w^2$, with C_3 not depending on A.

Replacing the previously found bounds on expression (2.7) we find that

$$\begin{aligned} \partial_t \int w^2 &\leq - \int (\partial_x w)^2 + \frac{C}{2} \left(\varepsilon^{-1} \int w^2 + \varepsilon \int (\partial_x w)^2 \right) + BC_3 + \frac{B}{2} \int w^2 - A \int w^2 \\ &= \left(\frac{C\varepsilon}{2} - 1 \right) \int (\partial_x w)^2 + \left(\frac{C}{2\varepsilon} + \frac{B}{2} - A \right) \int w^2 + BC_3 \\ &\leq \left(\frac{C}{2\varepsilon} + \frac{B}{2} - A \right) \int w^2 + BC_3, \end{aligned}$$

which is true for ε small enough (for example, $0 < \varepsilon \leq C^{-1}$). Defining $C_4 = \frac{C}{2\varepsilon} + \frac{B}{2}$, this implies that

$$\int w(t,x)^2 \mathrm{d}x \leq \int w(0,x)^2 \mathrm{d}x \, e^{(C_4 - A)t} - \frac{BC_3}{C_4 - A} \left(1 - e^{(C_4 - A)t} \right)$$

which tends to 0 for every t when A tends to ∞. This implies in turn that $w(t,x) \xrightarrow[A \to \infty]{} 0$.

We conclude that for any value of B, the function $w(t,x)$ tends to 0 when A tends to infinity. This implies that, in the limit, the population density $n_{\infty,B}$ satisfies the Fisher-KPP equation, so $n_{\infty,B} = n_F$. In other words, the Fisher-KPP equation can be seen as a case of the KB equations when population adaptation potential is infinitely high.

2.6.2 Numerical schemes

We present here the discretization we used to approximate the propagation speed in Kikpatrick and Barton's model. Since it is already cumbersome to analyze it for the one-species model, for the two species model we only present the used scheme and describe briefly the problems we encountered.

As usual for a finite differences scheme, we consider a discretization of a finite time interval $[0, T]$ and a time step Δt, giving a time mesh $t = 0$, $t_1 = \Delta t$, etc., with the general formula $t_k = k\Delta t$, $k \geq 0$; we also consider an one-dimensional space interval $[-L, L]$ and a fixed spatial step Δx so that we have mesh points $x_\ell = -L + \ell\Delta x$, $\ell \geq 0$.

When considering an explicit time-forward scheme, we find the system

$$\frac{n_{k+1,\ell} - n_{k,\ell}}{\Delta t} = \frac{n_{k,\ell+1} - 2n_{k,\ell} + n_{k,\ell-1}}{\Delta x^2} + n_{k,\ell}\left(1 - n_{k,\ell} - \frac{1}{2}\left(\bar{z}_{k,\ell} - Bx_\ell\right)^2\right) \tag{2.8a}$$

$$\begin{aligned}\frac{z_{k+1,\ell} - z_{k,\ell}}{\Delta t} =& \frac{\bar{z}_{k,\ell+1} - 2\bar{z}_{k,\ell} + \bar{z}_{k,\ell-1}}{\Delta x^2} - 2\frac{1}{n_{k,\ell} + \varepsilon}\frac{n_{k,\ell+1} - n_{k,\ell-1}}{2\Delta x}\frac{\bar{z}_{k,\ell+1} - \bar{z}_{k,\ell-1}}{2\Delta x} \\ & - A\left(\bar{z}_{k,\ell} - Bx_\ell\right).\end{aligned} \tag{2.8b}$$

Notice that the solution for $n_{k+1,\ell}$ in terms of the $n_{k,\cdot}$ is almost a convex combination of these terms, explicitly

$$n_{k+1,\ell} = \frac{\Delta t}{\Delta x^2}\left(n_{k,\ell+1} + n_{k,\ell-1}\right) + \left[1 - \frac{2\Delta t}{\Delta x^2} + \Delta t\left(1 - n_{k,\ell} - \frac{1}{2}\left(\bar{z}_{k,\ell} - Bx_\ell\right)^2\right)\right] n_{k,\ell}, \tag{2.9}$$

for it to be a (sub-)convex combination of the solution at different points of the mesh at the instant t_k, we need the coefficients to be greater than zero and for their sum to be at most 1. Supposing that any desirable solution satisfies $0 \leq n_{k,\ell} \leq 1$ for any $k, \ell \geq 0$, the first condition is verified to be true if

$$\frac{2\Delta t}{\Delta x^2} + \frac{\Delta t}{2}\left(\bar{z}_{k,\ell} - Bx_\ell\right)^2 \leq 1 \text{ for every } k, \ell \geq 0. \tag{2.10}$$

It is difficult to predict the values of $\bar{z}_{k,\ell}$ since it is ill-defined whenever $n = 0$ and simulations show instabilities when the $n_{k,\ell}$ are close to zero and the mesh is not well chosen, however, the well working cases show that at the front tip there is an almost constant distance between $\bar{z}_{k,\ell}$ and the optimal phenotype Bx_ℓ. Since in some simulations we imposed $\bar{z}_{0,\ell} = 0$ everywhere, and that locally this distance tends to decrease when $\bar{z}_{k,\ell}$ is too far from the optimum, then $|z_{k,\ell} - Bx_\ell|$ cannot be bigger than BL for a sufficiently big spatial window $[-L, L]$. We find thus that if the condition

$$\frac{2\Delta t}{\Delta x^2} + \frac{\Delta t}{2} B^2 L^2 \leq 1, \text{ or equivalently, } \Delta t \leq \frac{1}{2\Delta x^{-2} + \frac{1}{2} B^2 L^2} \tag{2.11}$$

is met, then the stability condition (3.10) is valid.

Notice that when Δx^{-2} is big enough compared to $B^2 L^2$ then condition (3.11) is just the usual CFL-condition for the stability of explicit finite differences schemes for reaction-diffusion equations. However if B and L are bigger, this stability condition becomes highly restrictive. This actually made explicit schemes of this kind unpractical for our study.

We proposed our own non-linear implicit scheme for Kirkpatrick and Barton's equations given, in the same presented mesh, by the following equations:

$$\frac{n_{k+1,\ell} - n_{k,\ell}}{\Delta t} = \frac{n_{k+1,\ell+1} - 2n_{k+1,\ell} + n_{k+1,\ell-1}}{\Delta x^2} + n_{k+1,\ell} \left(1 - n_{k,\ell} - \frac{1}{2} \left(\bar{z}_{k,\ell} - Bx_\ell\right)^2\right), \tag{2.12a}$$

$$\begin{aligned} \frac{z_{k+1,\ell} - z_{k,\ell}}{\Delta t} =& \frac{\bar{z}_{k+1,\ell+1} - 2\bar{z}_{k+1,\ell} + \bar{z}_{k+1,\ell-1}}{\Delta x^2} \\ & - 2\frac{1}{n_{k+1,\ell} + \varepsilon} \frac{n_{k+1,\ell+1} - n_{k+1,\ell-1}}{2\Delta x} \frac{\bar{z}_{k+1,\ell+1} - \bar{z}_{k+1,\ell-1}}{2\Delta x} - A\left(\bar{z}_{k+1,\ell} - Bx_\ell\right). \end{aligned} \tag{2.12b}$$

Notice that, for each time point t_{k+1}, equation (3.12a) is linear on the vector $n_{k+1,\cdot}$ given that the values $n_{k,\ell}$ and $\bar{z}_{k,\ell}$ are known for each $\ell \geq 0$ and thus it may be

solved by matrix inversion techniques, with coefficients depending on the solution at previous time step t_k. Once the vector $n_{k+1,\cdot}$ is known, the equation for the vector $\bar{z}_{k,\cdot}$ is just a linear one (with time-varying coefficients) that can also be solved with matrix inversion techniques.

Although we did not study the stability of the finite differences scheme (2.12), it behaved well for reasonable mesh parameters, and we were thus able to approximate the propagation speeds for a large family of (A, B) values.

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CHAPTER III

Effects of predation on evolutionary invasion speeds and species distributions

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Abstract

Confronted with global changes and their potential impacts on biodiversity, an important question is to understand the ecological and evolutionary determinants of species geographic distributions. In order to understand how adaptation in heterogeneous environments and interspecific interactions simultaneously constrain such distributions, we then analyze the geographic distribution of a predator-prey system where each species evolves to adapt to a similar environmental cline.

We find that predator dispersal affects their persistence. When their potential of adaptation is low, large dispersal rates lead to predator extinction. The distributions of the predator and the prey are related following one of three different outcomes: local predator extinction when adaptation potential is low, an advancing predator front lagging behind the prey's so that the overlap of the two distributions decreases in time, or a synchronous advancing front when predator adaptation and relative dispersal are potentially better than the prey's. These outcomes can be easily predicted based

on each species intrinsic propagation speed, which reflects their potential for local adaptation and invasion of the landscape in the absence of the other species. We also derive explicit formulae to approximate these speeds in extreme cases, then show that these approximations satisfactorily match our numerical simulations.

We discuss the utility of these propagation speeds as an eco-evolutionary index based on empirical studies.

Key words: antagonistic interaction; local adaptation; partial differential equation model; quantitative genetics.

3.1 Introduction

In response to the current climate changes, many species have been observed to shift their geographic distribution (Parmesan and Yohe 2003). Such changes in the spatial distribution of species may largely alter their co-occurence, thereby affecting the structure of ecological networks (Tylianakis et al. 2008), the functioning of ecosystems and the services they provide. To better understand such consequences, we urgently need to predict how species establish themselves along environmental gradients, but also to understand the mechanisms determining species distributions, so that we can forecast their future changes and thus adapt conservation policies.

To tackle this question, the most common approach relies on the development of niche-based species distribution models (SDMs), which provide predictions of species distributions based on presence/absence data and their association with a given set of environmental variables. Such approaches are thus based on statistical correlations (refer to Guisan and Zimmermann 2000 for an introduction to SDMs or to Guisan and Thuiller 2005 for a more recent review; see also Thuiller et al. 2003 for a comparison of the performance of some SDMs). While some SDMs now incorporate interspecific interactions (albeit in a phenomenological way; Ovaskainen, Abrego, et al. 2016, Ovaskainen, Roy, et al. 2016, Ovaskainen et al. 2017, Tikhonov et al. 2017) they

usually assume niche conservatism and range equilibrium, thus failing to include local adaptation and the dynamics of interspecific interactions. On some occasions, niche models alone may fail to describe the observed distribution of a species, especially in out-of-equilibrium cases such as species invasions. For instance, Broennimann et al. 2007 document a case study in which an invasive species has a different niche in its invasion range, although in this case data does not allow to determine if differences are adaptive (due to a shift in fundamental niche) or ecological (due to another possible realized niche taking place). Understanding such aspects would require the development of models which would simultaneously consider the niche model and a mechanistic approach of eco-evolutionary dynamics (e.g., Bush et al. 2016).

Although adaptation to local conditions should help a population expand its range, boundary populations may be constrained in their adaptation due to the negative effect of gene flows from more central populations, i.e. genetic swamping. For example, Sanford et al. 2006 and Dawson et al. 2010 observed high migration load in boundary populations of a fiddler crab (*Uca pugnax*) and a volcano barnacle species (*Tetraclita rubescens*), respectively, while showing that individuals from the range limit are able to produce offsprings that would survive past the limit. Adaptation may take place on relatively short timescales: Balanya 2006 has shown that *Drosophila subobscura* at the leading edge of an ongoing invasion are able to adapt to local conditions while establishing a cline of genetic characteristics linked to temperature adaptation, following climatic gradients. Rapid adaptation and genetic swamping are quite general phenomena not restricted to species with short generation time. High gene flow has for instance been suggested to occur in many tree species Kremer et al. 2012, with potentially important effects on genetic variance at edge populations. Such evolutionary constraints may play a critical role in the persistence of tree species and in the variations of their geographic distributions, affecting the future of forest ecosystems under climate change scenarios. These studies underline the crucial need of including

local adaptation when studying species distributions, and even more so when the aim is to understand and forecast future distributions under global change. One monospecific spatially-structured model accounting for both local adaptation and migration was presented by Kirkpatrick and Barton 1997. This model explains limited range distribution as an equilibrium between migration and genetic load from maladapted populations.

While this one-species model is a useful starting point, it ignores variation in ecological interactions among species. Such variations in the community context can influence species distributions. For instance, Bullock et al. 2000 showed that range boundaries are constrained by competition in two allopatric gorse species (*Ulex* sp.), since abiotic conditions at the range boundaries are part of both species' niches. Briers 2003 studied parasite prevalence in a snail species (*Lymnaea stagnalis*), suggesting that parasites are the main factor maintaining range boundaries. Competition has also been suggested to be the main driver determining range limits for andean birds Terborgh and Weske 1975. These examples suggest that interspecific interactions should not be neglected in a general framework for species distributions and range boundary studies.

A possible way forward is therefore to extend the model of Kirkpatrick and Barton 1997, accounting for variation in ecological interactions. Case and Taper 2000 studied the effects of interspecific competition on geographic distribution along an environmental gradient. Their results show that density decrease may have an important effect on gene flow at a competing edge, limiting the range of both competing species. Norberg et al. 2012 found that allowing for adaptation but neglecting competition may overestimate the proportion of persisting species submitted to climate change. They also found that extinction debts may arise even after global change has stopped, as eco-evolutionary dynamics eventually cause some species to become extinct. Holt et al. 2011 similarly found that demographic effects caused by predators

may alter gene flow in their prey, limiting ranges in some cases, or even expanding them in others. However, they neither considered predators' local adaptation to the environment nor the variation of their range.

Interactions between community and evolutionary contexts have also been observed in empirical studies. Charmantier et al. 2004 found that *Protocalliphora* parasites can decrease the heritability of tarsus length in the blue tit (*Parus caeruleus*). Carroll et al. 2005 showed that Australian soapberry bugs (*Leptocoris tagalicus*) rapidly evolved beak morphologies allowing them to feed on an invasive plant species (*Cardiospermum grandiflorum*). Brodie et al. 2002 even showed a coevolutionary geographic relationship between the poison resistance of garter snakes (*Thamnophis sirtalis*) and poisonous newts (of the genus *Taricha*) with varying degrees of coadaptation, establishing a continuously varying cline of phenotypes related to this poison. Although the existing cline in poison resistance was not tested to be a consequence of gene flow between differently adapted populations, this is a likely explanation. The interplay of ecological interactions and evolutionary dynamics first motivated the geographic mosaic of coevolution concept (Thompson 1999), relying on the idea that there exist hotspots and coldspots of coevolutionary relationships between species, allowing the emergence of continuous clines of co-adapted phenotypes.

In the present work, we propose to study how the combined effects of predation and adaptation, for both prey and predators alter their propagation and distribution. Our goal is to better understand the covariation of prey and predator distributions and the scenarios under which extinction may be likely. We propose a spatially explicit model accounting simultaneously for local adaptation to the environment along an optimal phenotypic cline and predator-prey interactions. We address two question of how trophic interactions alter species propagation. We explicitly tackle how the distribution of predators relate to the distribution of prey along the environmental cline and show that only three qualitative outcomes are possible. We also show that

these outcomes can be predicted based on intrinsic propagation speed of each species (prey and predator) and provide the expression of these intrinsic speeds. Based on this analysis, we discuss the role of species adaptation and dispersal.

We present our results obtained with a two-species predator-prey model, making explicit the link with the one-species model presented in Méndez-Vera et al. 2019.

3.2 Effects of dispersal and adaptation potential on the evolution of species ranges in a predator-prey framework

3.2.1 Model presentation and main assumptions

While single-species models in the line of Kirkpatrick and Barton 1997 and Méndez-Vera et al. 2019 highlight the effect of varying adaptation potential for a given species, they do not account for the fact that the community context may also vary along the environmental cline. The density of enemies may for instance vary, affecting species density, thereby modulating local adaptation and affecting the distribution of gene flows. To tackle this part of the question, we turn to a predator-prey model in the same heterogeneous space scenario and study the relationship between predator and prey geographic distributions. The derivation of the model builds on the one-species model described in Méndez-Vera et al. 2019. It follows that of Case and Taper 2000, but here we propose a predator-prey scenario instead of a competition one (refer to the Appendix 3.4.1 for details).

We consider two interacting prey and predator populations distributed along one-dimensional space parametrized by the variable $-\infty < x < \infty$, with dynamics taking place over continuous time $t \geq 0$. We suppose populations are structured by a phenotypic variable $-\infty < z < \infty$, and that there is an optimal phenotypic value for each spatial position, which we will denote $\theta_1(x) = b_1 x$ for prey and $\theta_2(x) = b_2 x$ for predators. Note that because b_1 and b_2 may differ, the environmental cline may be

relatively harsher for one of the two species.

We denote by $n_1(t,x)$ (resp. $n_2(t,x)$) the local density of prey (resp. predators) at time t and spatial location x, and we will consistently associate the subindex 1 (resp. 2) to prey-related (resp. predator-related) variables and parameters. We also denote by $\bar{z}_i(t,x)$, for $i=1$ or $i=2$, local mean trait value at time t and space x.

In order to simplify the system by reducing the number of intervening variables, we measure time in prey generation times and space in prey diffusion scale (refer to Appendix 3.4.1 for a detailed description of the model and its non dimensional form). With this consideration, our predator-prey system is given by the following equation system:

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \frac{1}{2}\left(b_1 x - \bar{z}_1\right)^2 - \beta n_2\right) \tag{3.1a}$$

$$\frac{\partial n_2}{\partial t} = \delta\frac{\partial^2 n_2}{\partial x^2} + r n_2\left(n_1 - d - n_2 - \frac{1}{2}\left(b_2 x - \bar{z}_2\right)^2\right) \tag{3.1b}$$

$$\frac{\partial \bar{z}_1}{\partial t} = \frac{\partial^2 \bar{z}_1}{\partial x^2} + 2\frac{\partial \log n_1}{\partial x}\frac{\partial \bar{z}_1}{\partial x} - a_1\left(\bar{z}_1 - b_1 x\right) \tag{3.1c}$$

$$\frac{\partial \bar{z}_2}{\partial t} = \delta\frac{\partial^2 \bar{z}_2}{\partial x^2} + 2\delta\frac{\partial \log n_2}{\partial x}\frac{\partial \bar{z}_2}{\partial x} - r a_2\left(\bar{z}_2 - b_2 x\right). \tag{3.1d}$$

Refer to Table 4.2 for an interpretation of the intervening parameters, which are assumed to be constant.

The first two equations model the population densities, accounting for intra- and interspecific interactions and a penalization in fitness due to deviation of a species-specific phenotype from the local optimum. The last two equations model each species trait dynamics, and they are similar for both species, but the one corresponding to the predator's trait must be corrected for different diffusion rate δ and growth rate r. In both of equations (3.1c) and (4.14b), the first term models the diffusion of genes that is linked to the diffusion of individuals, while the second term corrects for asymmetries in gene flows (gene flow being more important from large populations to contiguous

Parameter	Description
t	*Non-dimensionalized time coordinate, measured in the prey growth rate timescale.
x	*Non-dimensionalized space coordinate, measured in prey diffusion scale.
n_i	*Non-dimensionalized population densities, measured as a fraction of their respective carrying capacities.
$\bar{z}_i$	*Non-dimensionalized trait value.
δ	Predator diffusion relative to that of its prey.
r	Maximal predator growth rate relative to that of its prey
β	Predation rate in the non-dimensional system.
d	Compound death rate of the predators measured in the timescale given by t.
a_i	Adaptation potential.
b_i	Cline slope.

Table 3.1: Description and measure units of each of the variables (marked with a *) and parameters used in the non-dimensional predator-prey model. All of the parameters are assumed to be constant. We take the subscript $i = 1$ to indicate prey-related quantities, and $i = 2$ to indicate predator-related quantities.

small populations than the other way round). The third term corresponds to the effects of local adaptation due to directional selection, driving the mean phenotype value $\bar{z}_i$ toward the local optimum $b_i x$ at a rate a_1 or ra_2, respectively.

Notice that the parameters of relative diffusion δ and relative basic population growth rate r appear only in the predator equations. This is due to the fact that time and space are measured in prey-related scales, and predator dispersal and population growth rates may differ from the prey ones, so that these parameters are relative to the prey diffusion and population growth rate, respectively. Similarly, the predator adaptation potential a_2 may differ from the prey adaptation potential a_1.

In the following section, we will analyze system (3.1). First, we study its equilibrium points, which correspond to states of either coexistence or extinction (of one or both species). This allows us to provide a description of its solutions, which are understood as extinction or invasion fronts for each species. Then, we provide some

definitions that allow us to understand the described solutions; the characteristic speed of these definitions is an important quantity allowing us to understand what happens in most cases, which is analyzed in the third place, through numerical approximations.

3.2.2 Homogeneous equilibria and coexistence

A first intuitive question is whether the two-species model (3.1) allows for species coexistence. It turns out there are three equilibrium points where population density is spatially homogeneous, one where both populations are extinct, another where predators are non-existent and prey population attains its carrying capacity, and a third one with predator-prey coexistence.

More explicitly, there are three plausible constant equilibrium values for the population density pairs (n_1, n_2),

$$E_0 : (n_1, n_2) = (0, 0), \quad E_1 : (n_1, n_2) = (1, 0), \quad E_2 : (n_1, n_2) = \left(\frac{1 + \beta d}{1 + \beta}, \frac{1 - d}{1 + \beta} \right) \tag{3.2}$$

with $\bar{z}_i = b_i x$ whenever $n_i > 0$. We define $n_1^* = \dfrac{1 + \beta d}{1 + \beta}$ and $n_2^* = \dfrac{1 - d}{1 + \beta}$.

At the coexistence equilibrium, both predator and prey densities are decreasing functions of β. Prey equilibrium density only depends on predator parameters; as typical in top-down controlled, Lotka-Volterra like models (e.g. Oksanen and Oksanen 2000). Increasing predator compound death rate d (in the timescale t) has a positive effect on prey density and a negative one on predator density. For the coexistence equilibrium to make biological sense, we need $d < 1$ which means that death rate rd must be smaller than basic growth rate r when prey density is at its carrying capacity. For higher death rates, predators are extinct.

Species' geographic limits correspond to locations where population densities change from a positive value on one side to zero on the other side, with different possible

values for their phenotypes depending on their adaptation potentials. In order to study what happens at a range edge, we consider sharp transition functions going from the values in the coexistence equilibrium E_2 to the extinction one E_0, and we suppose that populations are initially perfectly adapted everywhere. Although this last assumption may not be valid in nature, we expect migration effects to transform hard boundaries into smooth ones, and to make asymptotic regimes independent from initial conditions, so that this assumption is superfluous.

3.2.3 Propagation fronts and intrinsic propagation speeds

Natural populations may progressively invade space if their adaptation potential is strong enough so that emigrating populations are able to adapt quickly. This is captured by our model and we found three different qualitative behaviors for its solutions with one of them having one important subtype. Examples for each one of these behaviors are depicted in Figure 3.1. In general, predator and prey densities are established through space as advancing or retracting fronts with phenotype variation growing away from or towards the optimal phenotype value at the same rate as its corresponding population density (as in the one-species system, see for example García-Ramos and Rodríguez 2002). In other words, solutions for each species are synchronous density and phenotype fronts advancing or retracting in space.

Panels I and II in Figure 3.1 show solutions with prey and predator having different propagation speeds. Panel I shows prey invading space (so it is an advancing front, or a propagating front with positive speed), while its predator is (locally) becoming extinct (a retracting front, or a propagating front with negative speed); panel II shows both a prey and its predator invading space but at different speeds, the predator lagging behind.

Panels III.1 and III.2 show both prey and predator populations invading simultaneously as a community. In both cases, predator and prey are established propagating

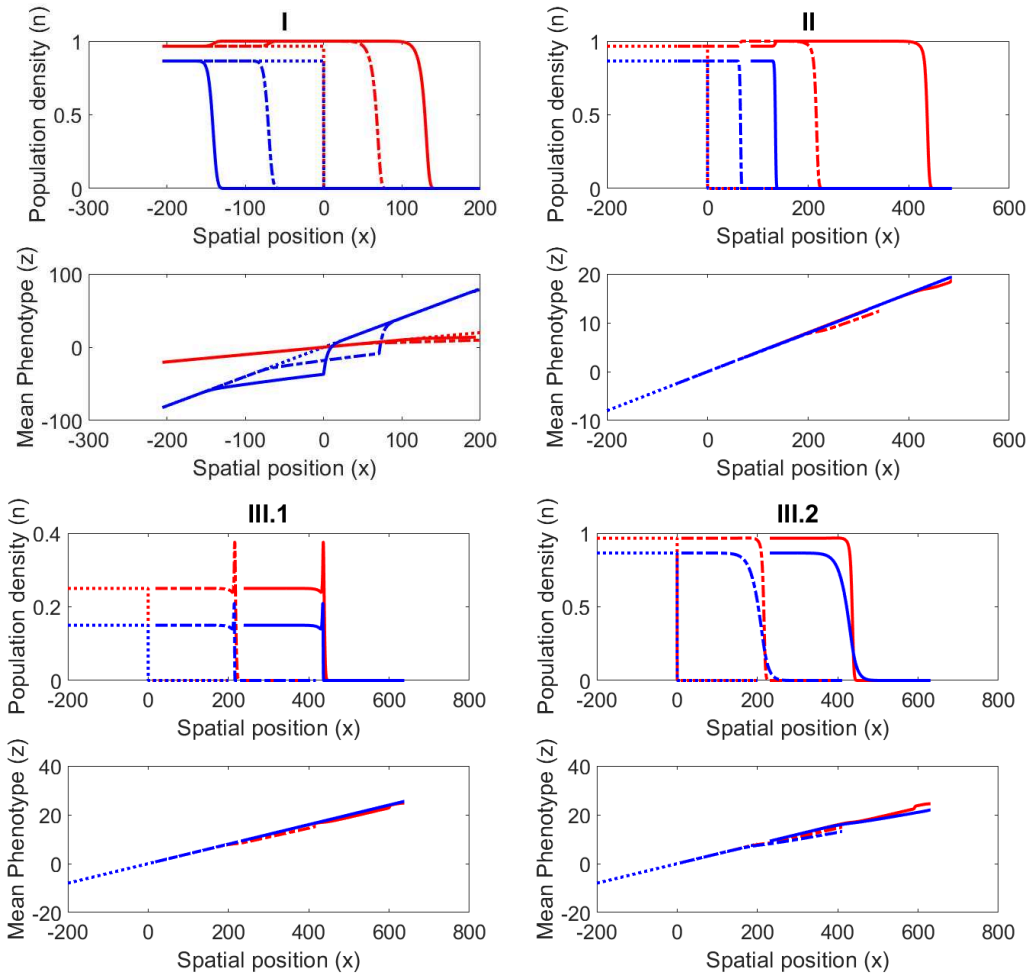

Figure 3.1: Four solution examples showing the different qualitative behaviors for the model (3.1). Red color indicates prey related data, while blue indicates predator related data. Dotted lines correspond to initial conditions, continuous lines show solutions for a large time value and the dash-dotted lines correspond to an in-between state. Each panel shows the two variables n and z for the same parameter set. The parameters that were used for each panel are: I. $a_1 = 0.05$, $b_1 = 0.1$, $a_2 = 0.5$, $b_2 = 0.4$, $\beta = 0.04$, $d = 0.1$, $r = 0.8$, $\delta = 5$; II. $a_1 = 0.1$, $b_1 = 0.04$, $a_2 = 0.5$, $b_2 = 0.04$, $\beta = 0.04$, $d = 0.1$, $r = 0.8$, $\delta = 0.1$; III.1. $a_1 = 0.1$, $b_1 = 0.04$, $a_2 = 0.5$, $b_2 = 0.04$, $\beta = 5$, $d = 0.1$, $r = 20$, $\delta = 0.1$; III.2. $a_1 = 0.1$, $b_1 = 0.04$, $a_2 = 0.5$, $b_2 = 0.04$, $\beta = 0.04$, $d = 0.1$, $r = 0.8$, $\delta = 100$.

fronts advancing at the same speed. Notice that in panel III.2 there is a predator spillover at the front tip, because of their large relative diffusion δ, which cause predators to invade space *before* their prey. In this subcase, the trophic interaction has an important effect on the invasion speed of the community, which is slower than it would be if predators were not present.

We can understand these three qualitative outcomes by considering the specific propagation speeds, defined as propagation speeds in ideal scenarios for prey and predators. The differences between these prey and predator's intrinsic speeds generally allows to discriminate between the three types of solutions (but not between the two subtypes of solution III). Thus,

- We call the *intrinsic prey speed* the speed of the traveling front established by prey in an environment where predators are absent everywhere. This corresponds to imposing $n_2(t, x) = 0$ for every t and x (and ignoring z_2) in equation (3.1), which results in Kirkpatrick and Barton's equations for one species. If we refer to this intrinsic prey speed as c_1^I and the speed of the one-species system (as presented in Méndez-Vera et al. 2019) with parameters A and B as $c_{KB}(A, B)$, it follows that $c_1^I = c_{KB}(a_1, b_1)$.

- We call the *intrinsic predator speed* the speed of a front composed of predator individuals in an ideal scenario where prey are present everywhere. In terms of the equilibrium points of the equation, this corresponds to a traveling front joining equilibria E_2 and E_1. We will refer to this speed as c_2^I, and it may depend on all eight parameters of the model (see equation (3.3)).

We finally introduce two more speeds, corresponding to prey and predator traveling speeds when they are simultaneously establishing traveling fronts (possibly advancing together or not), which we call *joint speeds*. We note them c_1^J and c_2^J, respectively. This corresponds to solutions joining E_2 and E_0.

The examples presented in Figure 3.1 already show two important types of solutions: one in which prey and predator populations advance together on a joint community front, and another in which predators advance independently from their prey on a slower front, with both fronts growing farther apart. In terms of the joint speeds we just defined, this means that solutions will have either $c_1^J = c_2^J$ or $c_1^J > c_2^J$.

We will see that for a large number of cases, the intrinsic speeds are good predictors of the joint speeds, furthermore, the predator intrinsic speed is well approximated by an equivalent speed in the one-species model (taking into account appropriate scalings), explicitly given by (refer to the appendix 3.4.3 for the proof)

$$c_2^I \approx \sqrt{\delta r\,(1-d)}\, c_{KB}\left(\frac{a_2}{\sqrt{1-d}}, \frac{b_2}{1-d}\sqrt{\frac{\delta}{r}}\right). \tag{3.3}$$

This formula is difficult to analyze because we do not have an explicit expression for the speed c_{KB}, but we can still draw some conclusions:

- Predator speed is increasing as a function of relative predator growth rate r due to two reasons. The first is that a larger r diminishes the perceived spatial heterogeneity $\frac{b_2}{1-d}\sqrt{\frac{\delta}{r}}$, so that faster breeding predators have less trouble adapting for a same level of adaptation potential a_2 and absolute heterogeneity b_2. The second is that speed is scaled as $\sqrt{r}$, so that even if adaptation was "infinite" in the sense that c_{KB} attained its (near) maximum value (refer to Figure 1 in Méndez-Vera et al. 2019), speed can still grow unboundedly. In other words, an increase in growth rate causes an increase in invasion speeds because it lets population not only colonize larger distances in a given amount of time (effect of $\sqrt{\delta r}$), but also because it helps surmount spatial heterogeneity (effect of $\sqrt{\delta/r}$).

- When predator death rate d approaches 1, the perceived spatial heterogeneity changes faster than the adaptation potential, which makes adaptation harder. Notice that speed is also proportional to $\sqrt{1-d}$ which scales the perceived

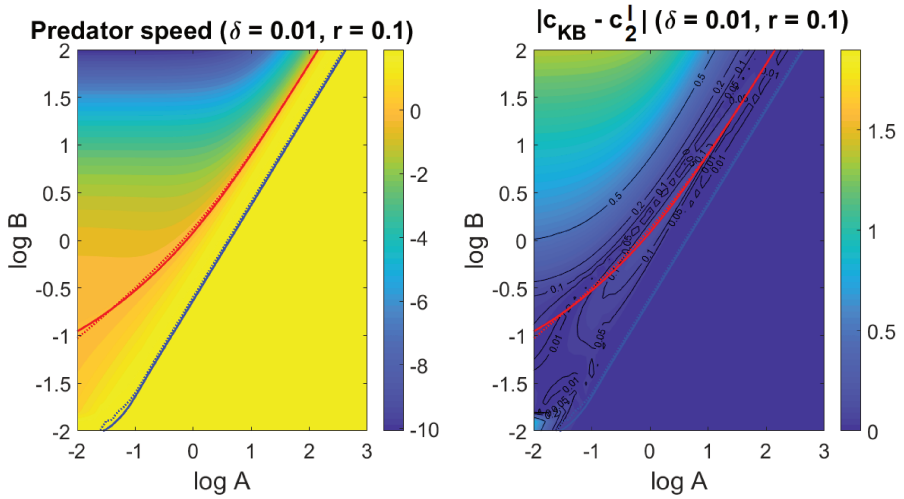

Figure 3.2: At the left, the independent predator speeds for a variety of parameters $A = \dfrac{a_2}{\sqrt{1-d}}$ and $B = \dfrac{b_2}{1-d}\sqrt{\dfrac{\delta}{r}}$. At the right, the absolute difference between the independent predator speeds and their approximation when using an equivalent one-species model. Blue color lines correspond to infinite adaptation limit, while the red ones correspond to the zero-speed limit.

speed closer to 0. The value of d which maximizes speed is $d = 0$, i.e., when there is no natural predator death rate.

- Concerning the effects of relative predator dispersal δ, on the one hand, speed is proportional to $\sqrt{\delta}$, which is the expected effect of diffusion on propagation speeds. On the other hand, larger δ also distort perceived geographic heterogeneity, making it more pronounced. This leads to a trade-off along which speed may increase or decrease as a function of δ, depending on the value of the adaptation potential.

We now assess the accuracy of our speed approximation, when relying on intrinsic speeds. To do so, we simulated system (3.1) and compared propagation speeds to those of the corresponding one-species systems. Our results are shown in Figure 3.2.

The left plot in Figure 3.2 shows the intrinsic predator speeds for the system (3.1) after a variable change correcting for the predator effective growth rate (refer

to Appendix 3.4.3 for details), with the blue line indicating the infinite adaptation limit and the red one showing the zero-speed limit, notice how it resembles the one species case (Méndez-Vera et al. 2019). The right plot in 3.2 shows the difference between these intrinsic predator speeds and the corresponding speeds when using an approximate one-species model. Notice how good the approximation is for positive speeds, i.e., to the right of the red line, and it is good to a considerable interval to the left of this line.

In other words, formula (3.3) can help us accurately approximate invasion speeds and, although it is flawed for a large portion of extinction speeds, it can help us discriminate propagation fronts as type I, II or III. Formula (3.3) is also useful because it links the two-species model with the one-species one (Méndez-Vera et al. 2019). When $A' = a_2/\sqrt{1-d}$ is small enough compared to $B' = b_2/(1-d)\sqrt{\delta/r}$, the small adaptation approximation in Méndez-Vera et al. 2019 (equation (3) therein) may be used to approximate intrinsic predator speed; and when it is large enough then $c_{KB}(A', B')$ is well approximated by Fisher's model speed $c_F = 2$. Of course this is also true for the prey applying the same reasoning to $c_{KB}(a_1, b_1)$.

To summarize, in terms of the relation between intrinsic and joint speeds, we expect that:

- Whenever $c_2^I < c_1^I$ the joint speeds of predator and prey will be equal to their respective intrinsic speeds, $c_2^I = c_2^J$ and $c_1^I = c_1^J$, and we will see a prey and a predator front separated by a growing gap.

- Otherwise $c_2^I > c_1^I$ which means that predator are intrinsically better at colonizing than their prey. Nevertheless, in the joint scenario they are limited by their prey, so that predator front tip will reach the prey one and both populations will invade at the same speed, $c_2^J = c_1^J$. In this case we will have $c_2^I > c_2^J$, and we will see that $c_1^I \geq c_1^J$, i.e., prey may experience a loss in speed due to predation effects.

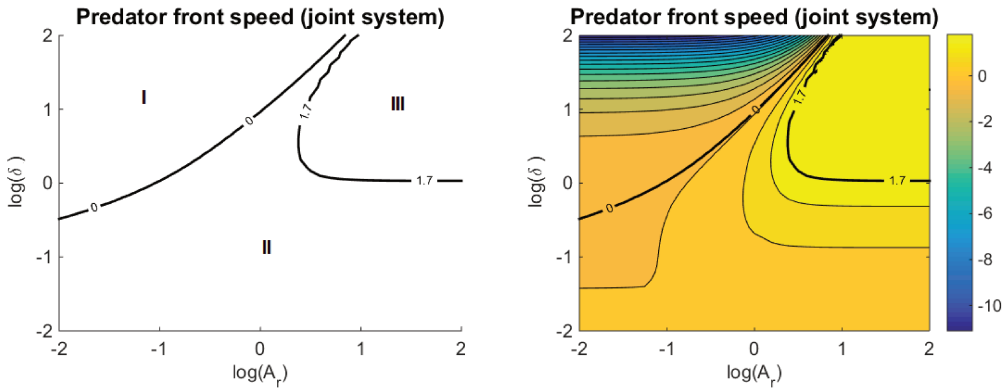

Figure 3.3: Phase graphic determining three different behaviors of the model depending on two parameters, relative adaptation potential $A_r = \frac{a_2}{a_1}$ and relative diffusion δ. The rest of the parameters, inclusing a_1, is left fixed. The zones correspond to local extinctions of the predators (zone I), local invasion of the predator population with a growing gap between the front tips (zone II) and simultaneous invasion of both populations (zone III).

There is a third case that we did not consider, $c_2^I = c_1^I$. Although mathematically possible, it seems highly unlikely that predators and prey from a same community will have the exact same intrinsic invasion speed because of fundamental differences in their growth rates, adaptation potential, dispersive capabilities, etc.

3.2.4 Front classification and geographic distribution

We studied more closely the solutions where both predator and prey establish invasion fronts, i.e., solutions joining equilibria E_2 and E_0 (as defined in (3.2)), and the dependence of asymptotic speed on relative diffusion δ and relative adaptation potential $A_r = \frac{a_2}{a_1}$, leaving a_1 and the other parameters fixed. We only studied the case where prey population can invade the whole space because predators are restricted by prey availability and thus prey extinction and limited range cases would not reveal interesting predator dynamics. In this scenario, we discovered that invasion fronts can be classified in three types depending on the propagation dynamics, as depicted schematically in the left panel of Figure 3.3 left.

These three kinds of fronts correspond respectively to:

I. *Local extinctions of predators*, which can result in total extinction or persistence in a limited area. These are characterized by negative traveling speeds $c_2^J < 0$ and total extinction and localized persistence cannot be distinguished based on speeds alone.

II. Predator invasion with a *growing gap* between predator and prey's front tips. Propagation speeds in this range satisfy the relation $0 < c_2^J < c_1^J$.

III. *Simultaneous invasion* of both prey and predators. For parameters in this range, prey and predator invade the whole space at the same speed $c_1^J = c_2^J$.

We can also distinguish a sub-pattern in the simultaneous invasion scenario, whereby predators may decrease the invasion speed of prey, as depicted in Figure 3.4. This sub-pattern is caused by interference on prey speeds due to interaction with predators. The left panel in Figure 3.4 shows prey speed is constant as a function of $A_r = a_2/a_1$ and δ except when predators' adaptation and diffusion are large, whereas the right panel in Figure 3.4 shows that predators' speed matches that of their prey, and that it decreases for large values of δ and A_r even though it should be expected to increase in this range. Under this scenario, predators have reached the prey's front tip and they are able to decrease their population noticeably, implying a decrease in invasion speed. Notice that this pattern also satisfies $c_2^J = c_1^J$, but $c_1^J < c_1^I$ in the interference zone.

Intrinsic speeds can explain much of what happens, and can thus help us classify these fronts. In Figure 3.3, prey speed was found to be constant and equal to its intrinsic value, so that variation in the outcome is only due to variations in relative predator characteristics (i.e., relative diffusion δ and adaptation potential a_2). On the one hand, when predator adaptation potential a_2 is small, or when it is large but its diffusion relative to its prey's, δ, is small, the intrinsic speed of predators will be smaller than their prey's, so each population advances in space at their own speed

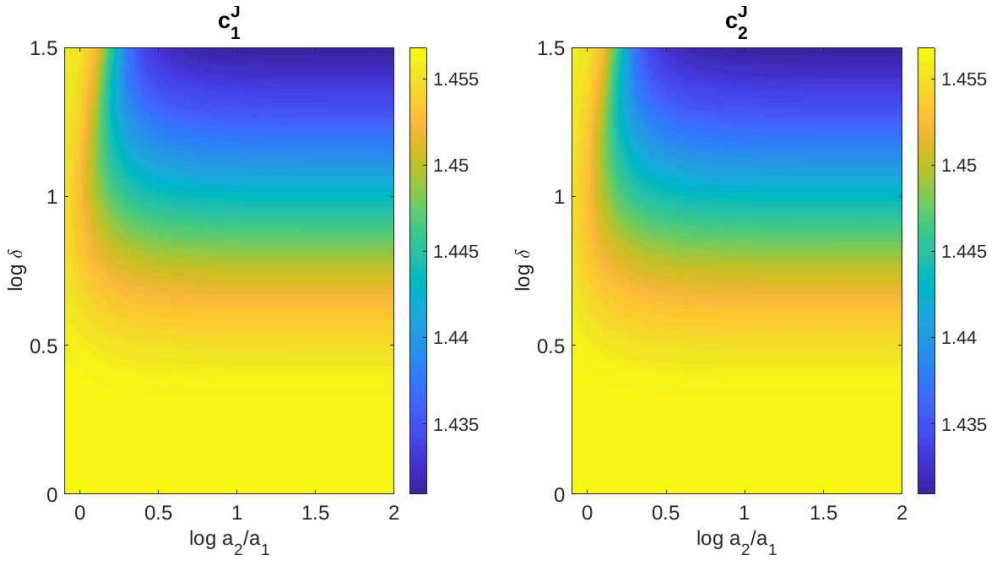

Figure 3.4: Variation of prey and predator speeds in the joint system depending on the parameters $A_r = \frac{a_2}{a_1}$ and $\delta = \frac{\delta_2}{\delta_1}$. Intrinsic speed of prey for all parameters is $c_1^I = 1.456$.

without interfering with the other's speed, thus leading to qualitative outcome I or II. On the other hand, the predator population may have a large relative diffusion and a strong adaptation potential so that predator intrinsic speed is larger than their prey's. In that case, both species will be invading together, but the predator front will reach the prey front, and both species will advance at the same speed, resulting in outcome III.

Figure 3.4 shows additionally that, when predators' and prey's range edges meet and advance together, they may do so at a speed even lower than the prey's intrinsic speed. This only happens when predator adaptation potential and diffusion are large enough. Referring back to Figure 3.1, the front depicted in panel III.2 matches this pattern, because beyond the prey range limit, the predator population is positive, due only to emigration from central populations. This presence of predators beyond the prey range limit hinders the prey speed of propagation.

In summary:

- Whenever $c_2^I < c_1^I$ predator and prey edges do not meet, so that there is not

interference at the front tips and each species advances at its own intrinsic speed. If $c_2^I < 0$ predators go extinct (outcome I), otherwise propagation of both species will occur, with a growing gap (outcome II). In terms of geographic distribution, this second case leads to a decrease in the overlap of the two distributions in time.

- When $c_2^I > c_1^I$ we get a simultaneously invading predator-prey front (outcome III). Whether predators interfere with prey speed or not is impossible to tell apart based on intrinsic speeds alone. Prey speeds are decreased when diffusion causes a non-negligible density of predators to be present at the prey's front tip (predator spillover). When outcome III occurs, the geographic distribution of both species eventually perfectly overlap and this overlap is maintained in time.

3.3 Discussion

Our model highlights how species adaptation and interaction can affect species extinctions and their geographic distributions. We relied in the single-species model presented in Méndez-Vera et al. 2019 which highlights how different mechanisms act when considering low vs high adaptation potential. In the present work, we added trophic interactions and showed that only three qualitative outcomes are possible: (i) the predator may go extinct even though the prey invades the system, it can also follow the prey, its geographic distribution either (ii) perfectly overlapping with the prey, or (iii) increasingly lagging behind.

We studied the effects of local adaptation on predator and prey's geographic distribution, by looking at propagation speeds. We found that the speeds of predators and prey can often be well approximated by intrinsic speeds, assessed for each species separately. These approximated speeds are easier to understand, and since they allow us to predict most of the presented scenarios, we deemed important to provide a way

to estimate them. It turns out that they are well approximated by one-species cases. The approximations found in the one-species model (Méndez-Vera et al. 2019) are then applicable to the predator-prey model, having explicit expressions in extreme cases.

Our predator-prey model is an effort towards understanding the effects of eco-evolutionary dynamics of predators and prey on species' geographic distribution. We explored mainly the dependence of the predator's front speed on its adaptation potential and diffusion, and consequences on the overlap of the two distributions. Our results, summarized in Figures 3.3 and 3.4, can be understood by focusing our attention on predator relative diffusion, in units of prey diffusion rate.

Prey intrinsic speed is simply their propagation speed when predators are absent, which coincides with the one-species model speed. Thus, for the predator-prey system, prey propagation is most often limited in the same way as in the one-species model, i.e., by their adaptation potential and the spatial heterogeneity of the environment (Méndez-Vera et al. 2019). Predators may, however, negatively affect prey propagation given two conditions: larger predator intrinsic speed and large predator dispersal. Spillover of predators in advance of the prey's invasion front then occur, diminishing prey fitness and thus reducing its propagation speed through top-down control. Such spillovers rely on two distinct components. The invasion component dictates the invasion speed of the predator: if adaptation potential and dispersal allow for intrinsic predator invasion speed to be larger than the prey's, then predators will be present at the front tip. The ecological component relies on the trophic top-down effect, which decreases prey speed through the control of prey fitness at the front. Predation rates need to be sufficiently high for this phenomenon to be noticeable. We did not explore whether this negative effect on speed might have critical effects on prey distribution, for example by limiting range for prey that would otherwise invade the whole space, but it is theoretically possible (as noticed for example in Holt

et al. 2011).

Predators are limited by prey availability, so that predator distribution is necessarily nested inside their prey's. We found that joint propagation speeds (when prey and predators propagate synchronously) correspond to predators not intrinsically faster than their prey. Thus, provided that predator intrinsic speed is smaller than their prey's, a well parameterized one-species system will govern predator propagation (see Appendix 3.4.3). Otherwise, predator and prey will propagate at the same speed. There are thus three possible outcomes for the co-variation of predator-prey geographic distributions: (I) predators may become extinct or persist in a limited range; (II) predators may invade, lagging behind their prey (III) both species may invade at the same speed, forming a community invasion front. These are the scenarios exemplified in Figure 3.1 and depicted in Figure 3.3. Interestingly, these qualitative scenarios can be predicted based only on intrinsic propagation speeds. When the two species invade at the same speed, however, two forms of invasion are possible which are not distinguishable based on intrinsic speeds alone: (III.1) the predator-prey community may advance at the prey's intrinsic speed or (III.2) prey may be slowed down and thus the predator-prey community invades at a lower speed than the prey's intrinsic one. This latter outcome is interesting, as it underlines how the interaction between species adaptation and top-down control can affect changes in species distributions.

Such outcomes may be compared to empirical datasets that consider the geographic ranges of several interacting species. Ongoing invasions offer good opportunities in this regard, because they provide a setting in which all three outcomes can possibly occur, depending on invader's traits and genetic variance for local adaptation. Coon and Martin 2014 observed that, for house sparrows (*Passer domesticus*) introduced in Kenya, the distribution of various heamosporidians parasites (of the genera *Haemoproteus*, *Plasmodium* and *Leucocytozoon*) did not depend on distance from the introduction site, being prevalent all along the sparrows' range, suggesting a

type III qualitative outcome. Regarding the case of the invasive cane toad (*Rhinella marina*) in Australia and one lungworm parasite (*Rhabdias pseudosphaerocephala*), however, Phillips et al. 2010 observed that the parasite follows its host with a lag between front tips, suggesting a type II outcome. The reduction of prey speed due to predator spillover and top-down effects can also be related to empirical data. On the mount St. Helens (Washington State), herbivory by insects limited the invasion of the *Lupinus lepidus* species (Fagan and Bishop 2000), suggesting a type III.2 outcome.

Our results could also be used for conservation or control policies. The checkered beetle species (*Thanasimus dubius*) is a predator of the southern pine beetle (*Dendroctonus frontalis*), a forest pest. The former was found to have a higher than expected diffusion rate (Cronin et al. 2000). One proposition made by Cronin et al. 2000 was to decrease this beetle's dispersal rate in order to effectively control the southern pine beetle pest. Our results suggest otherwise: high predator diffusion rates are only detrimental to their persistence when their adaptation potential is low. In other cases, a high diffusion rate of predators is required for them to properly regulate prey densities, while lower dispersal would lead to a distribution lag that would not allow a proper biological control.

Concerning the role of dispersal for species persistence, we note that in our model dispersal does not systematically have a positive role. It is usually believed that large species dispersal rates are necessary for populations to be able to track changes in their environment (e.g. to track their climatic niche; especially if inhabitable space is geographically constrained; see Clobert et al. 2012 for a review on this topic, Alex Perkins et al. 2013 for an approach to a particular case or Shigesada and Kawasaki 1997 more generally). However, Figure 3.3 shows that, when predator diffusion is too large relative to that of its prey, predators may be driven to extinction if their adaptation potential is limited. Gene swamping then leads to maladaptation, as large diffusion and poor adaptation potential lead to high fluxes of maladapted individuals

from central zones to the periphery (Kirkpatrick and Barton 1997). This is actually not surprising in the context of heterogeneous space: large diffusion rates for a population adapted to local conditions mean that emigrating individuals will have to face more adverse conditions than they would have had with less dispersal. If this new environment requires a phenotypic optimum that is too different from the dispersing individual's phenotypic trait, either this individual will not be able to survive, or it will bring about maladapted genes making it difficult for its progeny to survive if the population has not enough adaptation potential. Such outcomes can again be linked to empirical examples. Some plant species seem unable to establish themselves in areas far outside their range due to habitat unsuitability (Sanford et al. 2006), and some crab species maintain a defined range limit since limitations in larval development at low temperatures are due to gene flow from central, warmer populations (Primack and Miao 1992).

Changes in species distributions are nowadays commonplace, as species track changes in their environment and due to the accumulation of invasive species transported by human activities. While characterizing species abiotic niche is a legitimate first step to understand these phenomena, our model suggests that shifts in ecological interactions matter, especially when enemies disperse in efficient ways. Our results also highlight how geographic shifts may rely on different mechanisms when species adaptation happens slowly or fast. Understanding the future of diversity depends on the development of models of co-evolving ecological networks in heterogeneous space, and the gathering of empirical data documenting simultaneously changes in species trait and distribution.

3.4 Appendices

3.4.1 Model derivation

In a general setting, as described in the model presentation, we suppose that two species are distributed along a one-dimensional axis parametrized by the variable $-\infty < X < \infty$, and they are structured by a phenotypic trait $Z \in \mathbf{R}$ (possibly taking negative values depending on the measurement scale). Each individual's absolute fitness w may then depend on their location X, time $T \geq 0$ (assumed to be continuous) and their phenotype Z and it may be decomposed into two terms, one we call r which depends on the environment, and another one we call I depending on the interactions with other individuals. We further assume that there ir an optimal phenotypic value for each spatial position, which we will denote $\theta_1(X) = B_1 X$ for prey and $\theta_2(X) = B_2 X$ for predators. Thus, for each individual, the absolute fitness may be written as (refer to Tables 4.1 and 3.3 for the meaning of parameters)

$$w(T, X, Z) = r(T, X, Z) + I(T, X, Z).$$

Assuming that individuals follow the same dynamics and that resources regenerate infinitely fast, a prey individual's environmental fitness is given by

$$r_1(T, X, Z) = r_1^{max} - \frac{1}{2V_1^s} \left(Z - \theta_1(X)\right)^2,$$

and a predator's is given by

$$r_2(T, X, Z) = -\bar{d} - \frac{1}{2V_2^s} \left(Z - \theta_2(X)\right)^2.$$

In order to write down the interaction part of the fitness we need to specify the way phenotypes of other preys and predators affect each type of individual. We will do this

by specifying interaction kernels, which account for the increase or decrease in fitness when an individual of type Z' interacts with an individual of type Z. The competition kernel, α^i measures the per capita effect on fitness of intraspecific interaction:

$$\alpha^i\left(Z,Z'\right)=-\alpha_i\exp\left(-\frac{1}{4V_i^u}\left(Z-Z'\right)^2\right).$$

We may specify the per capita effect of predation by a predator of trait Z' over a prey of trait Z, but we will suppose that the trait does not have an effect on predation, so that predators chase equally prey of any trait Z. In terms of our equations, this corresponds to a constant predation kernel, $\beta_{21}(Z,Z')=\bar{\beta}$. We can similarly define the effect of interaction by prey on a predator by β_{12}; we will suppose that predators benefit from this interaction proportionally to the prey they catch, so that $\beta_{12}(Z,Z')=c\left|\beta_{21}(Z',Z)\right|=c\bar{\beta}$, where c is a conversion constant.

Let us call $p_i(Z,T,X)$ the local distribution of phenotypes at time T, i.e., $p_i(Z,T,X)dZ$ is the probability of finding the phenotypes Z through $Z+dZ$ at time T at location X for prey ($i=1$) and predators ($i=2$). This lets us specify explicitly the interaction terms, for prey,

$$\begin{aligned}I_1\left(T,X,Z\right)=&N_1(T,X)\int dZ'\alpha^1\left(Z,Z'\right)p_1\left(Z',T,X\right)\\&+N_2(T,X)\int dZ''\beta_{21}\left(Z,Z''\right)p_2\left(Z'',T,X\right),\end{aligned}$$

and for predators,

$$\begin{aligned}I_2\left(T,X,Z\right)=&N_2(T,X)\int dZ'\alpha^2\left(Z,Z'\right)p_2\left(Z',T,X\right)\\&+N_1(T,X)\int dZ''\beta_{12}\left(Z,Z''\right)p_1\left(Z'',T,X\right).\end{aligned}$$

We further assume that the local distribution of phenotype is Gaussian, i.e.,

$$p_i\left(Z,T,X\right)=\frac{1}{\sqrt{2\pi V_i^p}}\exp\left(-\frac{1}{2V_i^p}\left(Z-\bar{Z}_i\left(T,X\right)\right)^2\right).$$

We can thus explicitly write the interaction terms as:

$$I_1=-N_1(T,X)\alpha_1\sqrt{\frac{2V_1^u}{2V_1^u+V_1^p}}\exp\left(-\frac{\left(Z-\bar{Z}_1(T,X)\right)^2}{2\left(2V_1^u+V_1^p\right)}\right)-N_2(T,X)\bar{\beta}$$

and

$$I_2=-N_2(T,X)\alpha_2\sqrt{\frac{2V_2^u}{2V_2^u+V_2^p}}\exp\left(-\frac{\left(Z-\bar{Z}_2(T,X)\right)^2}{2\left(2V_2^u+V_2^p\right)}\right)+N_1(T,X)c\bar{\beta}$$

We can further find expressions for the mean fitnesses of the populations, $\bar{w}_i(T,X)=\int dZ p_i(Z,T,X)w_i(T,X,Z)$. Assuming the previous Gaussian distributions, we find:

$$\begin{aligned}
\bar{r}_1(T,X)&=r_1^{max}-\frac{V_1^p}{2V_1^s}-\frac{1}{2V_1^s}\left(\bar{Z}_1(T,X)-\theta_1(X)\right)^2,\\
\bar{I}_1(T,X)&=-\alpha_1\sqrt{\frac{V_1^u}{V_1^u+V_1^p}}N_1(T,X)-\bar{\beta}N_2(T,X),\\
\bar{r}_2(T,X)&=-\bar{d}-\frac{V_2^p}{2V_2^s}-\frac{1}{2V_2^s}\left(\bar{Z}_2(T,X)-\theta_2(X)\right)^2,\\
\bar{I}_2(T,X)&=-\alpha_2\sqrt{\frac{V_2^u}{V_2^u+V_2^p}}N_2(T,X)+c\bar{\beta}N_1(T,X).
\end{aligned}$$

The equations above define mean fitness for both prey and predator populations, which allow us to find local dynamics for mean population growth and mean trait. These are given, respectively by $N_i(T,X)\bar{w}_i(T,X)$ and $\partial\bar{w}_i(T,X)/\partial\bar{Z}_i$. Complete

spatiotemporal dynamics are found when adding temporal and spatial operators:

$$\frac{\partial N_1}{\partial T} = \delta_1 \frac{\partial^2 N_1}{\partial X^2} + N_1 \left(r_1^{max} - \alpha_1 \sqrt{\frac{V_1^u}{V_1^u + V_1^p}} N_1 - \frac{1}{2V_1^s} \left(\theta_1(X) - \bar{Z}_1 \right)^2 - \frac{V_1^p}{2V_1^s} - \bar{\beta} N_2 \right) \tag{3.4a}$$

$$\frac{\partial N_2}{\partial T} = \delta_2 \frac{\partial^2 N_2}{\partial X^2} + N_2 \left(c\bar{\beta} N_1 - \bar{d} - \alpha_2 \sqrt{\frac{V_2^u}{V_2^u + V_2^p}} N_2 - \frac{1}{2V_2^s} \left(\theta_2(X) - \bar{Z}_2 \right)^2 - \frac{V_2^p}{2V_2^s} \right) \tag{3.4b}$$

$$\frac{\partial \bar{Z}_1}{\partial T} = \delta_1 \frac{\partial^2 \bar{Z}_1}{\partial X^2} + 2\delta_1 \frac{\partial \log N_1}{\partial X} \frac{\partial \bar{Z}_1}{\partial X} - h_1^2 \frac{V_1^p}{V_1^s} \left(\bar{Z}_1 - B_1 X \right) \tag{3.4c}$$

$$\frac{\partial \bar{Z}_2}{\partial T} = \delta_2 \frac{\partial^2 \bar{Z}_2}{\partial X^2} + 2\delta_2 \frac{\partial \log N_2}{\partial X} \frac{\partial \bar{Z}_2}{\partial X} - h_2^2 \frac{V_2^p}{V_2^s} \left(\bar{Z}_2 - B_2 X \right) \tag{3.4d}$$

This model depends on eighteen parameters, which are explained on Table 4.1. Equations (3.4a) and (3.4a) govern prey and predator population densities, respectively, and they both account for migration effects (the first term in each equation modeling random movement of individuals) and absolute fitnesses (the term including the parenthesis). Prey fitness considers a maximal growth rate which is decreased by intraspecific competition, maladaptation and predation effects proportional to predator's density. Predator fitness considers a predation benefit proportional to local prey density which is decreased due to natural death, intraspecific competition and maladaptation. Equations (3.4c) and (3.4d) model trait dynamics, and they are similar to the corresponding trait equation in a one-species model (Kirkpatrick and Barton 1997, Méndez-Vera et al. 2019). Each equation has three terms which model, from left to right, gene diffusion due to random dispersal (at different rates δ_i for prey and predator), asymmetrical gene flow, and adaptation towards the optimal local value (accounting for different prey and predator adaptation potentials).

Parameter reduction

We define the parameters: $r_1 = r_1^{max} - \frac{V_1^p}{2V_1^s}$; $\bar{K}_1 = \frac{r_1}{\alpha_1 r_1^{max}}\sqrt{\frac{V_1^u + V_1^p}{V_1^u}}$; $r_2 = c\bar{\beta}\bar{K}_1$; $\bar{K}_2 = \frac{r_2}{\alpha_2}\sqrt{\frac{V_2^p + V_2^u}{V_2^u}}$; $\beta = \frac{\bar{\beta}}{r_1}\bar{K}_2$; $d = \frac{\bar{d}}{r_2} + \frac{V_2^p}{2r_2V_2^s}$; $a_i = \frac{h_i^2 V_i^p}{r_i V_i^s}$; $\tilde{b}_i = \frac{B_i}{\sqrt{r_i V_i^s}}$, resulting in the reformulation of the equations as:

$$\frac{\partial N_1}{\partial T} = \delta_1 \frac{\partial^2 N_1}{\partial X^2} + N_1\left(r_1 - r_1\frac{N_1}{\bar{K}_1} - \frac{r_1}{2}\left(\tilde{b}_1 X - \frac{\bar{Z}_1}{\sqrt{r_1 V_1^s}}\right)^2 - r_1\beta\frac{N_2}{\bar{K}_2}\right) \tag{3.5a}$$

$$\frac{\partial N_2}{\partial T} = \delta_2 \frac{\partial^2 N_2}{\partial X^2} + N_2\left(r_2\frac{N_1}{\bar{K}_1} - r_2 d - r_2\frac{N_2}{\bar{K}_2} - \frac{r_2}{2}\left(\tilde{b}_2 X - \frac{\bar{Z}_2}{\sqrt{r_2 V_2^s}}\right)^2\right) \tag{3.5b}$$

$$\frac{\partial \bar{Z}_1}{\partial T} = \delta_1 \frac{\partial^2 \bar{Z}_1}{\partial X^2} + 2\delta_1 \frac{\partial \log N_1}{\partial X}\frac{\partial \bar{Z}_1}{\partial X} - r_1 a_1\left(\bar{Z}_1 - \sqrt{r_1 V_1^s}\tilde{b}_1 X\right) \tag{3.5c}$$

$$\frac{\partial \bar{Z}_2}{\partial T} = \delta_2 \frac{\partial^2 \bar{Z}_2}{\partial X^2} + 2\delta_2 \frac{\partial \log N_2}{\partial X}\frac{\partial \bar{Z}_2}{\partial X} - r_2 a_2\left(\bar{Z}_2 - \sqrt{r_2 V_2^s}\tilde{b}_2 X\right) \tag{3.5d}$$

We can further normalize population densities, $n_i = \frac{N_i}{\bar{K}_i}$, and trait values, $\bar{z}_i = \frac{\bar{Z}_i}{\sqrt{r_i V_i^s}}$ and choose a different measurement scale for time and space, for example $t_1 = r_1 T$ and $x_1 = \sqrt{\frac{r_1}{\delta_1}}X$ which result in:

$$\frac{\partial n_1}{\partial t_1} = \frac{\partial^2 n_1}{\partial x_1^2} + n_1\left(1 - n_1 - \frac{1}{2}\left(\tilde{b}_1\sqrt{\frac{\delta_1}{r_1}}x_1 - \bar{z}_1\right)^2 - \beta n_2\right) \tag{3.6a}$$

$$\frac{\partial n_2}{\partial t_1} = \frac{\delta_2}{\delta_1}\frac{\partial^2 n_2}{\partial x_1^2} + \frac{r_2}{r_1}n_2\left(n_1 - d - n_2 - \frac{1}{2}\left(\tilde{b}_2\sqrt{\frac{\delta_1}{r_1}}x_1 - \bar{z}_2\right)^2\right) \tag{3.6b}$$

$$\frac{\partial \bar{z}_1}{\partial t_1} = \frac{\partial^2 \bar{z}_1}{\partial x_1^2} + 2\frac{\partial \log n_1}{\partial x_1}\frac{\partial \bar{z}_1}{\partial x_1} - a_1\left(\bar{z}_1 - \tilde{b}_1\sqrt{\frac{\delta_1}{r_1}}x_1\right) \tag{3.6c}$$

$$\frac{\partial \bar{z}_2}{\partial t_1} = \frac{\delta_2}{\delta_1}\frac{\partial^2 \bar{z}_2}{\partial x_1^2} + 2\frac{\delta_2}{\delta_1}\frac{\partial \log n_2}{\partial x_1}\frac{\partial \bar{z}_2}{\partial x_1} - \frac{r_2}{r_1}a_2\left(\bar{z}_2 - \tilde{b}_2\sqrt{\frac{\delta_1}{r_1}}x_1\right) \tag{3.6d}$$

Note that defining $b_i^* = \tilde{b}_i\sqrt{\frac{\delta_i}{r_i}}$ then the parameters δ_i and r_i only appear dividing

each other, so we define $\delta = \dfrac{\delta_2}{\delta_1}$, $r = \dfrac{r_2}{r_1}$, $b_1 = b_1^*$ and $b_2 = b_2^*\sqrt{\dfrac{r}{\delta}}$ to finally obtain:

$$\frac{\partial n_1}{\partial t_1} = \frac{\partial^2 n_1}{\partial x_1^2} + n_1\left(1 - n_1 - \frac{1}{2}\left(b_1 x_1 - \bar{z}_1\right)^2 - \beta n_2\right) \tag{3.7a}$$

$$\frac{\partial n_2}{\partial t_1} = \delta\frac{\partial^2 n_2}{\partial x_1^2} + r n_2\left(n_1 - d - n_2 - \frac{1}{2}\left(b_2 x_1 - \bar{z}_2\right)^2\right) \tag{3.7b}$$

$$\frac{\partial \bar{z}_1}{\partial t_1} = \frac{\partial^2 \bar{z}_1}{\partial x_1^2} + 2\frac{\partial \log n_1}{\partial x_1}\frac{\partial \bar{z}_1}{\partial x_1} - a_1\left(\bar{z}_1 - b_1 x_1\right) \tag{3.7c}$$

$$\frac{\partial \bar{z}_2}{\partial t_1} = \delta\frac{\partial^2 \bar{z}_2}{\partial x_1^2} + 2\delta\frac{\partial \log n_2}{\partial x_1}\frac{\partial \bar{z}_2}{\partial x_1} - r a_2\left(\bar{z}_2 - b_2 x_1\right). \tag{3.7d}$$

Each of the parameters appearing in this equation is summarized in Table 3.3 with its corresponding relation to the parameters in the full model (3.4) (which are explained in Table 4.1).

3.4.2 Numerical schemes

We present here the discretization we used to approximate the propagation speed in Kikpatrick and Barton's model. Since it is already cumbersome to analyze it for the one-species model, for the two species model we only present the used scheme and describe briefly the problems we encountered.

As usual for a finite differences scheme, we consider a discretization of a finite time interval $[0, T]$ and a time step Δt, giving a time mesh $t = 0$, $t_1 = \Delta t$, etc., with the general formula $t_k = k\Delta t$, $k \geq 0$; we also consider an one-dimensional space interval $[-L, L]$ and a fixed spatial step Δx so that we have mesh points $x_\ell = -L + \ell\Delta x$, $\ell \geq 0$.

When considering an explicit time-forward scheme, we find the system

$$\frac{n_{k+1,\ell}-n_{k,\ell}}{\Delta t} = \frac{n_{k,\ell+1}-2n_{k,\ell}+n_{k,\ell-1}}{\Delta x^2} + n_{k,\ell}\left(1-n_{k,\ell}-\frac{1}{2}\left(\bar{z}_{k,\ell}-Bx_\ell\right)^2\right) \tag{3.8a}$$

$$\begin{aligned}\frac{z_{k+1,\ell}-z_{k,\ell}}{\Delta t} =& \frac{\bar{z}_{k,\ell+1}-2\bar{z}_{k,\ell}+\bar{z}_{k,\ell-1}}{\Delta x^2} - 2\frac{1}{n_{k,\ell}+\varepsilon}\frac{n_{k,\ell+1}-n_{k,\ell-1}}{2\Delta x}\frac{\bar{z}_{k,\ell+1}-\bar{z}_{k,\ell-1}}{2\Delta x} \\ & - A\left(\bar{z}_{k,\ell}-Bx_\ell\right).\end{aligned} \tag{3.8b}$$

Notice that the solution for $n_{k+1,\ell}$ in terms of the $n_{k,\cdot}$ is almost a convex combination of these terms, explicitly

$$n_{k+1,\ell} = \frac{\Delta t}{\Delta x^2}\left(n_{k,\ell+1}+n_{k,\ell-1}\right) + \left[1-\frac{2\Delta t}{\Delta x^2}+\Delta t\left(1-n_{k,\ell}-\frac{1}{2}\left(\bar{z}_{k,\ell}-Bx_\ell\right)^2\right)\right]n_{k,\ell}, \tag{3.9}$$

for it to be a (sub-)convex combination of the solution at different points of the mesh at the instant t_k, we need the coefficients to be greater than zero and for their sum to be at most 1. Supposing that any desirable solution satisfies $0 \leq n_{k,\ell} \leq 1$ for any $k, \ell \geq 0$, the first condition is verified to be true if

$$\frac{2\Delta t}{\Delta x^2} + \frac{\Delta t}{2}\left(\bar{z}_{k,\ell}-Bx_\ell\right)^2 \leq 1 \text{ for every } k,\ell \geq 0. \tag{3.10}$$

It is difficult to predict the values of $\bar{z}_{k,\ell}$ since it is ill-defined whenever $n = 0$ and simulations show instabilities when the $n_{k,\ell}$ are close to zero and the mesh is not well chosen, however, the well working cases show that at the front tip there is an almost constant distance between $\bar{z}_{k,\ell}$ and the optimal phenotype Bx_ℓ. Since in some simulations we imposed $\bar{z}_{0,\ell} = 0$ everywhere, and that locally this distance tends to decrease when $\bar{z}_{k,\ell}$ is too far from the optimum, then $|z_{k,\ell} - Bx_\ell|$ cannot be bigger than BL for a sufficiently big spatial window $[-L, L]$. We find thus that if the condition

$$\frac{2\Delta t}{\Delta x^2} + \frac{\Delta t}{2}B^2L^2 \leq 1, \text{ or equivalently, } \Delta t \leq \frac{1}{2\Delta x^{-2} + \frac{1}{2}B^2L^2} \tag{3.11}$$

is met, then the stability condition (3.10) is valid.

Notice that when Δx^{-2} is big enough compared to B^2L^2 then condition (3.11) is just the usual CFL-condition for the stability of explicit finite differences schemes for reaction-diffusion equations. However if B and L are bigger, this stability condition becomes highly restrictive. This actually made explicit schemes of this kind unpractical for our study.

We proposed our own non-linear implicit scheme for Kirkpatrick and Barton's equations given, in the same presented mesh, by the following equations:

$$\frac{n_{k+1,\ell} - n_{k,\ell}}{\Delta t} = \frac{n_{k+1,\ell+1} - 2n_{k+1,\ell} + n_{k+1,\ell-1}}{\Delta x^2} + n_{k+1,\ell}\left(1 - n_{k,\ell} - \frac{1}{2}\left(\bar{z}_{k,\ell} - Bx_\ell\right)^2\right), \tag{3.12a}$$

$$\begin{aligned}\frac{z_{k+1,\ell} - z_{k,\ell}}{\Delta t} =& \frac{\bar{z}_{k+1,\ell+1} - 2\bar{z}_{k+1,\ell} + \bar{z}_{k+1,\ell-1}}{\Delta x^2} \\ & - 2\frac{1}{n_{k+1,\ell} + \varepsilon}\frac{n_{k+1,\ell+1} - n_{k+1,\ell-1}}{2\Delta x}\frac{\bar{z}_{k+1,\ell+1} - \bar{z}_{k+1,\ell-1}}{2\Delta x} \\ & - A\left(\bar{z}_{k+1,\ell} - Bx_\ell\right).\end{aligned} \tag{3.12b}$$

Notice that, for each time point t_{k+1}, equation (3.12a) is linear on the vector $n_{k+1,\cdot}$ given that the values $n_{k,\ell}$ and $\bar{z}_{k,\ell}$ are known for each $\ell \geq 0$ and thus it may be solved by matrix inversion techniques, with coefficients depending on the solution at previous time step t_k. Once the vector $n_{k+1,\cdot}$ is known, the equation for the vector $\bar{z}_{k,\cdot}$ is just a linear one (with time-varying coefficients) that can also be solved with matrix inversion techniques.

Although we did not study the stability of the finite differences scheme (3.12), it behaved well for reasonable mesh parameters, and we were thus able to approximate the propagation speeds for a large family of (A, B) values.

Inspired by the scheme (3.12) we used the following implicit finite differences scheme to approximate the solutions of the equation system (3.1) (this time we indi-

cate the mesh variables with superindices):

$$\frac{n_1^{k+1,\ell}-n_1^{k,\ell}}{\Delta t}=\frac{n_1^{k+1,\ell+1}-2n_1^{k+1,\ell}+n_1^{k+1,\ell-1}}{\Delta x^2}+n_1^{k+1,\ell}\left(1-n_1^{k,\ell}-\frac{1}{2}\left(b_1x_\ell-\bar{z}_1^{k,\ell}\right)^2-\beta n_2^{k,\ell}\right), \tag{3.13a}$$

$$\frac{n_2^{k+1,\ell}-n_2^{k,\ell}}{\Delta t}=\delta\frac{n_2^{k+1,\ell+1}-2n_2^{k+1,\ell}+n_2^{k+1,\ell-1}}{\Delta x^2}+rn_2^{k+1,\ell}\left(n_1^{k,\ell}-d-n_2^{k,\ell}-\frac{1}{2}\left(b_2x_\ell-\bar{z}_2^{k,\ell}\right)^2\right), \tag{3.13b}$$

$$\begin{aligned}\frac{z_1^{k+1,\ell}-z_1^{k,\ell}}{\Delta t}=&\frac{\bar{z}_1^{k+1,\ell+1}-2\bar{z}_1^{k+1,\ell}+\bar{z}_1^{k+1,\ell-1}}{\Delta x^2}\\&-2\frac{1}{n_1^{k+1,\ell}+\varepsilon}\frac{n_1^{k+1,\ell+1}-n_1^{k+1,\ell-1}}{2\Delta x}\frac{\bar{z}_1^{k+1,\ell+1}-\bar{z}_1^{k+1,\ell-1}}{2\Delta x}-a_1\left(\bar{z}_1^{k+1,\ell}-b_1x_\ell\right),\end{aligned} \tag{3.13c}$$

$$\begin{aligned}\frac{z_2^{k+1,\ell}-z_2^{k,\ell}}{\Delta t}=&\delta\frac{\bar{z}_2^{k+1,\ell+1}-2\bar{z}_2^{k+1,\ell}+\bar{z}_2^{k+1,\ell-1}}{\Delta x^2}\\&-2\delta\frac{1}{n_2^{k+1,\ell}+\varepsilon}\frac{n_2^{k+1,\ell+1}-n_2^{k+1,\ell-1}}{2\Delta x}\frac{\bar{z}_2^{k+1,\ell+1}-\bar{z}_2^{k+1,\ell-1}}{2\Delta x}-ra_2\left(\bar{z}_2^{k+1,\ell}-b_2x_\ell\right).\end{aligned} \tag{3.13d}$$

As before, equations (3.13a) and (3.13b) are linear on $n_i^{k+1,\cdot}$ knowing the values of $n_i^{k,\cdot}$ and $z_i^{k,\cdot}$, and once these values are known, equations (3.13c) and (3.13d) are also linear on $\bar{z}_i^{k+1,\cdot}$ so we can use a similar algorithm to approximate the solutions of this system.

Although we did not study the stability of this system, there seems to be a condition depending strongly on the parameters δ and r. Numerical approximations for the system (3.13) were usually done using $L=200$, $\Delta x=0.1$ and $\Delta t=0.1$ or $\Delta t=0.05$ unless instabilities arose, in which case we used $\Delta t=0.01$ (notice the difference with the explicit scheme, where for $\Delta x=0.1$ a value $\Delta t=0.005$ would not be good enough even in the best case scenario, this has a great impact in computation times).

3.4.3 Proof of expression (3.3)

Because intrinsic speeds largely determine the qualitative outcome, we also looked for approximations of the intrinsic predator speed. Since the predator intrinsic speed is defined when the prey is everywhere, going back to equation (3.1) this corresponds to imposing $n_1 = 1$ everywhere (and ignoring prey's adaptation). This results in the system

$$\frac{\partial n_2}{\partial t_1} = \delta \frac{\partial^2 n_2}{\partial x_1^2} + r n_2 \left(1 - d - n_2 - \frac{1}{2}\left(b_2 x_1 - \bar{z}_2\right)^2\right) \tag{3.14a}$$

$$\frac{\partial \bar{z}_2}{\partial t_1} = \delta \frac{\partial^2 \bar{z}_2}{\partial x_1^2} + 2\delta \frac{\partial \log n_2}{\partial x_1} \frac{\partial \bar{z}_2}{\partial x_1} - r a_2 \left(\bar{z}_2 - b_2 x_1\right). \tag{3.14b}$$

which is equivalent to a one-species model after changes of variables, namely:

$$t_* = r(1-d)t_1, \quad x_* = \sqrt{\frac{r(1-d)}{\delta}} x_1, \quad \bar{n}_2 = \frac{n_2}{1-d}, \quad \bar{z}_2 = \frac{z_2}{\sqrt{1-d}}, \tag{3.15}$$

with parameters $A = \dfrac{a_2}{\sqrt{1-d}}$ and $B = \dfrac{b_2}{1-d}\sqrt{\dfrac{\delta}{r}}$. Changing variables back we find the formula (3.3) for the approximation of intrinsic predator speed.

Parameter	Description	Units of measure
T	*Time variable measured in an appropriate scale (weeks, years, etc.) to observe changes in population densities and trait values.	$[T]$
X	*Space coordinate along a one-dimensional cline (longitudinal, altitudinal, etc.). The measurement scale should allow for changes in trait values (meters, kilometers, etc.).	$[X]$
N_i	*Population density. $N_i(T, X)$ is the number of individuals in location X at time T.	$[N_i]$
$\bar{Z}_i$	*Mean phenotypic trait. Its value at location X at time T is $\bar{Z}_i = \bar{Z}_i(T, X)$. Scale of measurement makes it normally distributed (locally).	$[Z_i]$
δ_i	A measure of population diffusion, typically half the variance between consecutive breeding or nesting sites.	$[X^2T^{-1}]$
r_1^{max}	Maximal prey growth rate, i.e, in absence of competition and maladaptation.	$[T^{-1}]$
α_i	Competition rate, measured as the penalization in fitness per individual.	$\left[N_i^{-1}T^{-1}\right]$
V_i^u	Variance of the resource utilization functions, assumed to be Gaussian, as in the McArthur-Levins model (Macarthur and Levins 1967).	$[Z_i^2]$
V_i^p	Variance of the phenotypic trait, assumed to be constant across space and time.	$[Z_i^2]$
V_i^s	Stabilization variance. A measure of stabilizing selection.	$[Z_i^2T]$
$\bar{\beta}$	Predation rate, measured as the decrease in fitness per individual per predator.	$\left[\frac{1}{N_2T}\right]$
c	Conversion rate from captured prey mass to predator mass.	$\left[\frac{N_2}{N_1}\right]$
$\bar{d}$	Predator death rate. Natural predator death rate when perfectly adapted and in the absence of prey and competitors.	$[T^{-1}]$
h_i^2	Heritability of the considered phenotypic trait.	$[1]$
B_i	Cline slope. Rate of change of the optimal phenotypic value across space.	$[Z_iX^{-1}]$

Table 3.2: Description and measure units of each of the variables (marked with a *) and parameters used in the model before parameter simplification. The parameters are assumed to be constant. Units of measurement are shown in square brackets. Variables and parameters with the subindex i correspond to prey ($i = 1$) or predators ($i = 2$).

Parameter	Description	Equivalence
r_1	Net prey growth rate.	$r_1 = r_1^{max} - \frac{V_1^p}{2V_1^s}$
$\bar{K}_1$	Effective prey carrying capacity.	$\bar{K}_1 = \frac{r_1}{\alpha r_1^{max}}\sqrt{\frac{V_1^u + V_1^p}{V_1^u}}$
r_2	Maximal predator growth rate.	$r_2 = c\bar{\beta}\bar{K}_1$
$\bar{K}_2$	Predator carrying capacity.	$\bar{K}_2 = \frac{r_2}{\alpha_2}\sqrt{\frac{V_2^p + V_2^u}{V_2^u}}$
t_1	*Non-dimensionalized time coordinate, in prey growth rate scale.	$t_1 = r_1 T$
x_1	*Non-dimensionalized space coordinates, measured in prey diffusion scale.	$x_1 = \sqrt{\frac{r_1}{\delta_1}}X$
n_i	*Non-dimensionalized population density.	$n_i = \frac{N_i}{\bar{K}_i}$
$\bar{z}_i$	*Non-dimensionalized trait value.	$\bar{z}_i = \frac{\bar{Z}_i}{\sqrt{r_i V_i^s}}$
δ	Predator diffusion relative to that of its prey.	$\delta = \frac{\delta_2}{\delta_1}$
r	Predator basic growth rate relative to that of its prey.	$r = \frac{r_2}{r_1}$
β	Predation rate in the non-dimensional system.	$\beta = \frac{\bar{\beta}}{r_1}\bar{K}_2$
d	Compound death rate of the predators measured in the timescale given by t_1.	$d = \frac{\bar{d}}{r_2} + \frac{V_2^p}{2r_2 V_2^s}$
a_i	Adaptation potential	$a_i = \frac{h_i^2 V_i^p}{r_i V_i^s}$
b_i	Cline slope	$b_i = \frac{B_i}{\sqrt{r_i V_i^s}}\sqrt{\frac{\delta_1}{r_1}}$

Table 3.3: Extended description of the non-dimensional variables (marked with a *) and parameters in Table 4.1 and their relations with the parameters in the full model. The first part of the table defines auxiliary variables which are used in the equivalent expressions for the final variables made explicit in the second part of the table.Variables and parameters with the subindex i describe twofold quantities: $i = 1$ for prey and $i = 2$ for predators.

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CHAPTER IV

Retracting fronts in pathogen-aided invasions

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4.1 Introduction

In this chapter we study a move to a complex interaction network where the important interspecific interaction is apparent competition (Holt 1977). We have thus three interacting species: two competitors and a common pathogen or predator. This study is largely inspired in the case of the invasive ladybird *Harmonia axyridis* which has been able to outcompete indigenous ladybird species in several countries. According to evidence, its success is due to a parasite they carry that does not affect them but is lethal to native species (Vilcinskas et al. 2013). A similar phenomenon was observed in the invasion of the gray squirrel (*Sciurus carolinensis*) in Great Britain, which has displaced the native red squirrel (*Sciurus vulgaris*) populations in a lot of places. Initially it was thought that the principal mechanisms causing the extinction of local red squirrel populations was exploitative competition, but further evidence showed that gray squirrels carried a virus having no effect on them but being lethal to red squirrels (Darby et al. 2014).

We wish to study more closely the link between predation and the invasion capabilities of a general foreign species. In other words, we aim to understand under

which circumstances and why it is advantageous for an invasive species to carry its pathogens (or alternatively its predators) to a new environment, and if so through which mechanism. We posit that after a considerable time under predation pressure local populations should be able to become resistant to pathogens whenever possible, so that we will also consider a trait accounting for resistance to predators. Its dynamics follow standard quantitative genetic laws. We will impose an additional hypothesis in order to observe interesting dynamics, namely that the native species is competitively stronger than the foreign species in the absence of the additional competition strength due to the influence of the pathogen.

4.2 Model presentation

In this section we will present in detail a framework to study the proposed scenario. We consider thus a local population of a certain species under intraspecific competition established along one-dimensional space at its carrying capacity, and we consider a foreign population to be a possible invader, which will initially appear in a localized, small area together with one of its natural pathogens. Although in the following we will consistently speak about pathogens, the model to be presented is also useful to speak about predators since it only quantifies the different interactions and the specific details of the underlying mechanism affecting population densities are not important.

The local population will suffer from intra- and interspecific competition with the foreign species and from predation as in a typical Lotka-Volterra like model. Predation is only partially suffered, where the proportion between effective predation and suffered predation is given by a ratio $(1-Z)^2$, where the quantity Z is a measure of adaptation. This quantity, thus, will evolve following standard population genetic dynamics.

4.2.1 Eco-evolutionary dynamics in time and space

We consider a local population whose density at time T and location X is given by $N_1(T, X)$, which is going to be affected by an invasive population and a predator whose densities are $N_2(T, X)$ and $P(T, X)$, respectively. The susceptibility of the first species to the predator is taken into account through an evolving trait Z, whose value at (T, X) is given by $Z(T, X)$. We will take $Z = 0$ to mean that the species is completely susceptible to the predator suffering from a maximal predation rate, while $Z = 1$ means the local population does not suffer from predation (thus Z can be thought of as the degree of immunity of the local population).

The equations governing the dynamics of this system are then

$$\frac{\partial N_1}{\partial T} = \delta_1 \frac{\partial^2 N_1}{\partial X^2} + N_1 \left(r_1 - a_{11} N_1 - a_{12} N_2 - \left(B_0 + B_1 (1 - Z)^2 \right) P - K Z^2 \right) \tag{4.1a}$$

$$\frac{\partial N_2}{\partial T} = \delta_2 \frac{\partial^2 N_2}{\partial X^2} + N_2 \left(r_2 - a_{21} N_1 - a_{22} N_2 - B_0 P \right) \tag{4.1b}$$

$$\frac{\partial P}{\partial T} = \delta_3 \frac{\partial^2 P}{\partial X^2} + P \left(\gamma_1 \left(B_0 + B_1 (1 - Z)^2 \right) N_1 + \gamma_2 B_0 N_2 - a_{33} P - d \right) \tag{4.1c}$$

$$\frac{\partial Z}{\partial T} = \delta_1 \frac{\partial^2 Z}{\partial X^2} + 2\delta_1 \frac{\partial \ln N_1}{\partial X} \frac{\partial Z}{\partial X} + r_1 h^2 \left(2 B_1 P (1 - Z) - 2 K Z \right) \tag{4.1d}$$

with parameters explained in Table 4.1. Equations (4.1a) through (4.1c) model the ecological dynamics of the system, meaning the changes in densities due to intra- and interspecific interactions, taking into account the effects of the resistance trait Z. Equation (4.1d) models the changes in the trait Z due to migration and selective pressure.

As it is usual, the model can be non-dimensionalized to reduce the number of

Parameter	Description
δ_i'	Diffusion rate of the i-th species ($i = 1, 2, 3$).
r_i	Maximal reproduction rate of the i-th species ($i = 1, 2$).
a_{ii}	Intraspecific competition rate ($i = 1, 2, 3$).
a_{ij}	Interspecific competition rate.
B_0	Predation rate towards species 1 and 2.
B_1	Additional cost of predation suffered by species 1.
γ_i	Conversion rate of predated species i.
d	Natural predator death rate.
K	Cost of developing resistance to the additional predation cost.
h^2	Trait's heritability.

Table 4.1: Description of the dimensional parameters.

intervening parameters, yielding the following equation system:

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \alpha_{12} n_2 - \beta p\left(1 + \bar{B}(1-z)^2\right) - \kappa z^2\right) \tag{4.2a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\partial^2 n_2}{\partial x^2} + r n_2\left(1 - n_2 - \alpha_{21} n_1 - \beta' p\right) \tag{4.2b}$$

$$\frac{\partial p}{\partial t} = \delta\frac{\partial^2 p}{\partial x^2} + Rp\left(n_2 - p + \gamma\left(1 + \bar{B}(1-z)^2\right) n_1 - \bar{d}\right) \tag{4.2c}$$

$$\frac{\partial z}{\partial t} = \frac{\partial^2 z}{\partial x^2} + 2\frac{\partial \ln n_1}{\partial x}\frac{\partial z}{\partial x} - A\left(\beta\bar{B}p(z-1) + \kappa z\right) \tag{4.2d}$$

with the parameters explained in Table 4.2. We also assume that the species under competition have the same diffusion rate.

We remind an useful result for the general competition equation with dispersal (see Volpert and Petrovskii 2009):

$$\frac{\partial n_1}{\partial t} = D_1\frac{\partial^2 n_1}{\partial x^2} + r_1 n_1\left(1 - a_{11} n_1 - a_{12} n_2\right) \tag{4.3a}$$

$$\frac{\partial n_2}{\partial t} = D_2\frac{\partial^2 n_2}{\partial x^2} + r_2 n_2\left(1 - a_{22} n_2 - a_{21} n_1\right) \tag{4.3b}$$

- When $a_{11} < a_{21}$ and $a_{22} > a_{12}$ the local species is resistant to invasion by n_2.
- When $a_{11} > a_{21}$ and $a_{22} > a_{12}$ then the foreign species, n_2, can invade without

making the local species go extinct, i.e., they coexist at equilibrium.

- When $a_{11} < a_{21}$ and $a_{22} < a_{12}$ then the foreign species may invade if initial population density and support are larger than a critical quantity. If invasion is successful, the local species is completely replaced.

- When $a_{11} > a_{21}$ and $a_{22} < a_{12}$ the foreign species is then invasive and completely replaces the local one.

Parameter	**Description**	**Formula**
n_1	Normalized local population density.	$n_1 = r_1^{-1} a_{11} N_1$
n_2	Normalized invasive population density.	$n_2 = r_2^{-1} a_{22} N_2$
p	Normalized predator population density.	$p = \frac{a_{22} a_{33}}{\gamma_2 B_0 r_2}$
δ	Non-dimensional predator diffusion rate.	$\delta = \frac{\delta_3}{\delta_1}$
α_{ij}	Non-dimensional intraspecific competition rate.	$\alpha_{ij} = \frac{r_j^{-1} a_{ij}}{r_i^{-1} a_{ii}}$
r	Invasive species relative maximal reproduction rate.	$r = \frac{r_2}{r_1}$
R	Predator relative maximal reproduction rate.	$R = \frac{\gamma_2 B_0 r_2}{r_1 a_{22}}$
γ	Conversion efficiency of the local species relative to that of the foreign one.	$\gamma = \frac{\gamma_1 r_1}{\gamma_2 r_2} \frac{a_{22}}{a_{11}}$
$\bar{d}$	Natural adjusted predator death rate.	$\bar{d} = \frac{d a_{22}}{\gamma_2 B_0 r_2}$
κ	Per generation cost of resistance.	$\kappa = r_1^{-1} K$
β	Non-dimensional predation rate towards local population.	$\beta = \frac{B_0}{r_1} \frac{\gamma_2 B_0 r_2}{a_{22} a_{33}}$
β'	Non-dimensional predation rate towards invasive population.	$\beta' = \frac{\beta}{r}$
$\bar{B}$	Extra cost of predation relative to base predation rate.	$\bar{B} = \frac{B_1}{B_0}$
A	Adaptation potential.	$A = 2 r_1 h^2$

Table 4.2: Description of the non-dimensional parameters and variables.

4.2.2 Ecological considerations and constraints

We want our model to be able to capture and explain the phenomena presented in the introduction, which can be summarized as conditions on the parameters of the equation system (4.2).

1. The native and the foreign species should occupy similar niches so as to be strong competitors, but since the native one should be better adapted to the environment, so in absence of predators the native species is resistant to invasion. This is summarized as the condition

$$\alpha_{12} < 1 \text{ and } \alpha_{21} > 1. \tag{4.4}$$

2. In the presence of predators, and being completely susceptible to them, the native population should lose competition strength due to apparent competition and become non resistant to invasion.

   Suppose that R is big enough that predators reach their equilibrium density $p = n_2 + \gamma(1 + \bar{B}(1-z)^2)n_1 - \bar{d}$ fast. Without adaptation to predators ($z = 0$) the equations thus read:

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 + (1+\bar{B})\beta\bar{d} - \left(1 + (1+\bar{B})^2\beta\right)n_1 - \left(\alpha_{12} + (1+\bar{B})\beta\right)n_2\right) \tag{4.5a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\partial^2 n_2}{\partial x^2} + rn_2\left(1 + \beta'\bar{d} - (\alpha_{21} + \beta'\gamma(1+\bar{B}))n_1 - (1+\beta')n_2\right) \tag{4.5b}$$

   The foreign species will establish itself in the new environment if:

$$\frac{1 + (1+\bar{B})^2\beta}{1 + (1+\bar{B})\beta\bar{d}} > \frac{\alpha_{21} + \beta'\gamma(1+\bar{B})}{1 + \beta'\bar{d}} \tag{4.6}$$

And given this condition, the local species will be replaced if

$$\frac{1+\beta'}{1+\beta'\bar{d}} < \frac{\alpha_{12}+(1+\bar{B})\beta}{1+(1+\bar{B})\beta\bar{d}} \tag{4.7}$$

We may reorder this conditions to obtain, equivalently:

$$0 < \alpha_{21} - 1 < \beta(1+\bar{B})\left(\gamma(1+\bar{B}) - \alpha_{21}\bar{d}\right) - \beta'\left(\gamma(1+\bar{B}) - \bar{d}\right) \tag{4.8a}$$

$$0 < 1 - \alpha_{12} < \beta(1+\bar{B})\left(1-\bar{d}\right) - \beta'\left(1-\alpha_{12}\bar{d}\right) \tag{4.8b}$$

3. Perfect resistance: if the local species were perfectly resistant to pathogens (i.e., the effect on the local species is the same as on the foreign species, thus $z = 1$ everywhere) so that they are not affected by the pathogens, supposing additionally that $p = n_2 + \gamma n_1 - \bar{d}$, the equations become

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1+\beta\bar{d} - \kappa - (1+\beta\gamma)\,n_1 - (\alpha_{12}+\beta)\,n_2\right) \tag{4.9a}$$

$$\frac{\partial n_2}{\partial t} = \frac{\partial^2 n_2}{\partial x^2} + r n_2\left(1+\beta'\bar{d} - (\alpha_{21}+\beta'\gamma)\,n_1 - (1+\beta')\,n_2\right) \tag{4.9b}$$

In this scenario, the local population is resistant to invasion by n_2 if

$$\frac{1+\beta\gamma}{1+\beta\bar{d}-\kappa} < \frac{\alpha_{21}+\beta'\gamma}{1+\beta'\bar{d}} \qquad \text{and} \qquad \frac{\alpha_{12}+\beta}{1+\beta\bar{d}-\kappa} < \frac{1+\beta'}{1+\beta'\bar{d}}, \tag{4.10}$$

or equivalently

$$\alpha_{21} > \frac{(1+\beta\gamma)\left(1+\beta'\bar{d}\right)}{1+\beta\bar{d}-\kappa} - \beta'\gamma = 1 + \beta'(\bar{d}-\gamma) + \frac{\left(\beta(\gamma-\bar{d})+\kappa\right)\left(1+\beta'\bar{d}\right)}{1+\beta\bar{d}-\kappa} \tag{4.11a}$$

$$\alpha_{12} < \frac{(1+\beta')\left(1+\beta\bar{d}-\kappa\right)}{1+\beta'\bar{d}} - \beta = 1 - \beta + \frac{\beta'\left(1-\bar{d}\right)\left(1+\beta\bar{d}-\kappa\right)}{1+\beta'\bar{d}}. \tag{4.11b}$$

Notice that conditions (4.8a) and (4.11a) both impose constraints on α_{12}, α_{21} and β, revealing a relation between predation and competition rates so that the proposed scenario is valid. For a fixed value of β conditions (4.8a) give an upper bound on α_{21} and a lower bound on α_{12} (given that $\beta'\bar{d} < 1$, or $d < a_{33}\dfrac{r_2}{B_0}$ in the dimensional variables), and conditions (4.11a) give a lower bound on α_{21} and an upper bound on α_{12}. In other words a given predation level determines an interval for possible values of competition.

4.2.3 Evolutionary approximations ignoring spatial structure

Considering the non-spatial system (i.e., system (4.2) ignoring the spatial variable), the equation for the evolving trait is given by

$$\frac{dz}{dt} = A\left(\bar{B}\beta p(1-z) - \kappa z\right). \tag{4.12}$$

From this expression, we see that the optimal trait value is given by

$$z^* = \frac{\bar{B}\beta p}{\bar{B}\beta p + \kappa}. \tag{4.13}$$

Notice that this expression depends on the density of pathogens, since the selection strength depends on the incurred predation loss, which is proportional to pathogen concentration; this loss is $\bar{B}\beta p$. The optimal trait is not exactly $z = 1$, as there is a cost κ to perfect resistance.

For general $p = p(t)$ the solution to equation (4.12) is given by

$$\begin{aligned} z(t) =& z(0)\exp\left(-A\int_0^t \left(\bar{B}\beta p(s) + \kappa\right) ds\right) \\ &+ A\bar{B}\beta\int_0^t p(s_1)\exp\left(-A\int_{s_1}^t \left(\bar{B}\beta p(s_2) + \kappa\right) ds_2\right) ds_1 \end{aligned}$$

However, assuming that p is approximately constant, this expression reduces to:

$$z(t) = z(0)\exp\left(-A\left(\bar{B}\beta p + \kappa\right)t\right) + \frac{\bar{B}\beta p}{\bar{B}\beta p + \kappa}\left(1 - \exp\left(-A\left(\bar{B}\beta p + \kappa\right)t\right)\right) \quad (4.14a)$$

$$= \frac{\bar{B}\beta p}{\bar{B}\beta p + \kappa} + \left(z(0) - \frac{\bar{B}\beta p}{\bar{B}\beta p + \kappa}\right)\exp\left(-A\left(\bar{B}\beta p + \kappa\right)t\right), \quad (4.14b)$$

which shows that, without spatial structure, the local population adapts exponentially fast.

Suppose we want to know the time it takes the population for its trait to grow near the equilibrium value $\bar{B}\beta p/(\bar{B}\beta p + \kappa)$ only differing by a relative amount $0 < \eta < 1$, i.e., the time t_η such that

$$z\left(t_\eta\right) = (1-\eta)z^* = \frac{\bar{B}\beta p}{\bar{B}\beta p + \kappa}(1-\eta).$$

Equation (4.14b) lets us compute this time exactly, as:

$$t_\eta = -\frac{\ln \eta}{A(\bar{B}\beta p + \kappa)} \quad (4.15)$$

(A useful approximation can be derived by noting that $e^{-3} \approx 0.05$, so that $t_{95\%} \approx 3/A(\bar{B}\beta p + \kappa)$).

The expression (4.15) is decreasing in A, $\bar{B}\beta p$ and κ. It is in fact inversely proportional to A, meaning that doubling the trait's heritability will reduce the time needed to see the same level of adaptation η in half. It is also not surprising that it is decreasing in $\bar{B}\beta p$ since the presence of predators induce selection, and the bigger the attack rate $\bar{B}\beta$ the stronger this selection will be.

The dependence on the cost of making defenses κ is perhaps less intuitive, but this has to do with the fact that the optimum depends on κ. This dependence on κ measures a counter-selection due to the fact that excessive defenses are harmful to the population. It is also a way to account for the fact that if predation pressures

suddenly disappeared, the optimal trait is $z^* = 0$, so selection should drive the value of the mean trait towards this optimum.

4.2.3.1 Model considerations

Suppose $z(0)$ is a fraction η_0 away from the optimum, and let us measure $z(t)$ in the same way, i.e. $z(t) = (1 - \eta(t))z^*$, then equation (4.14b) can be rewritten as

$$\frac{\eta(t)}{\eta_0} = \exp\left(-A\left(\bar{B}\beta p + \kappa\right)t\right) \tag{4.16}$$

This expression is valid for any initial maladaptation amount η_0. So that the time we should wait for the population's trait to be half closer to the optimum, i.e. $t_{1/2}$ s.t. $\eta\left(t_{1/2}\right) = \eta_0/2$, according to this expression, is given by

$$t_{1/2} = \frac{\ln 2}{A(\bar{B}\beta p + \kappa)}. \tag{4.17}$$

This means that regardless of the initial relative maladaptation η_0 we should always wait the same half-time $t_{1/2}$ to be as close to the optimum as we are far from the initial condition, i.e., for the trait to be mid-way from the optimum value. This is probably not realistic, as it may be increasingly more difficult to build more defenses the closer the trait is to the optimum.

In other words, this particular property for the adaptation time in the non-spatial system is due to the way we chose to model predator-resistance and adaptation, but we should expect its qualitative behavior to remain similar (e.g., adaptation time decreasing in A, $\bar{B}\beta p$, κ) if we had chosen other reasonable models.

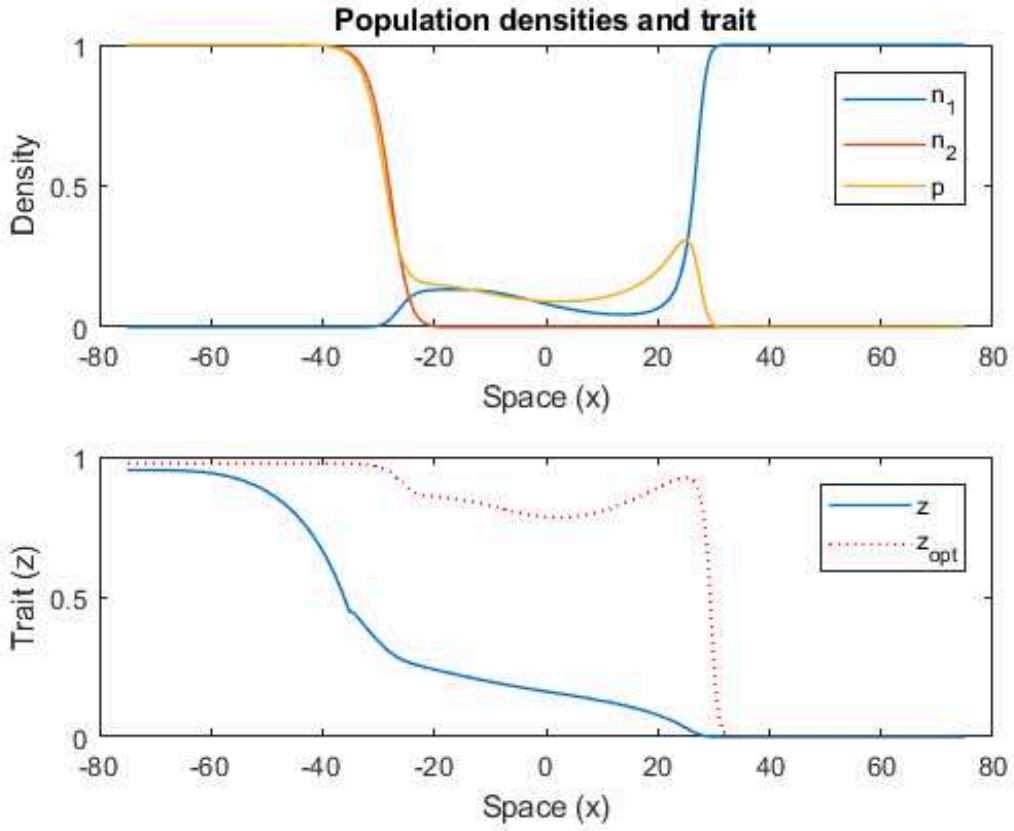

Figure 4.1: Example of a solution for an "intermediate" time (in the previous model). Parameter values: $h^2 = 0.01$, $\kappa = 1/2$, $\beta = 20$, $\alpha_{12} = 1/2$, $\alpha_{21} = 3/2$, $r = 1$, $R = 4$, $c = 5$, $\delta_2 = 1$, $\delta_3 = 1$.

4.3 Adaptation in the spatial model

The purpose of the initial spatial model (4.2) is to understand under which conditions the native population n_1 is able to stop invasion and to what extent resistance to predation may help the population in doing so. Although adaptation in the non-spatial model can give us clues as to how much time we should expect populations in the spatial model to become locally adapted, we should not lose sight of the objective to study conditions for the invasion to be stopped.

Figure 4.1 shows an example of an expected solution for an intermediate time value (i.e. we do not recognize the initial condition, but the solution is far from

equilibrium). By looking at the population densities we can distinguish two phase transitions, corresponding to the predator front and to the invading population front.

Since local population is resistant to invasion when well-adapted to the new predators (i.e. when z is close to 1), we will see the invader speed to be positive, $c_i > 0$ until local populations becomes well adapted due to pressure from the predators (which is what is taking place between the two front tips) and then this invasion front will be reverted, $c_i < 0$. It is interesting to study the moment this will happen and what proportion of the space will be affected by this invasion. Thus, this speed is variable in time, $c_i = c_i(t)$, and it may depend on all of the model parameters. Two interesting quantities are the time t_{stop} needed for the speed to be reversed, i.e. the first moment when $c_i(t_{\text{stop}}) \leq 0$, and x_{stop}, the invasion distance before speed reversal. They are related through the expression

$$x_{\text{stop}} = \int_0^{t_{\text{stop}}} c_i(s)ds. \tag{4.18}$$

Because the native population can only stop invasion when well-adapted, it is interesting to determine if there is a relationship between $t_{1-\eta}$ as given by (4.15) and this time t_{stop}: how do they compare to each other?

Let us also denote by t_p the first time the predators arrive at location x_{stop}. It is only from that moment on that the native population begins to adapt. Suppose it takes them a time t_* to build the necessary defenses, then it is true that

$$t_{\text{stop}} = t_p + t_*. \tag{4.19}$$

since at x_* the local population only begins to evolve once the predators have arrived (at time t_p), then it evolves until it becomes resistant (taking a time t_*), which is the same time it should take for it to stop invasion (i.e., t_{stop}).

We can relate these temporal measures to spatial ones through invasion speeds. Let us denote the predator invasion speed by c_p, then:

$$x_{\text{stop}} = \int_0^{t_{\text{stop}}} c_i(t)dt, \qquad \text{and} \qquad x_{\text{stop}} = \int_0^{t_p} c_p(t)dt. \tag{4.20}$$

We can approximate the front speeds c_i and c_p to obtain useful expressions for the associated times and displacements. Notice that:

1. In front of the predator front, the local population has not undergone selection, so that $z = 0$. We can then approximate the speed c_p as that corresponding to the equation system (4.2) when $z = 0$ and $n_2 = 0$. This approximation of the speed is given by

$$\tilde{c}_p = 2\sqrt{\delta R\left(\gamma\left(1+\bar{B}\right)-\bar{d}\right)} \tag{4.21}$$

2. In front of the invaders front, the local population and the predator population are interacting, but supposing that adaptation does not take place so fast and that predators reproduce fast enough so that they quickly achieve equilibrium density (or, in mathematical terms, R is large enough), we can approximate native and predator population densities as being at equilibrium and $z = 0$. Again we may use equation (4.2) under these conditions to approximate v_i as the front speed of n_2. The approximation of this speed is given by

$$\tilde{c}_i = 2\sqrt{r+\beta\bar{d}-\left(r\alpha_{21}+\beta\Gamma\right)\frac{1+\Gamma\dfrac{\beta}{\gamma}\bar{d}}{1+\Gamma^2\dfrac{\beta}{\gamma}}}. \tag{4.22}$$

   Notice that condition (4.6) ensures that this speed is always well defined.

Notice that condition 1 gives the actual traveling speed for the predator population, since it is valid in the front tip. Condition 2, however, will give only a

crude approximation, because in the zone between fronts native population is adapting to predation, which increases population density and, thus, competition strength towards the invading population, making the speed c_i to be decreasing in time. Condition 2 will then give an overestimation of the actual invasion speed.

Approximating these two speed by adequately chosen constants, equation (4.19) then yields

$$\frac{x_{\text{stop}}}{c_i} = \frac{x_{\text{stop}}}{c_p} + t_*, \text{ or equivalently, } x_{\text{stop}} = t_* \left(\frac{1}{c_i} - \frac{1}{c_p} \right)^{-1}. \tag{4.23}$$

This last expression lets us relate observable variables to the model predictions. Knowing that $x_{\text{stop}} = t_p c_p$ we can, for example, relate the time of predator arrival to the time until adaptation, through the approximations of invasion speeds:

$$t_p = \frac{t_*}{\frac{c_p}{c_i} - 1}; \tag{4.24}$$

or the time until invasion is stopped:

$$t_i = t_p + t_* = t_* \left(\frac{1}{\frac{c_p}{c_i} - 1} + 1 \right) = \frac{c_p}{c_p - c_i} t_*. \tag{4.25}$$

The question of how to approximate t_* remains. Equation (4.14b) (or equivalently, (4.16)) let us approximate the time needed for the trait to attain a given value $z(t)$ starting from $z(0)$. We need to clarify what an "adapted trait" is in order to derive an useful approximation. As before, we can fix a relative distance from the optimum, $0 < \eta < 1$, say $\eta = 5\%$, and measure t_* as the time needed to achieve this distance, i.e., $t_* = t_{1-\eta}$ as given by formula (4.15). Other possible definitions of what a "time until adaptation" should be are presented in the appendix (not yet corrected for the new model).

Observation: Here we have described the "adaptation dynamics" in terms of eco-evolutionary times given by different aspects of the dynamics (4.2), but we can transform these times into spatial measures by knowing the respective advancement speeds. This may be more useful were we to study a real adaptation scenario: it is more or less easy to measure distances between front tips (corresponding to distribution limits), but sometimes it is not easy to determine where and when an invasion began taking place, as to approximate the ongoing times from invasion. We will continue to analyze the dynamics of the model (4.2) in terms of these temporal measures, but keeping in mind that they can be translated into spatial ones.

4.4 Results

Here we present some preliminary results for a previous version of the model. While te equations differ in some parameters, the qualitative behavior of both models are similar, so we expect to find similar results for the corrected version of the model (i.e., the version presented in the previous version).

More specifically, all of the presented results are valid for the uncorrected version of the pathogen-aided invasion model, which is given by (the meaning of the parameters is similar to those presented in the main text, with a similar notation):

$$\frac{\partial n_1}{\partial t} = \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \alpha_{12}n_2 - \frac{1}{2}\beta p(1-z)^2 - \frac{1}{2}\kappa z^2\right) \tag{4.26a}$$

$$\frac{\partial n_2}{\partial t} = \delta_2\frac{\partial^2 n_2}{\partial x^2} + r n_2\left(1 - n_2 - \alpha_{21}n_1 - \beta' p\right) \tag{4.26b}$$

$$\frac{\partial p}{\partial t} = \delta_3\frac{\partial^2 p}{\partial x^2} + Rp\left(n_2 - p + cR^{-1}n_1\right) \tag{4.26c}$$

$$\frac{\partial z}{\partial t} = \frac{\partial^2 z}{\partial x^2} + 2\frac{\partial \ln n_1}{\partial x}\frac{\partial z}{\partial x} + h^2\left(\beta p(1-z) - \kappa z\right). \tag{4.26d}$$

All of the analyses made in the previous sections are still valid for this model, although the explicit formulae differ.

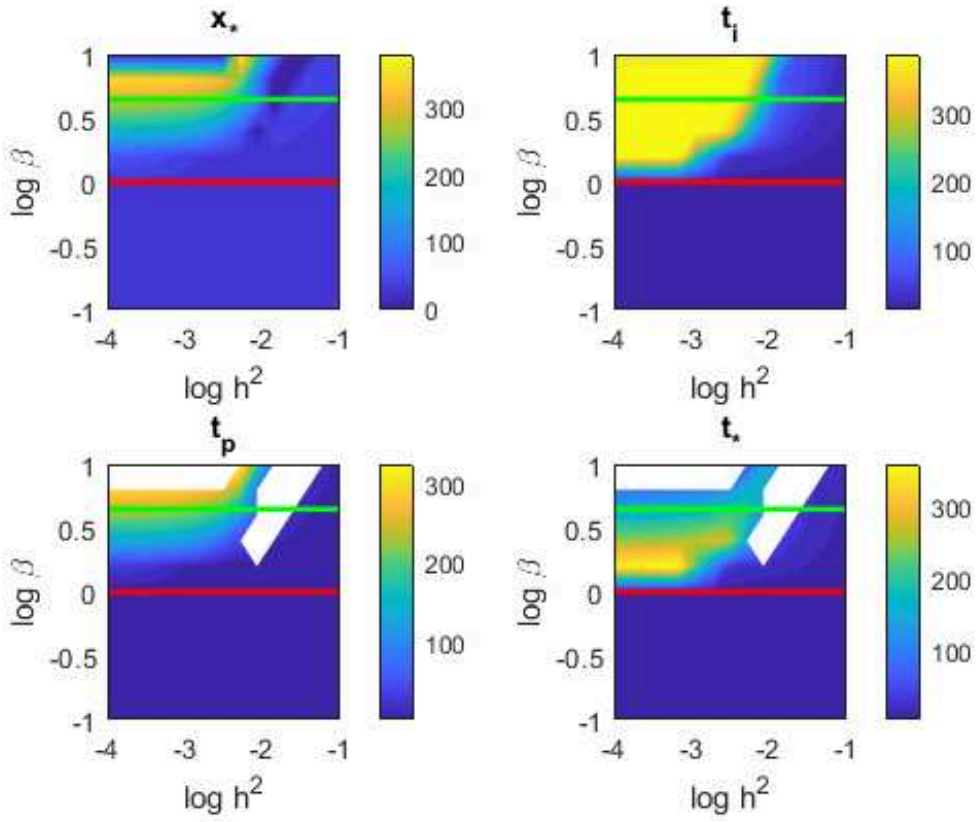

Figure 4.2: Simulation results for a set of parameters, varying β and h^2. The graphs indicate the values of four interesting variables describing important aspects of the dynamics as a function of the heritability h^2 and predation rate β.

Before introducing the results, note that condition 2 in the "Ecological considerations and constraints" section imposes two restrictions on β (taking $\beta' = 0$), so that β must be bigger than the two critical values β_1^* and β_2^* for our predictions to make sense. These critical values are marked with a green and a red line, respectively, in figures 4.2 and 4.3 which summarize our preliminary results.

We studied the dependence of the four interesting quantities x_{stop}, t_{stop}, t_p and t_* for two different set of parameters. We detail the results of each set separately.

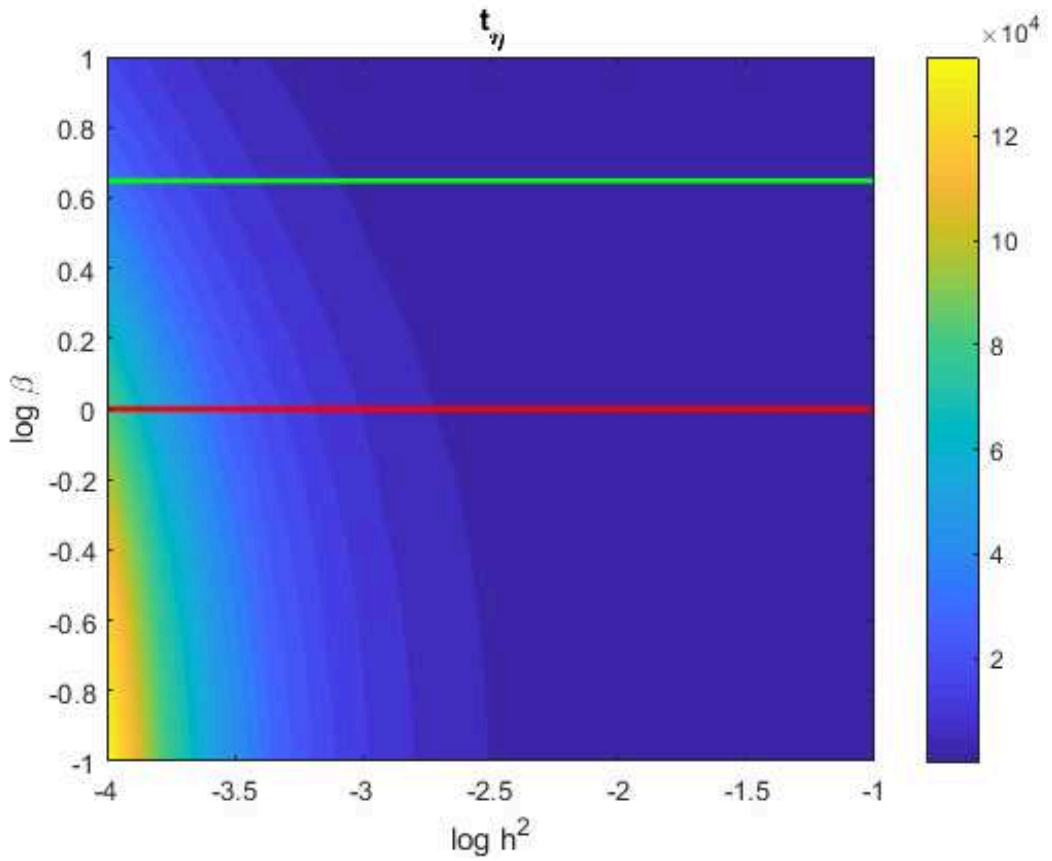

Figure 4.3: Estimated values for t_η for the chosen parameter values, indicating the critical values of β. This should be a theoretical approximation for t_*.

4.4.1 First set of parameters

Figure 4.2 summarize the values of four interesting quantities for a first set of variables, as found through a simulation of equation system (4.26a). Notice that when β is simultaneously under the two critical values (i.e. under the these two lines, the local population is always resistant to invasion, so that it does not make sense to analyze t_* for β under the smallest of these critical values.

Notice that above the green line the values of t_* are decreasing with β which is as expected, although they do not coincide well with t_η (Figure 4.3).

There are two zones where data for t_p (and thus t_*) is missing. This is because at the upper left corner adaptation h^2 is too little to see a front reversal in the total

simulation time.

At the right, the missing data in the blank zone is due to an unexpected phenomenon. Closer look upon the simulation show that while there is a propagating front of foreign species with a positive speed, it simultaneously decreases in magnitude until the foreign species becomes extinct. This may be due to high values of h^2 causing the trait z to grow so fast towards the optimum that, combined with diffusion, the contribution of the term βz^2 becomes negligible very fast. Thus, there is no front reversal and the quantity t_p becomes undefined.

4.4.2 Second set of parameters

Figure 4.4 shows the four interesting quantities x_{stop}, t_{stop}, t_p and t_* for another set of parameters, and Figure 4.5 shows the estimated adaptation time t_η for the same parameters. This time we took values of β above the critical values mentioned in the model presentation, so that we expect t_* to be decreasing as a function of β in the totality of the explored cases.

The blank spaces in figure 4.4 for the quantity t_p are due to another unexpected behavior. A close look of the simulations (an example is shown in Figure 4.6) revealed that, in this zone, there is a positive density of invaders n_2 only in the zone where there is important gene flow for the local population (i.e., the zone with predators where the local population is still maladapted) and it travels at a constant speed. Thus there is no front reversal, and the invading population does not become extinct, but only migrates. This may be due to the fact that heritability is high enough that individuals behind the predator front adapt fast enough to resist and displace invaders, but competitive strength is not high enough inside the predator front.

Notice also that at the invading front tip, predators and the invading population advance together ar the same rate, there is no decoupling between the two fronts, which was what allowed the local population to become resistant in advance.

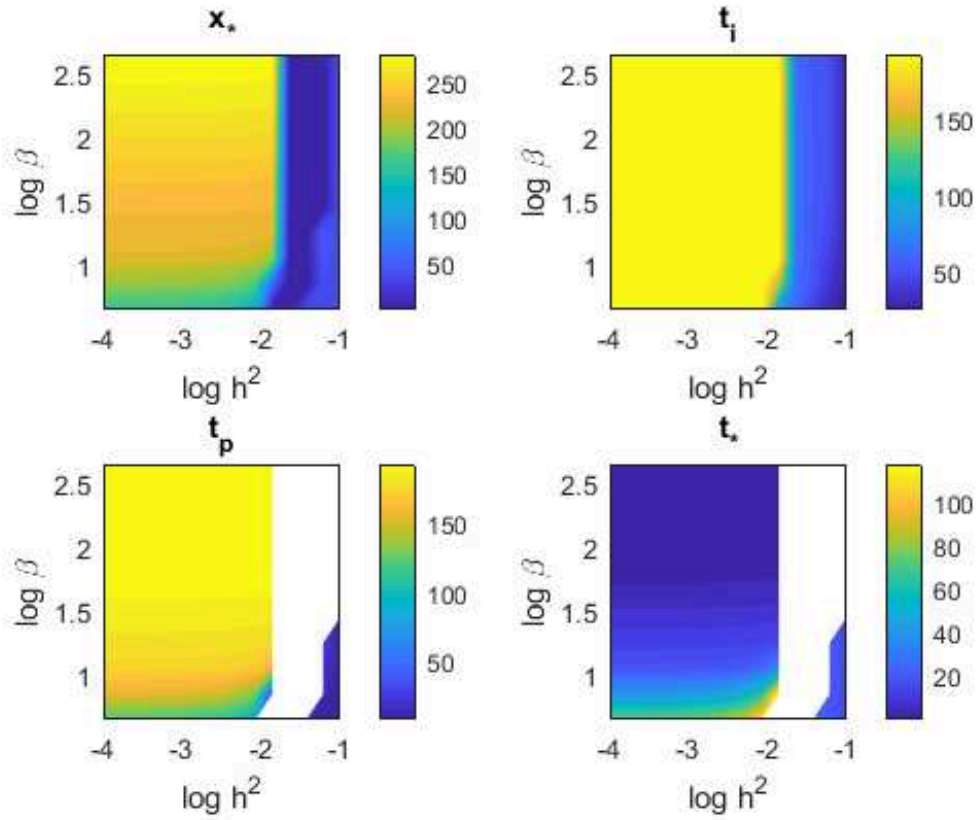

Figure 4.4: Simulation results for a second set of parameters, varying β and h^2. The graphs indicate the values of four interesting variables describing important aspects of the dynamics as a function of the heritability h^2 and predation rate β.

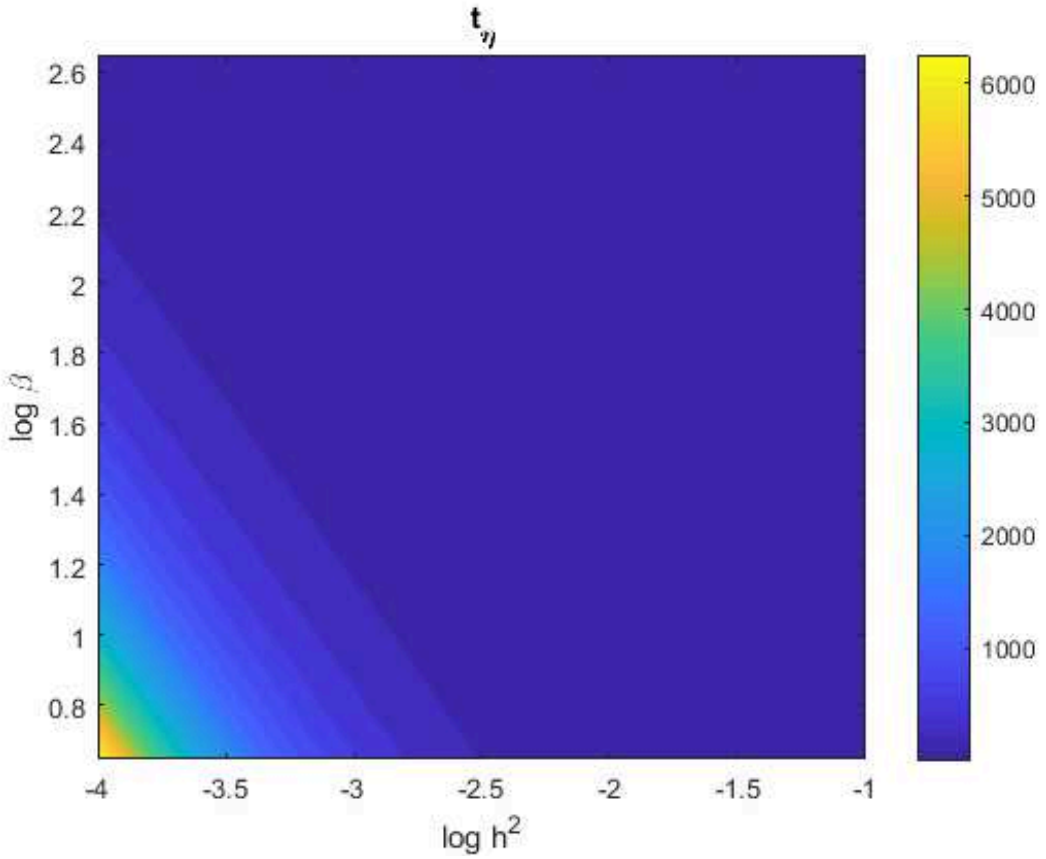

Figure 4.5: Estimated values for t_η for the chosen parameter values, indicating the critical values of β. This should be a theoretical approximation for t_*.

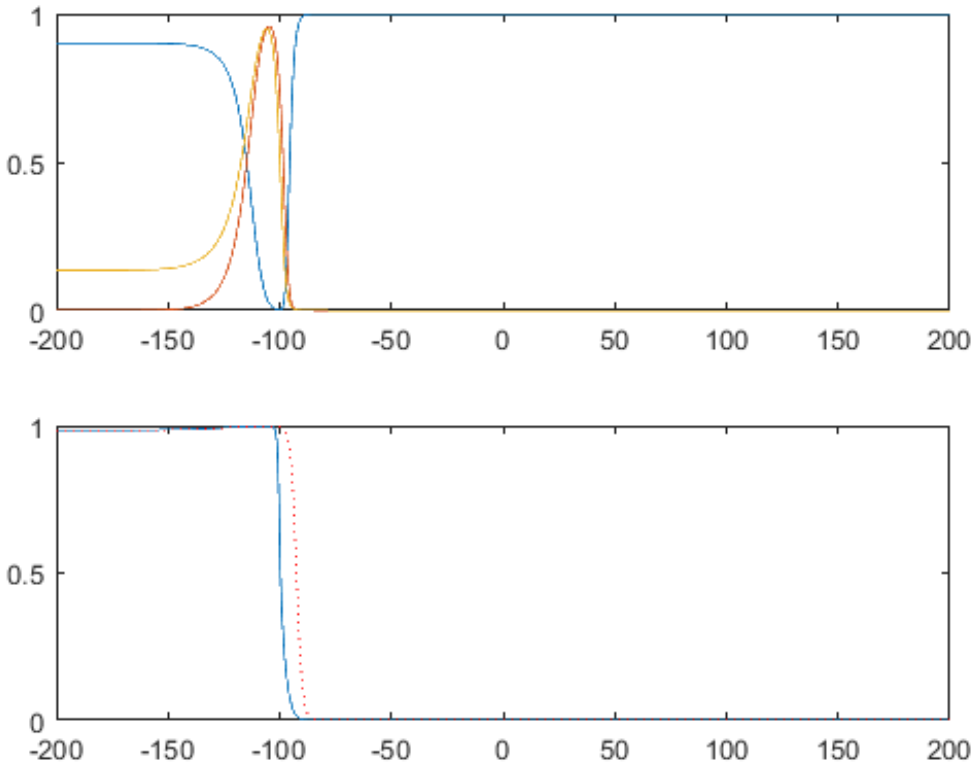

Figure 4.6: Example of a simulation in the blank space of figure 4.5.

4.4.3 Summary of the results

Although x_{stop} and t_{stop} are increasing with β, simulations show that whenever β is bigger than the critical values indicated above, the adaptation time t_* is decreasing with β, which is expected since larger β means a stronger selective pressure. Our successful simulations do not agree quantitatively with predictions, although they do agree qualitatively, and it seems that the shape of the time t_* as a function of h^2 and β higher than critical values agrees with that given by formula (4.15).

In other words our preliminary results suggest that for our simulations, when t_* is well defined, although it is not the case that $t_* \approx t_\eta$ as predicted, t_* behaves qualitatively as t_η. It remains to study simulations with a better accuracy, to test whether $t_* \propto t_\eta$.

4.5 Discussion

The case of the invasion by the gray squirrel is specially interesting in the context of shifts in apparent competitions. As said in the introduction, it was discovered that the principal mechanism helping the gray squirrels displacing the red squirrels was found to be via the squirrelpox virus (Darby et al. 2014). While this virus has had devastating consequences on the red squirrel populations in Great Britain, possible adaptations to its virulence had not been observed as it exterminated populations quickly. However, it was discovered only recently that some squirrels developed an immunity to the virus (Sainsbury et al. 2008; McGowan et al. 2018).

Our model predicts that the time to adaptation t_η should be smaller the stronger selection is, however this first estimation does not consider the population density. A strong mortality by the virus may accelerate selection, but it also decreases population sizes considerably, not leaving any adapted individuals. Besides, due to a previous extinction possible caused by hunting, red squirrels are believed to have low levels

of genetic variation to act on, with most populations being genetically isolated and related to squirrels on the mainland thanks to reintroduction efforts (O'Meara et al. 2018). This finding, combined with the fact that some squirrels can resist the virus, suggests that some populations may have the necessary genetic variance to resist mortality due to virulence. Our model predicts that, if in absence of the virus the red squirrels are competitively stronger than the gray squirrels, those resistant populations may be able to expand their ranges and stop the invasion, although the necessary time to do should be long.

Some recent studies have also found that gray squirrel populations are retracting in some locations, being replaced by red squirrels. Close inspections upon the squirrels' ecology revealed that there is a re-invasion of pine martens (*Martes martes*), a native predator of the red squirrels, which also feeds upon the invasive gray squirrels (Sheehy and Lawton 2014; Sheehy et al. 2018). The geographic distribution of the red squirrels is positively correlated to that of the pine martens, suggesting that this predator is shifting the competitive strength once again, but towards the red squirrels. Since the gray squirrel's geographic distribution in the UK has widely expanded now, by neglecting the effects of other interspecific interactions we may once again analyze this situation by means of our model, but exchanging the roles of invaders and native populations. In other words, we can apply the model to study the retraction of gray squirrels by considering red squirrels as the "invaders" of our model with pine martens as their co-adapted predators. To this moment, there does not seem to be evidence of gray squirrels adapting to predation by this species, and it is not clear whether they have enough genetic variation to grow resistance. If our suggested time to adaptation is big enough, it may take gray squirrels so long to adapt that they may have been reduced to a small area before doing so. In other words, the outcome of this re-invasion depends on the adaptation potential of gray squirrels, and the relation of time to adaptation to the spatial advancement of the fronts. If

gray squirrels manage to adapt, then the dynamics of the invasion should be well described by the competition forces between the two squirrel species (accounting for the new shifts in apparent competition due to the pine martens' presence) and the shifts in competition due to the virulence of the squirrelpox virus.

The case of the re-invasion by pine martens is complicated by the fact that the species were previously hunted by humans, and this late re-invasion is related to efforts of protection of this species in some locations in Ireland (Sheehy and Lawton 2014). However, this protection is not generalized through the UK. This last observation suggests that conservation policies taken by authorities do not always have obvious effects, and if the objective is to preserve the native squirrels, efforts may need to be directed towards protecting their native predators.

4.6 Appendix

This appendix reviews the derivation of the studied model and the definition of the time to adaptation. However, it refers to an previous version of the model which assumed that the native species could perfectly adapt to predators, while the foreign one could not. This assumption is very unnatural, but the ideas presented here are applicable to the corrected version of the model (i.e., the one presented in the main text) so we present them anyway.

For reference, the uncorrected version of the pathogen-aided invasion model is given by (the meaning of the parameters is similar to those presented in the main text, with a similar notation):

$$\begin{aligned}
\frac{\partial n_1}{\partial t} &= \frac{\partial^2 n_1}{\partial x^2} + n_1\left(1 - n_1 - \alpha_{12} n_2 - \frac{1}{2}\beta p(1-z)^2 - \frac{1}{2}\kappa z^2\right) \\
\frac{\partial n_2}{\partial t} &= \delta_2 \frac{\partial^2 n_2}{\partial x^2} + r n_2\left(1 - n_2 - \alpha_{21} n_1 - \beta' p\right) \\
\frac{\partial p}{\partial t} &= \delta_3 \frac{\partial^2 p}{\partial x^2} + Rp\left(n_2 - p + cR^{-1} n_1\right) \\
\frac{\partial z}{\partial t} &= \frac{\partial^2 z}{\partial x^2} + 2\frac{\partial \ln n_1}{\partial x}\frac{\partial z}{\partial x} + h^2\left(\beta p(1-z) - \kappa z\right).
\end{aligned}$$

4.6.1 Model derivation

We will closely follow the model derivation proposed by Mirrahimi and Raoul 2013, adapting it to describe the dynamics of a trait v corresponding to the resistance to a certain predator. We consider the local density $d_{n_1}(t, x, v)$ of native individuals whose trait is v at location x and time t. Similarly we denote by $p(t, x)$ the density of predators at that same time and location, and by $n_2(t, x)$ the density of invaders.

Denoting by ϱ the birth rate of an individual of trait v, the absolute fitness of such an individual is given by

$$s_{n_1}(t, x, v) = r - a_{11}\int d_{n_1}(t, x, w)dw - \frac{\beta}{2}(1-v)^2 p(t, x) - \frac{\kappa}{2}v^2 - a_{12} n_2(t, x)$$

So that in an spatial setting, the density of such individuals evolves according to the equation

$$\begin{aligned}\partial_t d_{n_1}(t,x,v) - \frac{\sigma^2}{2}\partial^2_{xx} d_{n_1}(t,x,v) = &\Bigg[(r-\varrho) - a_{11}\int d_{n_1}(t,x,w)dw \\ &-\frac{\beta}{2}(1-v)^2 p(t,x) - \frac{\kappa}{2}v^2 - a_{12}n_2(t,x)\Bigg] d_{n_1}(t,x,v) \\ &+ \varrho \int\int \frac{d_{n_1}(t,x,w_1)d_{n_1}(t,x,w_2)}{\int d_{n_1}(t,x,w)dw} Q(v,w_1,w_2)dw_1 dw_2\end{aligned}$$

Integrating along v we can derive the dynamics for the population density $n_1(t,x) = \int d_{n_1}(t,x,v)dv$, which is given by

$$\begin{aligned}\partial_t n_1(t,x) - \frac{\sigma^2}{2}\partial^2_{xx} n_1(t,x) = &\left[r - a_{11}n_1(t,x) - a_{12}n_2(t,x)\right] n_1(t,x) \\ &- \int \left(\frac{\beta}{2}(1-v)^2 p(t,x) + \frac{\kappa}{2}v^2\right) d_{n_1}(t,x,v)dv\end{aligned}$$

Denote by $f_1(t,x,v) = \dfrac{d_{n_1}(t,x,v)}{n_1(t,x)}$ the probability density function of individuals with trait v at location x at time t. The average population trait is defined by $z(t,x) = \int v f_1(t,x,v)dv$. The previous equation can then be rewritten as

$$\begin{aligned}\partial_t n_1 - \frac{\sigma^2}{2}\partial^2_{xx} n_1 = &\left(r - a_{11}n_1 - a_{12}n_2\right) n_1 - \left(\frac{\beta}{2}(1-z)^2 p + \frac{\kappa}{2}z^2\right) n_1 \\ &- n_1\left(\frac{\beta}{2}p + \frac{\kappa}{2}\right)\int (z-v)^2 f_1(v)dv\end{aligned}$$

We can derive an equation for the average trait $z(t,x)$ by computing the expression

$$\begin{aligned}\partial_t z - \frac{\sigma^2}{2}\partial^2_{xx} z = &\partial_t \int v\frac{d_{n_1}}{n_1}dv - \frac{\sigma^2}{2}\partial^2_{xx}\int v\frac{d_{n_1}}{n_1}dv \\ = &\int \frac{v}{n_1}\left(\partial_t d_{n_1} - \frac{\sigma^2}{2}\partial^2_{xx} d_{n_1}\right)dv - \int \frac{v}{n_1}\frac{d_{n_1}}{n_1}\left(\partial_t n_1 - \frac{\sigma^2}{2}\partial^2_{xx} n_1\right)dv \\ &+ \sigma^2 \int v\frac{\partial_x n_1}{n_1}\partial_x\left(\frac{d_{n_1}}{n_1}\right)dv\end{aligned}$$

from where we see that

$$\partial_t z - \frac{\sigma^2}{2}\partial_{xx}^2 z = \int \frac{v}{n_1}\left(\partial_t d_{n_1} - \frac{\sigma^2}{2}\partial_{xx}^2 d_{n_1}\right) dv - \frac{z}{n_1}\left(\partial_t n_1 - \frac{\sigma^2}{2}\partial_{xx}^2 n_1\right) + \sigma^2 \partial_x \ln n_1 \partial_x z$$

which boils down to

$$\begin{aligned}\partial_t z - \frac{\sigma^2}{2}\partial_{xx}^2 z =& z\left(\frac{\beta}{2}(1-z)^2 p + \frac{\kappa}{2}z^2\right) + z\left(\frac{\beta}{2}p + \frac{\kappa}{2}\right)\int (z-v)^2 f_1(v)dv \\ &- \int v\left(\frac{\beta}{2}(1-v)^2 p + \frac{\kappa}{2}v^2\right) f_1(v)dv + \sigma^2 \partial_x \ln n_1 \partial_x z\end{aligned}$$

this is easily computable noting that

$$\begin{aligned}\frac{\beta p}{2}(1-v)^2 + \frac{\kappa}{2}v^2 =& \frac{1}{2}(\beta p + \kappa)\left[(v-z)^2 + 2(v-z)\left(z - \frac{\beta p}{\beta p + \kappa}\right) + \left(z - \frac{\beta p}{\beta p + \kappa}\right)^2\right] \\ &- \frac{1}{2}\frac{\beta p \kappa}{\beta p + \kappa}\end{aligned}$$

yielding

$$\begin{aligned}\partial_t z - \frac{\sigma^2}{2}\partial_{xx}^2 z =& z\left(\frac{\beta}{2}p + \frac{\kappa}{2}\right)\int (z-v)^2 f_1(v)dv + \sigma^2 \partial_x \ln n_1 \partial_x z \\ &- \frac{1}{2}(\beta p + \kappa)\int \left[(v-z)^3 + 2(v-z)^2\left(z - \frac{\beta p}{\beta p + \kappa}\right) + z(v-z)^2\right] f_1(v)dv \\ =& \sigma^2 \partial_x \ln n_1 \partial_x z - (\beta p + \kappa)\left(z - \frac{\beta p}{\beta p + \kappa}\right)\int (v-z)^2 f_1(v)dv \\ &- \frac{1}{2}(\beta p + \kappa)\int (v-z)^3 f_1(v)dv.\end{aligned}$$

It can be shown that when the reproduction kernel $Q(v, v_1, v_2)$ is Gaussian, the second moment of the distribution of the phenotypical trait v converges exponentially fast to a constant value, and the third moment around the average converges to zero at a similar rate (Mirrahimi and Raoul 2013). Thus we can approximate $\int (z - v)^2 f_1(v)dv \approx G$, the additive genetic variance of the trait by a constant, and $\int (z - v)^3 f_1(v)dv \approx 0$.

We finally find the equation system

$$\begin{aligned}
\partial_t n_1 - \frac{\sigma^2}{2}\partial^2_{xx} n_1 &= n_1\left[r - a_{11}n_1 - a_{12}n_2 - \frac{\beta}{2}(1-z)^2 p - \frac{\kappa}{2}z^2 - \frac{G}{2}(\beta p + \kappa)\right] \\
\partial_t z - \frac{\sigma^2}{2}\partial^2_{xx} z &= \sigma^2 \partial_x \ln n_1 \partial_x z - G(\beta p + \kappa)\left(z - \frac{\beta p}{\beta p + \kappa}\right) \\
\partial_t n_2 - \frac{\sigma_2^2}{2}\partial^2_{xx} n_2 &= n_2\left[r_2 - a_{12}n_1 - a_{22}n_2 - \frac{\beta'}{2}p\right] \\
\partial_t p - \frac{\sigma_3^2}{2}\partial^2_{xx} p &= p\left[\gamma_1\beta n_1 + \gamma_2\beta' n_2 - \alpha p\right].
\end{aligned}$$

The proposed model can then be found after a renormalization of the variables.

4.6.2 What is an "adapted trait"?

We proposed a definition of time to adaptation t_η in the main text by measuring the time it takes the treat to be at a certain relative distance from the optimum. This is, however, not the only option. In this appendix we review alternative ways if defining what an "adapted trait" is in the context of pathogen-aided invasions.

- As before, we can fix a relative distance from the optimum, $0 < \eta < 1$, say $\eta = 5\%$, and measure t_* as the time needed to achieve this distance, i.e., $t_* = t_{1-\eta}$ as give by formula (4.15).

- In fact z does not need to be at its equilibrium value for n_1 to survive, we just need it to be big enough so that the local population has a positive fitness. Another idea is to find z_* so that n_1 has a positive fitness and let t_* be the time so that $z(t_*) = z_*$.

  The local population has a positive fitness if

$$1 - n_1 - \alpha_{12}n_2 - \frac{1}{2}\beta p(1-z)^2 - \frac{1}{2}\kappa z^2 > 0$$

which is equivalent to

$$\left(z - \frac{\beta p}{\beta p + \kappa}\right)^2 < 2\left(1 - n_1 - \alpha_{12} n_2\right) - \frac{\beta p \kappa}{\left(\beta p + \kappa\right)^2} \tag{4.28}$$

Given that the right hand side of this expression is positive, this implies that (considering that $z(0) = 0$)

$$z_* = \frac{\beta p}{\beta p + \kappa} - \sqrt{2\left(1 - n_1 - \alpha_{12} n_2\right) - \frac{\beta p \kappa}{\left(\beta p + \kappa\right)^2}} \tag{4.29}$$

and expression (4.14b) lets us approximate the time to achieve this trait value as

$$t_* = -\frac{1}{2h^2(\beta p + \kappa)} \ln\left(2(1 - n_1 - \alpha_{12} n_2)\left(1 + \frac{\kappa}{\beta p}\right)^2 - \frac{\kappa}{\beta p}\right) \tag{4.30}$$

- Another useful idea to approximate this time is to see how the trait z impacts the intra- and interspecific competition coefficients, so that this adaptation time t_* can be interpreted as the first time local population is competitively strong enough to stop invasion. In other words, considering fitness of the local population as a function of the mean trait

$$r_1(z) = 1 - n_1 - \alpha_{12} n_2 - \frac{1}{2}\beta p (1 - z)^2 - \frac{1}{2}\kappa z^2$$

Again, supposing R is large enough that p quickly attains its equilibrium density, we may approximate $p = cR^{-1}n_1 + n_2$, yielding

$$r_1(z) = \left(1 - \frac{\kappa}{2} z^2\right)\left(1 - \frac{1 + \frac{\beta c}{2R}(1 - z)^2}{1 - \frac{1}{2}\kappa z^2} n_1 - \frac{\alpha_{12} + \frac{\beta}{2}(1 - z)^2}{1 - \frac{1}{2}\kappa z^2} n_2\right)$$

So that, focusing on the relation between the competition coefficients for the

local population to be resistant to invasion, we find that the following condition must be met

$$\frac{1+\frac{\beta c}{2R}(1-z)^2}{1-\frac{1}{2}\kappa z^2} < \alpha_{21}, \text{ and } \frac{\alpha_{12}+\frac{\beta}{2}(1-z)^2}{1-\frac{1}{2}\kappa z^2} < 1$$

These conditions are equivalent to

$$\frac{1}{2}\left(\frac{\beta c}{R}+\kappa\alpha_{21}\right)\left(z-\frac{\beta c/R}{\kappa\alpha_{21}+\beta c/R}\right)^2+\frac{1}{2}\frac{\kappa\alpha_{21}\beta c/R}{\kappa\alpha_{21}+\beta c/R}+1-\alpha_{21}<0 \quad (4.31)$$

and

$$\frac{1}{2}(\beta+\kappa)\left(z-\frac{\beta}{\beta+\kappa}\right)^2+\frac{1}{2}\frac{\beta\kappa}{\beta+\kappa}+\alpha_{12}-1<0 \quad (4.32)$$

Notice that for these conditions to be feasible it is necessary that

$$\alpha_{21} > \frac{1}{2}\frac{\kappa\alpha_{21}\beta c/R}{\kappa\alpha_{21}+\beta c/R}+1 \text{ and } \alpha_{12} < 1-\frac{1}{2}\frac{\beta\kappa}{\beta+\kappa},$$

otherwise, the previous quadratic functions will always be strictly positive.

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CHAPTER V

Synthesis and Discussion

In this chapter, I summarize the principal results of this thesis, and I discuss possible applications and extensions.

In this work, I studied the effects of interspecific interactions and adaptation on the geographic dynamics of species. With this objective in mind, I explored different kinds of interactions and modes of adaptation. Thus, Chapter II deals with the problem of spatial distribution of a single species suffering an adaptation pressure from the environment. Chapter III shifts the focus in interaction to predation, studying the distribution of a predator-prey system where both species undergo adaptation to the environment. Finally, in Chapter IV I explored a different mode of adaptation by considering changes in the interaction strength between two species. That last chapter deals with the adaptation to pathogens and its consequences on apparent competition, which can explain the success or failure of pathogen-aided invasions. The three chapters reveal the different effects adaptation and interactions can have on spatial dynamics of species, complementing each other in the way adaptation and interactions can act upon a community. As a consequence, different types of feedbacks are also revealed.

The important components of each model can be summarized in three levels, depending on the described interactions, the nature of the spatial context, and how

selection acts upon the involved species. These components reveal several the important similarities and differences in the three studied problems. They are presented in Table 5.1 and we review them in the next paragraphs.

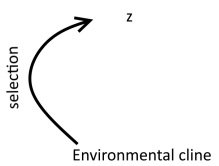
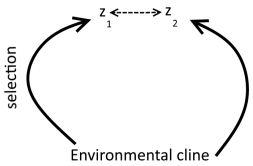
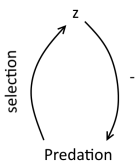
Chapter	II	III	IV
Species and Interactions	1 species under instraspecific competition.	2 species: a prey and its predator.	3 species. A predator mediating apparent competition.
Space	Environmental gradient.	Environmental gradient.	No variation in abiotic environment. Heretogeneity in competition and predation context.
Adaptation			

Table 5.1: Summary of the different important components of the problems studied in each chapter. They are related to the number of species and the nature of their interactions, the importance of space in their dynamics, and the way selection acts upon them.

Concerning interactions, the model studied in Chapter II is a monospecific model, with one species undergoing **instraspecific competition**. The interest of studying such a model is that, when studying the effects on adaptation of species in an interacting network, the difference between effects that arise independently for each species and those that are caused by interaction can be clearly stated. In Chapter III the focus in interactions was shifted, moving onto a two-species predator-prey system, marking a difference with respect to the previous chapter, as **predation** was the main interaction in the system. A similar case focused on interspecific competition was already studied by Case and Taper (2000), but the study of the effects of predation is

new in this context. At last, Chapter IV treats a complex three-species community, where we analyzed the effects of predation and the adaptation to the predators on the dynamics of a possible invasion. We can understand the changes in distribution as competitive shifts mediated by the degree of predation on the native species, so in this context we can say that the focus is on **apparent competition**.

Regarding the spatial context in each chapter, in Chapters II and III the studied models considered a **heterogeneous environment** because for each location there is an optimal phenotype value that varies linearly with distance. This implies that different phenotypes are not equally performing on each location, so that space has an important explicit influence on species dynamics. In Chapter III, it was additionally supposed that the phenotypic optima were not necessarily the same for both species, which gives the model some versatility. But even supposing that these optima were the same for both species at each location, the model reveals that the perceived heterogeneity would be different anyway, since these species can differ greatly in dispersal, genetic structures, growth rates, etc. Chapter IV, however, considers **homogeneous space** in the sense that interactions and dynamics of species do not depend explicitly on the abiotic condition on the environment where they take place, but on the local values of variables. Nevertheless, there is a heterogeneity that appears as a consequence of a new species disturbing the local dynamics, native species adapting to them, and selected traits diffusing due to gene flow. In other words, space is homogeneous in nature, but the local adaptation to interactions is a source of heterogeneity that is allowed to propagate. This last chapter reveals an interesting effect of space indirectly impacting on the dynamics of the system. In this context, the adaptation to the pathogens and the shifts in apparent competition are what cause a boom and bust dynamics in invasion, which are commonly observed in nature (Mack et al. 2000; Simberloff and Gibbons 2004; Simberloff et al. 2013; David et al. 2017).

Finally, in terms of selection, a similarity all of the models share is that adaptation

is only to local conditions, i.e., only the local values of the involved variables are relevant, while links between localities are modeled through diffusion. However, an important difference is that in Chapters II and III the adaptation is directly to their abiotic environment, as the optimal phenotype value is an spatial cline, depending on the difference between the population's phenotype and the optimal one; while in Chapter IV individuals adapt to their predators or pathogens, depending on their local densities, and this induces a direct eco-evolutionary feedback. While in Chapter II the spatial gradient is unique and fixed for the considered species, when adding another interacting species it is necessary to define a way space is going to act on it. In Chapter III we did not restrict the spatial gradient to be the same for both species, but even if it was, the differences between adaptation capabilities in both species (which depend on their genetic variances, strength of selection and population growth rates) can cause the perceived heterogeneity in space to be different for each one of them. In other words, relative heterogeneity for both species differ because of selective differences. The spatial context of interactions is mediated through each species' traits, thus being indirectly linked. Selection in Chapter IV, on the other hand, concerns only a trait of one species and acts upon the interaction between this one and the introduced predator. Since adaptation modifies the strength of selection, this last problems introduces a previously absent feedback in interactions (refer to Table 5.1).

5.1 Understanding the effects of evolution through propagation speeds

An important characteristic of Kirkpatrick and Barton's model is that it has solutions that behave like traveling waves with a characteristic speed. This is an idea that is useful to analyze and understand the three problems we studied.

The first studied problem is a new analysis of Kirkpatrick and Barton's equations, focusing the study on the propagation speed of its solutions. We found a clear link with the classical solutions described by Kirkpatrick and Barton (1997): solutions with positive propagation speed correspond to solutions with unlimited range in their analysis, while those with negative propagation speed correspond to solutions in a limited range or to extinct populations. We also showed that when the adaptation potential of the species tends to infinity or to zero, we recover Fisher's model (Fisher 1937) or the approximation derived by Mirrahimi and Raoul (2013), respectively. This is very advantageous, since we have explicit approximations for the propagation speed in these two cases, leaving only a small number of cases to be approximated numerically.

When changing the interaction to deal with a predator-prey system, Chapter III is easily understood when looking independently at each species' propagation speed. We state three main results. First, we found that speeds in the joint system are the same as the respective one-species speed separately for prey and predator (after an appropriate rescaling of variables) unless the predator's propagating speed is larger than the prey's. This implies that when prey or predator's adaptation potential is large (resp. small), its propagation speed is just given by the speed of Fisher's (resp. Mirrahimi and Raoul's) models, after an appropriate rescaling of the variables. Second, when predator's intrinsic speed is larger that its prey's, both species propagate through space as a community, with a possibly lower speed than the prey's intrinsic one (the lower of the two, when predator's diffusion and predation rate are strong enough). In this case, the approximations of the first chapter are not valid, although they do provide an upper bound to the propagation speed of the propagating community. Third, a large diffusion rate for predators may not be advantageous, as the perceived environmental heterogeneity becomes larger for individuals locally adapted to farther locations, thus possibly making propagation

speed to decrease. This third result is actually present in Kirkpatrick and Barton's analysis: the perceived heterogeneity B in their model is proportional to the species diffusion rate, so for a fixed level of adaptation A very large diffusion rates can increase heterogeneity to a point where heterogeneity is unsurmountable so the species become extinct.

The last problem, about an invading competitor with its pathogen, was analyzed through propagation speeds of the pathogen in its foreign range and of its co-introduced host. We found that the spatial extension of the pathogen plays a crucial role in the evolution of defenses of the native species, which in turn is what allows it to change its apparent competition strength to stop the foreign competitive species' invasion. This stopping condition is understood simply as the moment where the propagation speed of the foreign competitor changes sign. The time required to stop the invasion is inversely related to the native species' adaptation potential, thus the larger the adaptation potential, the sooner the invasion is stopped. An important difference with the previous models is that adaptation is linked to a biotic interaction, and not to the spatial characteristics of the environment. The existence of a feedback between interaction and adaptation also causes the spatial source of selection to be in constant change, so this important difference with respect to the first problem does not let us apply the results found there.

All of these observations reveal that species propagation speeds contain important relevant information about their adaptive capabilities, even when adaptation is not directly related to the environment (Chapters II, III) but to the interactions (Chapter IV). It is an indicator linking the ecological and evolutionary characteristics of the observed species. We can compare these speeds more directly by looking at the formulae we derived in each chapter. Table 5.2 summarizes the principal results in terms of the propagation speeds. Notice that there is a close relationship between the results of Chapters II and III as the speeds for the interacting predator-prey case

can be well described by the speeds for the one-species case. In other words, after an appropriate renormalization of the variables, the predator and the prey propagation speeds can be derived from the one-species case. This result is not surprising for prey propagation, as they do not need the predator in order to invade new areas, but for predators it means that as long as prey availability is guaranteed, their dynamics are governed by a monospecific model, meaning that the principal restrictions to their range expansion are their relative adaptation capabilities and the perceived spatial heterogeneity. However, this analysis is not applicable to the system studied in Chapter IV because of the fundamental differences in space and the nature of adaptation. Notice that in this last chapter there is a direct evolutionary effect on the strength of interaction, while interactions are the principal drivers of heterogeneity. This feedback between adaptation and interaction is what causes the heterogeneity to change constantly, so that propagation speeds change too. A fundamental feature of Kirkpatrick and Barton's framework is that the environmental cline does not vary in time, whereas the heterogeneities in this invasion model are constantly changing.

An important observation that links the three models, and which can also be observed in the equations, is that larger adaptation potential (A) implies better persistence for the considered species. In other words, better adaptation capabilities imply that the species will not become extinct to easily, as they are able to invade more space (Chapters II and III) or they take less time to recover their own space by adapting to foreigners (Chapter IV).

However, the quality of adaptation is not the same for the three models. An important difference between the first two models and the third one concerns the nature of gene flow. For the first two models gene flow is bad as immigrants from far locations are better suited for those external conditions, depressing the fitness of local populations due to the intrinsic heterogeneity in space. In fact, this is the main motivation for the original model by Kirkpatrick and Barton 1997, who showed that

Chapter	Speed	Comments
II	$c = c_{KB}(A, B)$	The propagation speed in Kirkpatrick and Barton's equation depends positively on the adaptation potential A and negatively on the spatial heterogeneity B.
	$c_{KB}(A \to \infty, B) = 2$	When the adaptation potential tends to infinity, c_{KB} tends to the propagation speed of Fisher's model (Fisher 1937), which is $c_F = 2$ in its non-dimensional form.
	$c_{KB}(A \to 0, B) = c_D$	When the adaptation potential is small while keeping the environmental challenge $D = B/\sqrt{A}$ constant, the propagation speed depends mostly on it (refer to equation (2.3)).
III	$c^I_{\text{prey}} = c_{KB}(a_1, b_1)$ $c^I_{\text{pred}} = \sqrt{\delta r s}\times$ $c_{KB}\left(\frac{a_2}{\sqrt{s}}, \frac{b_2}{s}\sqrt{\frac{\delta}{r}}\right)$	Propagation speeds are well approximated by the respective one-species intrinsic speeds in most cases, behaving similar to c_{KB}. (Predator speed needs rescaling. Here $s = 1-d$, the "inverse" of the death rate).
	$c^J_{\text{prey}} \leq c^I_{\text{prey}}$ $c^J_{\text{pred}} \leq c^I_{\text{pred}}$	When prey and predators interact at the front tip, propagation speeds may be smaller than individually.
IV	$t_\eta = \frac{-\ln \eta}{A(\beta B p + \kappa)}$	The time needed to reverse the speed of invasion is proportional to t_η, the time it takes the trait to be at a proportional distance η from the optimum (with no spatial influence and constant selection).

Table 5.2: Summary of the important formulae in each chapter, mostly speeds, which reveal the links between each result.

species can have a restricted range despite not having evident barriers to dispersal, due to gene flow from maladapted populations. On the contrary, gene flow is desirable in the third model, as individuals well adapted to predators can pass their genes to their progeny, making this adaptation to propagate in space. This mechanism allows populations that may have never been exposed to the pathogens to take shorter times to adapt.

In each case, the propagation speed of each population depends on an evolution-

ary parameter, which was dubbed the adaptation potential, directly related to the species' genetic variance. Thus, the propagation speed of expanding or retracting populations can be interpreted as an eco-evolutionary index, quantifying simultaneously the capabilities of the species to disperse and adapt. Ecological features of biological systems being interpreted as evolutionary indexed is not a new idea (e.g. Yoshida et al. (2003) and Hiltunen et al. (2014)), and evolutionary speeds in the context of environmental clines have also been studied in comparison to typical Fisher's speed (García-Ramos and Rodríguez 2002). A straightforward conclusion is that by knowing the evolutionary parameters of each population (genetic variance and heritability of the considered trait), the ecological characteristics of the communities (growth and dispersal rates, competition and predating costs for each species) and measures of spatial heterogeneity (the steepness of the environmental cline), we can predict the propagation speeds in each case. However, here I highlight the conclusion is that by measuring the speeds of advancement or retraction, plus the relevant ecologic and environmental variables, we may obtain information on the species evolutionary characteristics, through possible values of the adaptation potentials dictated by the corresponding models. Chapter III tells us that for a predator-prey system we can derive this conclusions from one-species dynamics, unless possibly if prey and predator are simultaneously expanding (since joint speeds may be lower than individual intrinsic speeds). At the same time, equation (2.4) in Chapter II tells us that if, for example, and species is invading space at a speed well-aproximated by Fisher's model, i.e., $c = 2\sqrt{\delta r}$ despite an important environmental cline, then the adaptation potential must be bigger or equal than the solution A^* of this equation. This gives us an upper bound on the species' genetic variance since $A = G/(2V_s r)$. It is difficult to obtain similar conclusions for Chapter IV as the results are only qualitative, but provided this is a good approximation, we could still find conclude one population has double the heritability in resitance as another one, for example if the time it took

for it to stop an invasion was half the time it took the other population do the same.

To summarize, as a general conclusion, evolution changes the distribution of species by positively impacting their propagation speeds when the adaptation potential grows larger. This is a direct result in Chapter II as indicated in Figure 2.3, and it thus valid for the predator-prey system through intrinsic speeds. Note that in the invasion problem, a negative impact on the foreign competitor is equivalent to a positive impact on the native species, so the conclusion still follows. For a predator-prey system, there may be an additional ecological effect when predators reach their prey front; while in the invasion system the evolution of resistance to pathogens modifies the strength of apparent competition. In each case the quality of adaptation is different, as evidenced by the nature of gene flow, which is advantageous in the invasion problem, but brings maladaptation in the previous ones.

5.2 Under what conditions can we make predictions about the species distributions?

The underlying hypotheses used to propose these models are based on the hypotheses of the infinitesimal model. This implies that traits are genetically determined in an additive manner by an infinite number of traits with infinitesimal contribution. The traits are also assumed to be normally distributed with a constant phenotypic variance at the local scale, individuals reproduce sexually and the traits of the offspring follow a normal whose mean equals the mean of the parent's traits. Selection is also assumed to be constant through space, except in the last model, where its strength depends on the local pathogen density.

There are also additional hypotheses on the form of the individual's fitnesses. For example, the incurred cost in fitness due to deviation of the trait from the optimal is always a quadratic function.

This approach is useful in that species distributions are easily predictable: knowing the propagation speed of a species, the new distribution will only be the variation given by this speed over time.

The three studied problems differ in that they treat different ecological communities. The first chapter analyzes the effects of evolution in a monospecific scenario, which allows us to clearly understand the effects of evolution on spatial distributions, complementing the original approach by Kirkpatrick and Barton and linking it other simpler models. The second chapter is a complexification of the first model by adding a predator. In turn, this complements the problem studied by Case and Taper (2000), which is also a two-species model, but where the interaction is competition. These two first chapters are similar in the type of environment considered: the selective pressure is exerted by the environment, of which the heterogeneity is measured through a cline. The third problem is different in that the interesting interaction is apparent competition, so that three interacting species need to be considered: two competitors and a common predator. The environment does not directly play an important role, since the heterogeneity in space is caused by the wave of advancement of predators and the successive adaptation to their predation rate, instead of an adaptation directly to the environment.

An implication for species distribution models is that our results reveal that the niche of a species is expected to change constantly over time. Indeed, the eco-evolutionary speeds of Chapters II and III are a measure of the rate of change of allowed phenotypes over space permitting the species persistence, so it measures a change in fundamental niche; while adaptation in Chapter IV modifies the strengths of interactions, so speeds change as a consequence of adaptation to interaction strengths, and thus they measure a change in realized niche. As the conditions (biotic or abiotic) for the persistence of a species change over time, the niche of a species cannot be understood as a fixed entity, unless, for example, the species has a small adapta-

tion potential A or it has a small growth rate r. For species with large adaptation potentials, the results of Chapter II suggest that their distributions are mostly limited by dispersal (since their spatial distribution obeys to Fisher's model), so that for these species a niche envelope approach mat not be accurate in predicting future distributions.

For interacting predator-prey pairs, there are more important considerations to observe. Predators may not only be limited by dispersal or by evolutionary factors, but also by prey availability, so that their eco-evolutionary speed matches that of their prey. In other words, when a predator's intrinsic eco-evolutionary speed is larger than its prey's, as the two propagating fronts meet, the predator populations at the edge will not be able to advance faster because of the absence of prey. As a result, they geographic distribution of predators will be perfectly correlated to that of their prey. An implication for niche models will be, thus, that for specialist predators with important adaptation capabilities (compared to their prey's), the interesting abiotic niche to be measured is not theirs, but their prey's.

Notice, however, that when predators are good dispersers they may be slowing their prey down, so that prey's niche changes might not be explained by their characteristics alone. In other words, as the eco-evolutionary speed of prey does not coincide with its intrinsic speed, the observed changes in niche do not accurately correspond to their adaptation capabilities. Were one to measure the adaptation potential of a predated species in such a scenario, one would obtain an underestimation. Thus, ecological interactions are important to be observed when comparing expected evolutionary speeds to observed propagation speeds (see e.g. García-Ramos and Rodríguez (2002)).

5.3 Possible applications

As already said, an important implication of the analysis in terms of invasion speeds is that there is a clear link between propagation speed and adaptation potential. Knowing the propagation speed of a single species following a climate gradient can let us draw conclusion about its adaptation potential, and thus about its genetic variances.

The results suggest alternative ways to understand the limits of ranges of existing populations. For example, for the one species model, as the speed changes in terms of the spatial heterogeneity B, an abrupt change in heterogeneity may constrain a population from advancing. In other words, this suggests that space may be continuous with no clear barriers to dispersal, but species may still be constrained in their ability to invade since there is an important change in spatial heterogeneity, difficult or impossible to surmount given their level of adaptation potential.

In the invasion case, the conclusion is that if native species is capable of excluding their competitors in absence of their predators and when optimally adapted to them, then it should be able to stop the invasion. The time it takes to stop the invasion is given by t_η to an order of magnitude. Notice that this also implies that the extent of the invasion is proportionally to this time, so that species constrained to a limited space (for example, in a small island) may face extinction even if they are potentially able to adapt.

5.4 What kind of extensions are possible?

There are several useful ways in which the studied scenarios can be extended. One of the most relevant ones is to study the effects of climate change on interacting networks. A monospecific scenario was already studied in the article by Lande (1976). Although the population growth rate was different, they show that given that the

species has enough adaptation potential and dispersal, it can follow the changes in the environmental gradient with a certain lag. An interesting scenario was studied by Norberg et al. (2012), who show that there are important differences when considering separately interactions, evolution or both.

Our results let us already make some predictions about what can happen in cases where climate change acts. Suppose the optimal phenotype changes over time at a rate v, so that $\theta(x,t) = bx + vt = b\,(x - v/bt)$. This implies that the optimal phenotype advances through space at a rate v/b which then may be a critical speed for the considered species to follow. In other words, it is interesting to compare the ability to track space, c_{KB} with the speed of perceived environmental change v/b.

Another insight of this first approximation is that in the two species system, since predator and prey have different adaptation potentials and dispersal capabilities, even when they are subject to the same environmental cline, the perceived spatial heterogeneity may be different. This implies that both species would not perceive the climate change in the same way so that one of them could be more affected than the other. In the case where prey are constrained by the predator front, this implies that asymmetries in climate perception may slow down the predator front while not so much the prey one, and thus releasing prey from predator control, with prey invading faster than predicted.

The invasion by the cane toad in Australia is a interesting case because it is ongoing and it resurfaces several questions about evolution and interactions. A particularly interesting point is the evolution of dispersal: expansion rates at the invasion front have been observed to accelerate (Phillips and Shine 2006; B. L. Phillips et al. 2010). This poses the question of whether some traits related to dispersal can be selected for at the range edges, just through means of spatial sorting (Shine et al. 2011). When space is homogeneous and there are no barriers to dispersal, there does not seem to exist an obvious trade-off to dispersal, other than energetic costs for movement.

Such a study has already been carried in a stochastic setup by Benjamin L. Phillips et al. (2008). They found that when energetic cost are a trade-off, measured as an increasing probability of death the longer displacements are, there is effectively a limiting dispersal, while faster individuals concentrate at the front's edge. However, note that Kirkpatrick and Barton's model evidences another possible trade-off, given by fitness costs brought by maladapted gene flow at farther populations. In other words, individuals that disperse farther are affected by the fact that they are better adapted elsewhere.

Such a study can be carried out via a multi-trait extension of our model presented in Chapter II, where one of the evolving traits is dispersal. Following the derivations presented in the introduction, multi-trait models can be proposed in a straightforward fashion (see e.g. Lande (1979) and Duputié et al. (2012)). However, it also poses the question of how genetic correlations can alter the dynamics of dispersal.

5.5 Final thoughts and perspectives

The current global context evidences an urgent need to understand the complex mechanisms driving the dynamics of species distributions. Climate change has important effects on the structure and interactions in ecological communities, and exotic species are nowadays commonplace. While species distribution models are undoubtedly powerful tools describing the presence of a species in terms of its environment, we need to be careful when interpreting the results, as dispersal and interactions may impact dynamics in ways more complex than initially thought. This thesis is an effort towards describing these impacts, revealing important implications for the estimation of niche of species in space and their change over time. Understanding current and possible future distributions thus asks for tools integrating multiple approaches. This work has shown that even considering the effects of small interaction networks can give us enlightening insights.

The presented results show that the adaptation potential of a species is a very relevant factor to consider when interpreting the results of a species distribution model. Concerning species subject to selection on an environmental gradient, on the one hand, when the adaptation potential is large, the species is mostly limited by dispersal. Thus, with fair confidence one can say the realized niche will be an underestimation of the fundamental niche as abiotic conditions are not a limiting determinant of the species range. An important consideration is whether such a species is a predator or is under predation, as the way a predator-prey pair interacts can alter our interpretation of their niche. Prey may be slowed down by predators that are good dispersers, while predators may be slowed-down by prey with small dispersal due to prey availability at the edge of their range. On the other hand, small adaptation potentials imply small eco-evolutionary speeds, thus in this case usual species distribution models may serve well, as niche is not expected to change pronouncedly over time. In the context of ecological invasions, co-introduced pathogens that are good dispersers may help native species overcome infections. This adaptation can induce shifts in apparent competition, thus stopping the range expansion of an exotic species. This result suggests an explanation for boom and bust cases in invasions: they are possible and expected when such a native species has good adaptation potential. The better adaptation is, the sooner the bust phase will occur.

In summary, the presented results show that niche models need to be interpreted carefully, as the adaptation potential of a species and its dispersal affect its realized niche and its change over time the ways that were described. A more integrated approach should consider these two factors, together with the important interactions such a species experiments.

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