CHAPTER 2

# The theory of dispersal under multiple influences

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## 2.1 Introduction

In this book, dispersal is defined as movement that leads to gene flow. This is a fairly wide definition of dispersal, as it is not limited to what type of entities that move (adults, juveniles, seeds, or pollen), mechanism of movement (passive dispersal, active locomotion, etc.), and whether or not the movement is seen as an evolutionary adaptation. To survey models that deal, in some sense, with movement that leads to gene flow is therefore difficult. Here we will briefly chart the established ultimate factors affecting dispersal evolution, as well as the proximate factors that can be of importance in individuals' decisions to disperse or to stay philopatric.

A prerequisite in understanding the theory of dispersal is that not every model is built with the same scope and purpose. A classic in the modelling literature is the trade-off between generality, realism, and precision, presented by Levin (1966). Though this view has been criticized for being somewhat unclear (see, for instance, Odenbaugh 2006), it does point to the fact that not all models yield the same *kind* of insight or knowledge. The scope of some models is to fit a specific biological system (e.g. Zheng *et al.* 2009), while others can be seen as proof-of-principle models, where a specific mechanism, pattern, or causality can be shown to be, in principle, possible; for example, that optimal distances or rates of dispersal can differ between mothers and offspring (Starrfelt and Kokko 2010; Motro 1983). In this chapter we focus mainly on mathematical models where we are interested in the consequences of a set of fairly reasonable assumptions more than in making accurate predictions; i.e. models that favour generality and realism over precision. Also, we focus on reasons behind dispersal and their interactions (including feedback effects), while spending less time exploring the consequences of dispersal in non-evolutionary models. Lastly we highlight the importance of incorporating models where not only the mean effects are taken into account.

## 2.2 Dispersal and its consequences: a feedback loop

The collection of models of dispersal can reasonably be divided into those models where dispersal is primarily seen as an effect (i.e. something to be explained) and those where dispersal is seen as a cause of something else. In the first case, we are attempting to explain why the patterns of dispersal are the way they are, instead of some different pattern, by manipulating some variable other than dispersal. Models attempting to explain dispersal *per se* range from models that are tailored towards analysing movement and dispersal (e.g. Turchin 1998) to evolutionary models attempting to find general mechanisms selecting for dispersal-enhancing traits.

In the second type of model, the goal is not to explain dispersal but to examine the consequences of it; i.e. by manipulating dispersal propensities and studying their effect on something else. In such models, the ecological or evolutionary pattern to be explained can be, for example, a large-scale ecological pattern such as the species distributions described by island biogeography (MacArthur and Wilson 1967). Other examples include the study of species ranges (e.g. Holt and Keitt 2005; Holt 2003),

*Dispersal Ecology and Evolution*. First Edition. Edited by Jean Clobert *et al.*

or population persistence (e.g. Hanski 2001); both can be studied either in a temporally invariant world or in a changing one—the latter often referring to climate change. Finally, spatial distributions of populations (e.g. Mueller and Fagan 2008) can also be the pattern to be explained, and often such work considers the evolutionary dynamics of traits other than dispersal (e.g. Rice and Papadopoulos 2009). Dispersal in such contexts is often assumed to be a population-level trait that does not evolve: for example, in their study of pollen and seed flow and their effect on species range expansion, Hu and He (2006) considered selection on viability loci but dispersal was a non-evolving trait—for other similar studies of local adaptation and plasticity, see for example, Sultan and Spencer (2002), and for effects on other traits such as helping and harming conspecific individuals, see for example, Johnstone and Cant (2008).

This leads us to our third and, in our opinion, extremely important viewpoint. Dispersal can produce ecological patterns, but these patterns can again influence the selective pressures on dispersive traits. Only by closing this loop, that is, by realizing that dispersal can at the same time act as both a cause and an effect, will we get the full picture of the evolutionary ecology of dispersal. This principle applies whether or not dispersal is considered the only evolving trait or whether it co-evolves with other traits such as cooperation (Le Galliard *et al.* 2005) or some trait influencing fitness in a spatially and temporally heterogeneous landscape (Blanquart and Gandon 2011). This, of course, makes the study of interacting causes of dispersal challenging, and most models attempt to improve our understanding of the feedback by considering one or a few factors at a time.

A good example is the study of Blanquart and Gandon (2011), who considered two traits (two loci), the first of which influences a fitness-related trait such as the immune response to a parasite, and the second controls dispersal. Spatial variation in the environment then creates local adaptation (since locally unfit individuals produce fewer offspring). The effect of local adaptation that is easiest to comprehend is that it selects against dispersal: the very existence of an individual in its natal patch implies that its parents must have been fit enough to produce young. Given that traits are heritable and environmental challenges are autocorrelated spatially as well as temporally, dispersing elsewhere is likely to lead to lower fitness than staying in the natal patch (Hastings 1983; McNamara and Dall 2011).

However, when there is temporal variation, the story is considerably more complex, as some dispersal can be selected for despite local adaptation (Blanquart and Gandon 2011). Simultaneously, dispersal itself can increase the population-wide level of local adaptation, measured as the difference between the mean fitness of subpopulations in their native environment *versus* elsewhere (i.e. expected fitness if these subpopulations were transplanted to another deme at random). Dispersal has this effect because it provides a flux of individuals with different genotypes that local selection can act on; without genetic variation there can be no response to selection, and thus no adaptation. The net result is complicated, but in a nutshell, selection for more dispersal can increase local adaptation until the linkage disequilibrium between the two alleles (philopatric individuals are more adapted to local conditions) is strong enough to select against further dispersal, leading to a predicted rate of dispersal that depends on the timescale of environmental fluctuations (Blanquart and Gandon 2011).

This is a beautiful example of eco-evolutionary feedback, but as noted by the authors themselves, their analysis ignores many forces known to promote dispersal: for example, another type of determinant of local fitness is the degree of inbreeding of the offspring, which obviously can depend on the distance dispersed (assuming that mating occurs after dispersal). Since the relatedness structure of a population depends on dispersal, selection for dispersal will co-evolve with this structure (e.g. Gandon and Rousset 1999; Lehmann and Perrin 2003). What happens when 'everything occurs simultaneously and interacts with everything'? This is a difficult question to answer, because our understanding of complex systems is not much advanced by throwing every conceivable causality into a model and staring at the outcome. There is probably no alternative to first understanding each mecha-

nism on its own, before joining two (and then maybe more) into a joint model to see how predictions change (Gandon and Michalakis 2001). To some extent, this is how the field indeed has progressed.

## 2.3 Ultimate and proximate factors in explaining dispersal

There are two very distinct answers to the question 'what causes dispersal'. These are most easily viewed by recapping Niko Tinbergen's (1963) four questions which he instructed scientists to ask about any animal behaviour. The questions, loosely based on Aristotle's four types of causes, can be exemplified as:

1. Proximate:
   a. Mechanism. What stimuli elicit the behaviour?
   b. Development or ontogeny. How does the behaviour change with age or develop in a given individual?
2. Ultimate:
   a. Phylogeny. How does the particular behaviour compare with similar behaviours in similar species?
   b. Adaptation or function. How does the behaviour affect the individuals' chances of survival and reproduction?

The major part of the theory of explaining dispersal has dealt with the last ultimate question. Dobzhansky's statement that 'nothing in evolution makes sense, except in the light of evolution' (Dobzhansky 1973) is an example of how ultimate thinking should always guide our quest for answering all of these questions. We would like to emphasize, however, that in evolutionary models the converse is also important; understanding how ultimate causes influence dispersal evolution may require some knowledge about how the dispersal strategy develops and is triggered. For instance, whether the development of a dispersive morph is under parental or offspring control (i.e. how dispersive behaviour develops), changes the predictions of ultimate models of dispersal rates (Motro 1983) and distances (Starrfelt and Kokko 2010). Likewise, since dispersal strategies can be dependent on the state of individuals such as age (Bowler and Benton 2009) or condition (see Chapter 11), evolutionary trajectories may depend on how the dispersive traits develop in the individual organism. Dispersal strategies that are conditional on some attribute of the subpopulation in which individuals find themselves can also show markedly different patterns of movement (particularly invasions) than unconditional strategies (e.g. Travis *et al.* 2009). Thus, a too explicit focus on ultimate questions only, without information of proximate processes, could fail, and we therefore briefly review proximate factors now before continuing to ultimate factors.

## 2.4 Proximate factors

In the modelling literature, proximate factors are included in both studies where dispersal is seen as an explanation and in the literature on explaining dispersal (see, for example, Tables 1 and 2 in Bowler and Benton 2005). Proximate factors must induce movement of an individual, and often the organism makes use of these factors in a way that makes adaptive sense: for example, female parasitoid wasps *Cotesia glomerata* are observed to leave their natal patch disproportionately often when the local mating opportunities are confined to inbreeding (Ruf *et al.* 2011). Similarly, ontogenetic changes in dispersiveness are often argued to be adaptive; e.g. the greater mobility of young versus old individuals can be seen to be an example of the 'asset-protection principle' (Clark 1994)—the young should be less risk averse than old individuals, since they do not yet have an established home range or territory, they have less to lose by moving. But dispersal can also be triggered at any age, e.g. by breeding failure of self and/or neighbours (Rioux *et al.* 2011). This is called the 'win-stay, lose-switch' strategy (Switzer 1993). There appears to be little work that attempts to connect such adaptive rules of breeding dispersal to a more general understanding of why, ontogenetically, individual dispersal propensities decline with age: early work by Johst and Brandl (1999) addresses this issue, suggesting that the age structure of a population is important, but this modelling work has been followed up only scantily.

Thus we do not know much about what factors determine the relative frequency of natal *versus* breeding dispersal.

Against the general impression that proximate cues of dispersal often serve to trigger movements that prove adaptive, it is also healthy to remember that dispersal of organisms often is not 'intentional' in this sense. A flock of birds displaced by a storm during migration can potentially establish a new breeding area; fieldfares breeding in Iceland are a potential example. If all non-intentional movement (currents, winds, seed carrying by animals, etc.) occurred completely irrespectively of traits of individuals, the evolutionary impact of such effects might remain limited, although even then the spatio-temporal structure of the populations would be influenced by displacements, and this will have feedback effects back to evolutionary traits. Importantly, and adding a direct component to such indirect effects, physical forces often displace individuals differentially depending on the morphology as well as behaviour of individuals. Migrant bird species, for example, show longer dispersal distances than year-round residents living in the same area (Paradis *et al.* 1998). Likewise, within a species, more mobile individuals might forage and search for mates more efficiently, but such individuals can also end up exiting the local habitat patch more often, forcing them become dispersers (especially in small organisms such as butterflies that are easily displaced by wind; Haag *et al.* 2005; Niitepõld *et al.* 2009).

It may be as difficult to disentangle 'voluntary' and 'involuntary' dispersal decisions in such cases as it is to ask whether the shape of the dispersal kernel is the precise 'intended' (i.e. adaptive) result when temporally varying wind conditions interact with the wing shape of seeds. For the purpose of deriving evolutionary and ecological predictions, luckily, this does not matter. The spread of organisms over space results from the trait distribution interacting with the conditions found in the environment, and the evolution of traits proceeds according to the expected dispersal kernel predicted by a trait (see Chapter 16). As the above examples show, it is important to be aware of the proximate mechanisms by which a certain trait produces a particular type of dispersal kernel. Ultimate explanations (selection) will be based on this kernel (the outcome of dispersal; see Chapter 16), while the evolving trait may be a morphological or behavioural characteristic. Most models of dispersal evolution lump these two together. The newly emerging field of 'movement ecology' shows some promise in making the link much more explicit (Jordano 2011; Nathan *et al.* 2008; Mueller and Fagan 2008; Kesler *et al.* 2010), but progress in this area is yet to be linked to evolutionary theory on dispersal.

## 2.5 Ultimate factors

As stated earlier, evolutionary models of dispersal typically do not let the morphological or behavioural characteristics and the consequent movement rules evolve. Instead, they simplify the world by considering that the evolving trait is either a rate of dispersal (usually defined as the proportion of young that leave their natal patch) or a parameter describing the dispersal kernel (e.g. the mean distance in an exponential distribution). The focus away from proximate explanations is that this allows the models to concentrate on the basic and general conundrum: why do individuals show adaptations that enhance movement even though movement is risky? Risks may involve a combination of energetic costs and risks of moving through unfamiliar matrix habitat, as well as not easily finding a new suitable place to settle and breed. In addition, under spatial variation, natal patches yielding many potential dispersers will be better breeding sites than average, which selects against dispersal (McNamara and Dall 2011).

Since Hamilton and May (1977), it has been known that kin selection offers a powerful explanation: by alleviating competition for resources and thus enhancing the reproductive success of kin that do not move, it can select for dispersal even if the environment is temporally and spatially invariant. Of course, spatio-temporal variation often exists in the environment and it too can select for dispersal (Crespi and Taylor 1990; McPeek and Holt 1992; Doebeli and Ruxton 1997; Heino and Hanski 2001); this category includes the study and effects of range margins (Dytham 2009). Density dependence

similarly creates spatio-temporal variation among habitat patches that can favour dispersal (Olivieri *et al.* 1995). Compared to these factors, inbreeding avoidance is a relatively distinct mechanism that can select for dispersal. It is often argued to lead to a sex bias in dispersal, because for inbreeding to be avoided, it is sufficient for one sex to disperse (Motro 1991; Perrin and Mazalov 1999; Lehmann and Perrin 2003).

Naturally, all these factors can interact, and much theoretical progress has focused on predicting evolutionary trajectories when several causal routes to dispersal are considered simultaneously. To mention examples, Ozaki (1995) and Gandon and Michalakis (1999) consider kin competition in scenarios that include spatio-temporal variation, and Parvinen *et al.* (2003) include density dependence, demographic stochasticity, and temporal variation in habitat suitability in their model. An interesting feature of such work in general is that verbal explanations of models may focus on only a subset of factors that are at work in the model. A good example is provided by kin competition. This potential route to positive dispersal rates is present whenever related individuals have the chance to interact locally: for example, they may potentially breed in the same patch but there is within-patch density-dependence, which predicts either that not all attempts to gain a breeding site within a patch will be successful, or the breeding success of all local breeders declines with local density. Clearly, there then exists a component of indirect fitness that increases by dispersing. However, a model that is not explicitly framed in terms of inclusive fitness—it might be formulated as an individual-based simulation of births and deaths—may have its author emphasizing the effects of density-dependence or temporal or spatial variation, although kin effects have implicitly (and correctly) been incorporated. This makes it somewhat challenging to read the literature on the various causes of dispersal, although there are attempts to tease apart the relative influence of different causes (Poethke *et al.* 2007).

As stated earlier in this chapter, there may be no shortcut to the hard work of first considering each effect in isolation and building a more complex system of interacting forces thereafter (Gandon and Michalakis 2001). In this endeavour it may prove helpful to strive towards a conceptual framework where all the factors that favour the evolution of dispersive traits are seen as manifestations of a single cause of dispersal as an adaptation. This single cause is variation in expected genotypic fitness over space and/or time. Such variability, in turn, can come about through four different mechanisms (or factors): variation in inclusive fitness over space (avoidance of kin competition), externally varying environmental conditions (and hence fitness) over space and time, variability in conditions over space and time driven by the population itself (e.g. demographic stochasticity), or variation in fitness due to relatedness structure of the population. The last fact may include kin interactions in a non-mating context (cooperation among kin) but also reduced survival or fecundity of inbred offspring, leading to avoidance (or tolerance, Kokko and Ots 2006) of inbreeding.

The conditions for these general mechanisms to select for dispersive traits differ slightly. For dispersal to be favoured over philopatry through the first three mechanisms, we minimally require an assumption of local competition for resources. If competition is global (that is, if competition between individuals is independent of spatial position), there is no general incentive to move at all, since it will by definition not affect an individual's chance of reproductive success. For dispersal to evolve to avoid negative fitness consequences of inbreeding, competition need not be local, but mating must be, so that moving decreases the chance of mating with kin, and hence the probability of reduced fitness of inbreed offspring.

Any adaptation will be affected by both costs and benefits, and there are a number of factors that will reduce the fitness of a dispersing individual/genotype compared to a philopatric one. Earlier, we have commented on the fact that evolutionary dispersal models tend to lump the entire movement process into one variable such as 'dispersal rate'. While this has the advantage of keeping the model simple, a better understanding of selection on actual dispersal-enhancing traits will require considering the movement process in more detail. It can be instructive to divide dispersal into the

three different phases of emigration, inter-patch movement, and immigration together with the corresponding potential fitness-reducing factors. For the emigration phase, there might be costs associated with developing the phenotypic mechanism of dispersal; often models posit a trade-off between dispersal abilities and survival or fecundity, though this might not be empirically grounded in all systems (Hanski *et al.* 2006). There are also several factors that can increase the expected fitness in the natal patch compared to other patches, therefore favouring philopatry: loss of kin cooperation and familiarity with the natal territory are potential examples that can be viewed as costs paid by dispersers that relate to the decision to emigrate. The transfer phase (i.e. movement of individuals through unsuitable matrix habitat) has its own costs, as individuals may experience a direct mortality risk while dispersing, in addition to a probability of not finding a suitable habitat at all. This last factor relates to success as an immigrant, although the difficulties of finding suitable vacant breeding habitat obviously can interact with mortality risks as such difficulties can prolong the phase of moving through matrix habitat (e.g. McCarthy 1999).

In addition to understanding the effects of these ultimate factors alone and in concert, there is yet another perspective on the multicausal nature of dispersal, and in fact evolutionary change in general (Rice 2008; Rice and Papadopoulos 2009); these factors can affect all moments (i.e. means, variances, skewness, etc.) of fitness distributions. For instance, the degree of kin competition among individuals in a patch will not be exactly equal for all individuals, but can be seen as a distribution of competitive impacts of kin, with a mean, variance, etc. In most analytical models, assumptions are made so as to capture the effects of the mean of such distributions, mostly by assuming infinite population sizes or number of patches making the higher moments negligible. Another example is that of mortality risk during the movement phase of dispersal. Assuming that the number of dispersers is very large, one disregards the probabalistic nature of this risk: a given fraction of the dispersers are assumed to die instead of focusing on how this risk leads to a distribution of individuals either surviving or dying, with an accompanying degree of variation. In general it is very difficult to derive how these fitness distributions at the level of the individuals have an effect on the fitness at the level of genotype(s), and one of the major challenges of modelling the evolution of dispersal lies here; many approaches (e.g. the direct-fitness method) do not take this variability into account and only deal with the mean fitness of individuals (and genotypes) (Ronce *et al.* 2001; Ronce 2007). This also contributes to the difficulty of linking individual-based models, where the effects of the full distribution of individual fitnesses will be taken into account, to more analytical methods focusing on mean effects only.

## 2.6 Dispersal homeostasis

The distribution of individual fitness, particularly the higher moments, depends on what proximate mechanisms of dispersal decisions are incorporated in a model. As an example of how these higher moments can affect the evolution of dispersal, we will now construct a simple individual-based model mimicking the classic direct-fitness treatments of Frank (1986, 1998). For a related model, see Lehmann and Balloux 2007.

The model considers dispersal homeostasis, by which we mean that for a given dispersal trait $d$, a focal mother will divide her clutch into a dispersive fraction ($d$), and a philopatric fraction ($1–d$). This differs from letting each offspring develop into a dispersive morph with probability $d$ not only in that it assumes a different developmental process, but also in that this latter option introduces variance among broods in the number of dispersers. We refer to these two options as homeostatic and randomized dispersal, respectively. Our aim now is to mimic the simple direct-fitness model of dispersal to evaluate if there is potential for dispersal homeostasis to perform better than randomized dispersal in our simple system, despite the mean dispersal rate being the same for both.

We posit $P$ patches, wherein one asexual individual can give rise to offspring whose dispersal phenotype she determines. Under the homeostatic interpretation of $d$, all individuals of genotype $d$ will always produce $n \times d$ dispersing offspring (as

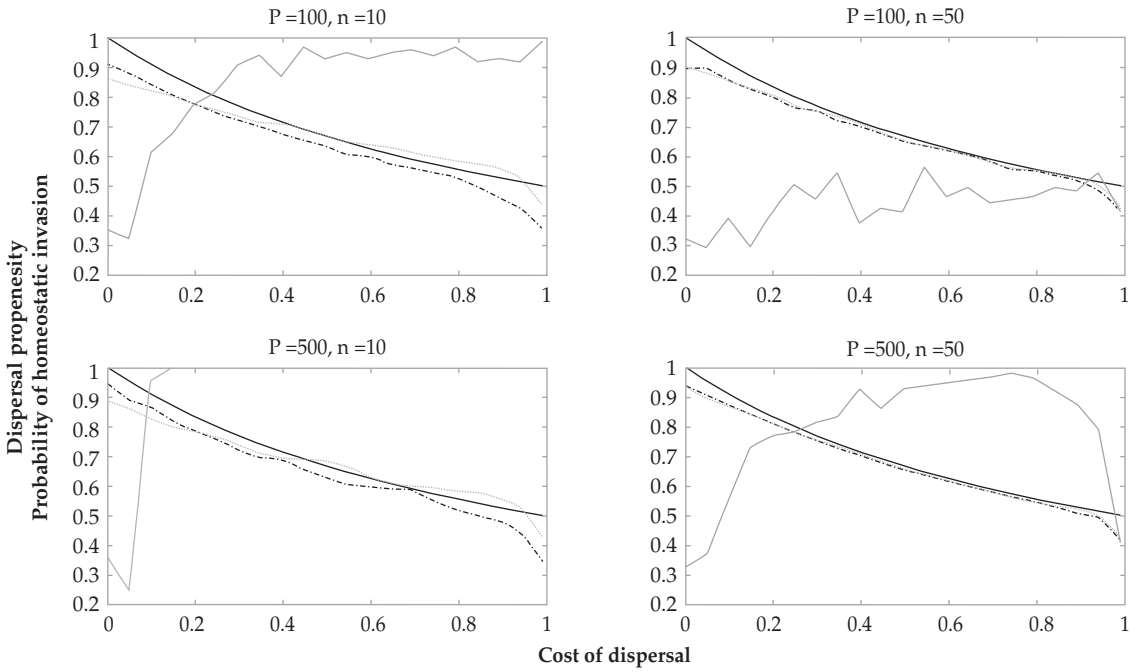


**Figure 2.1** Consider *P* patches where one sole individual can breed. This individual is every generation chosen randomly among all the philopatric individuals from last generation and immigrants from other patches. Each reproducing individual gives rise to *n* offspring, each of which either develops into a dispersive phenotype and disperses, or stays philopatric. The mother dies after reproduction. The maternal allele at a haploid locus determines either the probability that each of her offspring will disperse (randomized model) or the fraction of her offspring that will disperse (homeostatic model) to a randomly chosen different patch. We therefore only consider allelic values $d \in \left\{\frac{0}{n}, \frac{1}{n}, \frac{2}{n}, \ldots \frac{n}{n}\right\}$. For each birth there is a small probability (1/500) that the allele will mutate to a randomly chosen different value. For the dispersing phenotype there is a probability of mortality (c) during the movement phase which varies between simulations from 0 to 0.99. The mean allelic values for 100 simulations for each cost setting are shown for the randomized model (dotted line) and the homeostatic (dash-dotted line). The predictions from the analytical treatment of Frank (1986, 1998) are given by the full black line.

For comparing the two different types of phenotype determination or development, the population is initialized with randomized dispersal monomorphic for the optimal dispersal propensity predicted by the direct-fitness model and there are no mutations in the dispersal propensity. After 500 generations, individuals have a slight probability (1/500) of changing to a homeostatic determination for 50 generations. We then let the simulations run until one of the types have outcompeted the other. The grey line in the figure shows the fraction of 100 replicates in which the homeostatic dispersal strategy outcompeted the randomized one, despite the fact that the mean fitness of these two strategies are identical.

well as n ×(1–*d*) non-dispersing offspring). Under a randomized view, each individual of genotype *d* will produce a distribution of dispersing offspring that has the mean $n \times d$ and variance $n \times d \times (1 - d)$. Since the mean number of offspring, and therefore the mean fitness of mothers, is the same for both of these setups, we can detect the effect of higher moments of the distribution by comparing evolution under randomized and homeostatic determination of dispersal.

Our individual-based model has the same setup as the direct-fitness model of Frank (1986, 1998). In our first runs of the simulations, we vary the cost of dispersal from 0 to 0.99 under both homeostatic and randomized dispersal (see legend for Figure 2.1 for details). After 2000 generations, the mean dispersal propensity under both assumptions conform fairly nicely to the direct-fitness predictions (Frank, 1986, 1998), at least for moderate costs (Figure 2.1). The simulation results are also closer to these predictions when we increase both the number of patches and the number of offspring, which is an expected pattern since both are assumed to be large in the direct-fitness approach. Thus, these simulations

provide reassurance that our stochastic individual-based implementation can reproduce the basic results from the direct-fitness model.

To investigate the potential for selection for homeostatic dispersal, we can now initialize simulations with randomized strategies where $d$ equals the predicted optimal dispersal propensity from the direct-fitness model, and see if a homeostatic strategy with the same dispersal propensity $d$ will invade and take over the population.

When comparing the two different proximate mechanisms (homeostatic and randomized dispersal), a useful baseline is provided by setting the mortality cost of dispersal to zero and the dispersal propensity to 1. With this setting, there is no difference between the two different proximate mechanisms of dispersal, since 'dispersing with probability 1' and 'dispersing the whole clutch' both imply that all offspring develop as the dispersive morph. In this particular setup, a neutral allele (use of homeostasis instead of randomized dispersal) grows to fixation with probability between 0.3 and 0.4. For all the other dispersal propensities, the invasion probability of a homeostatic determination of dispersal morphs is higher (Figure 2.1). This is a clear indication that, even though both proximate interpretations of the dispersal propensity in the direct-fitness model are valid, they can be teased apart in our model, and that homeostatic (i.e. with completely deterministic proximate causality) dispersal strategies are favoured over randomized ones (i.e. with probabilistic proximate causality). Since the mean (or expected) fitness of the parents in these models are identical, homeostatic dispersal strategies can evolve through their effect of the higher moments of the fitness distributions. In general, all moments of a fitness distribution contribute to evolutionary change (Rice, 2008), and thus the fact that different proximate mechanisms of dispersal can imply clear differences in these distributions appears as understudied as the linking of movement rules to evolutionary change in general.

## 2.7 Summary

Our chapter highlights that dispersal can be seen as multicausal in many different perspectives. It is multicausal in the sense that researchers may be interested in what causes dispersal, or dispersal as a cause of other ecological or evolutionary patterns and processes—or, ideally, the feedback between these two. It is also multicausal in the sense that we often both choose to, and have to, deal with different notions of causality. The decision for a particular individual to emigrate, alter tactics in the movement phase, or when and where to settle, can all occur both in a probabilistic and a deterministic way. Dispersal is also multicausal in the sense that the ultimate 'factors' identified as selecting for dispersive traits (spatio-temporal variability, avoidance of kin competition, and inbreeding) affects not only the mean fitness of individuals and genotypes, but also the higher moments. Finally, the multicausal nature of dispersal relates to how we explain dispersal patterns; we can do so by referring to either proximate and ultimate factors, both of which can be either deterministic or probabilistic. We suggest that a tighter link between the proximate and ultimate two would bring the field forward. We thus urge more explicit treatment of proximate mechanisms (developmental and mechanistic) in ultimate models, both to study the effects of including different proximate causes of dispersal, but also to clarify the full impact on the fitness distributions and evolutionary consequences of these.

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