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Group living features can challenge predictions for the evolution of altruistic behaviors

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Abstract

Altruistic behaviors occur when an individual decreases its personal fitness to help another individual. Such behaviors occur across a range of species and environments, and they take different forms. The diversity of altruistic behaviors is also characterized by various group living features, including group structure and social interactions. In this thesis, I develop models to study how the specificities of group living can influence the evolution of altruistic behaviors. I use inclusive fitness models to understand how the social environment the group creates, the ecological factors, and the benefits of altruistic behaviors impact the evolution of social behaviors. In the first model, I study the evolution of delayed dispersal with group size benefits. Dispersal tends to be delayed when breeding opportunities are scarce, i.e., when the habitat is saturated. I find that habitat saturation is not always associated with a high level of dispersal. This finding challenges previous results and highlights the need to model environmental feedback explicitly. In a second model, I measure how redirected help can emerge when individuals disperse near their relatives. Redirected help happens when an individual whose entire brood fails reallocates the effort it would have expended on parental care to help a related neighbor. The adaptive significance of this strategy may look straightforward, but if the population is viscous, the helper also competes with its relatives. This population viscosity creates additional costs and benefits that can restrain the evolution of altruism. To investigate the evolution of redirected help in a viscous population, I use an infinite-island model where redirected help can provide survival or fecundity benefits to the recipients. I find that the survival benefits associated with redirected help sometimes promote the emergence of help better than fecundity benefits, which contradicts previous

findings. In a third model, I delve more deeply into the evolution of redirected help by explicitly accounting for spatial structuring within the population. I find that switching to a spatially explicit model has repercussions for the evolution of redirected help. For instance, the influence of offspring dispersal on the evolution of redirected help is reversed between the two models. My findings highlight the impact of spatial structure on the evolution of social behaviors. Overall, my thesis shows that different group living features can challenge predictions on the evolution of social behaviors.

Keywords: kin selection, altruistic behaviors, kin competition, spatial structure, game theory, mathematical model

Summary for lay audience

In nature, some animals diminish their reproduction or survival to help another individual. These animals display what is referred to as altruistic behavior. This puzzling behavior is common in many species and environments and takes many forms. For example, some individuals help defend a nest, and some will help feed the offspring of another. Displaying these behaviors does not seem to be the best strategy to adopt. Why would an individual help another individual at its own expense? This question has long puzzled scientists, and many answers have been proposed. One of these relies on the fact that family members share some genes. By helping a family member, the helper increases the representation of its genes in the next generation. When studying a species in the wild, it can be hard to determine if altruism is truly adaptive. Every species has its unique life cycle, is possibly subject to a specific set of environmental conditions, and sometimes engages in elaborate social interactions. These details influence the selective advantage enjoyed (or not) by altruism. In this thesis, I study how features of a species' life cycle and environment can influence the evolution of altruism. I find that different features inherent to the group organization, such as ecological feedback and spatial structure, can challenge previous predictions about the evolution of altruistic behaviors. For example, the conditions favoring the evolution of altruism can change depending on the spatial structure or the benefits provided.

I, Alan Flatrès, declare that this thesis titled "Group living features can challenge predictions for the evolution of social behaviors" has been written by me under the supervision of Dr. Geoff Wild.

Chapter 2: This chapter is a paper co-authored by G. Wild, and published as in Theoretical Population Biology. It is included here under the "Author Rights" set out by Elsevier Ltd. in the publishing agreement signed by AF and GW. AF developed the model, performed analysis of the model, and drafted the manuscript with input from GW.

Chapter 3: This chapter is in revision for the journal Evolution and is coauthored by G. Wild. AF developed and performed the analysis of the model, and drafted and finalized the manuscript, with supervisory input from GW.

Chapter 4: This chapter is being prepared for submission for publication with coauthor GW. AF developed the framework, performed the analysis of the illustrative examples, and drafted the manuscript with supervisory input from GW.

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Chapter 1

Introduction

1.1 Altruism as a social behavior

Cooperative behaviors are described using various definitions. We need to define this type of social behavior properly in order to study it. In this thesis, I consider social behaviors between two parties: the actor performing the behavior and the recipients who bear the consequences of the behavior. Note that a recipient can also be an actor. I can now differentiate the type of behavior depending on the benefits or cost it provides to the actor and the recipients. I define fitness as the average number of offspring an individual produces. The effect of the behavior, negative or positive, can then be measured as the effect on the fitness of the recipient and the actor. In regards of my thesis, I use Taylor and Frank [90] chain rule decomposition of inclusive fitness [38] to characterize the inclusive fitness change, which considers the change in the fitness of the actor and the recipients

$$BR + C, \tag{1.1}$$

where B denotes the effect for the recipient, R is the relatedness (i.e., the genetic link) between the actor and the recipient, and C is the effect of the behavior for the actor. In nature, distinguishing the effects of social interactions can be difficult to separate. For example, one interaction's benefits can compensate for another's cost.

Following the terminology of Bourke [3], I separate behaviors into four categories. The first one benefits the actor and the other recipients: $B > 0$, $C > 0$; this is called cooperation or mutualism. In this situation, both the actor and the recipient produce, on average, more offspring; this is the top left corner of Table 1.1. In the second category (top right corner of Table 1.1), the actor has benefits at the recipient's expense: $B < 0$, $C > 0$. This is referred to as selfishness; in that case, the actor has more offspring on average while the recipient has less. In the third category (bottom right corner), the actor pays some cost to harm the recipients: $B < 0$, $C < 0$. This is referred to as spite. In that case, both actors and recipients have, on average, fewer offspring. Finally, when the actor pays some cost to give benefits to the recipients: $B > 0$, $C < 0$. We referred to it as altruism (bottom left corner). The evolution of cooperation and selfishness can be understood straightforwardly, as it benefits the actor. However, why would an individual bear the cost of helping another individual, i.e., by behaving altruistically?

In this thesis, I focus on the evolution of different altruistic behaviors in various scenarios.

1.2 Models of evolution of social behaviors

The evolution of social behaviors can be studied mathematically using evolutionary models. One very common tool used in theoretical evolution is

		Effect on the recipient(s)	
		+	-
Effect on the actor	+	Cooperation	Selfish
	-	Altruism	Spite

Table 1.1: Classification of social behaviors based on the effect on the fitness of the actor (C) and on the other recipients B.

neighbor-modulated and inclusive fitness models [35, 28]. In these models, we consider the fitness change for the actor and the other individuals impacted by the change of behavior, i.e., the recipients. As some of these recipients share some genes with the actor, changing their fitness changes the number of copies of these genes passed to the next generation. Impacts on recipients are weighted by, among other things, measures of kinship. The fitness is then inclusive as it includes the fitness of different individuals. I will describe neighbor-modulated and inclusive fitness using a method previously developed [90] [see also 78, 92].

Focusing on an individual (focal individual), I now denote x as the genotypic value (continuous) controlling the behavior. I use $y = y(x)$ as the phenotypical value of the actor, $z = z(x)$ as the recipient's phenotypical value (continuous) and $\hat{z}$ as the phenotypical value of the average individual in the population. The variable y (respectively z) describes then the level at which the actor (respectively the recipient) expresses the behavior. For example, y can give the probability that the actor is helping to feed the offspring of another individual, namely the recipient, and z is the probability of the same behavior by the recipient. The fitness of the focal individual (actor) is then $W(y, z)$, where I suppose that W is a continuous function. Changing the

genotypic value x gives

$$\frac{dW}{dx} = \frac{dW}{dy}\frac{dy}{dx} + \frac{dW}{dz}\frac{dz}{dx}. \tag{1.2}$$

The values $\frac{dy}{dx}$ and $\frac{dz}{dx}$ are the slopes of the phenotypical values on the genotypic value of the actor. We can replace them with the statistical regressions of the phenotypical values (y and z) to the genotypical value x equal to $cov(y, x)$ and $cov(z, x)$, respectively. We then evaluate this expression when $y = z = \hat{z}$ as we compute the fitness change of changing the behavior $y(x)$ and $z(x)$ from the initial population $\hat{z}$. Dividing by $cov(y, x)$ we obtain

$$\frac{dW}{dx} \propto \Delta W(\hat{z}) = \frac{dW}{dy}1 + \frac{dW}{dz}R \tag{1.3}$$

where 1 is the relatedness between the actor and itself, and $R = \frac{cov(z,x)}{cov(y,x)}$ is the relatedness between the actor and the recipient (non-actor) [65, 68]. As the actor can also be a recipient, the term $\frac{dW}{dy}$ is equal to the effect the actor has on itself. Equation (1.3) gives the neighbor-modulated fitness change of changing genotypic value x.

Equation (1.3) can be seen from two different points of view, depending on whether we look from the actor or recipient perspective. First, in a recipient-centered view, we can interpret the fitness change as the change of fitness of the actor given its own change of behavior and of its relatives. The change in the relatives' behavior is proportional to the relatedness between the actor and the relatives. This recipient-centered view is the neighbor-modulated fitness [28]. Looking at the actor-centered view, Equation (1.3) sums the fitness changed of each recipient given the change of behavior of the actor. Each recipient's fitness change is weighted by the relatedness between the actor and

the recipient. This actor-centered view gives the inclusive fitness change [28].

As a first simple example, we can consider an altruistic behavior that decreases the actor's fitness by $C > 0$ and increases the recipient's fitness by $B > 0$. We have

$$\frac{dW}{dx} \propto -C + BR, \tag{1.4}$$

which corresponds to Hamilton's rule for altruistic behavior [36]. This rule tells us that the inclusive fitness change experienced by the helper is the sum of its personal cost, weighted by a relatedness coefficient of 1 and the fitness change B of the recipient, related to the helper by R. Assuming a selfish population, when Equation (1.4) is positive, that is when the benefits surpass the cost of helping, altruism can emerge. When Equation (1.4) is negative, that is when the benefits are lower than the cost, altruism cannot emerge. Inclusive fitness change explain us how altruism can emerge in a population.

The simplicity of Hamilton's rule makes it appealing, but some limitations on the evolution of altruism can occur if individuals interact with and compete with their relatives. This situation can arise when dispersal is either slow, occurs over on short distances, or only partially. This dispersal features leads to population viscosity: individuals remain close to their relatives if they disperse locally. This population viscosity can create new costs and benefits when an individual helps another with whom they are in competition. Let's, for example, consider the simple case developed by Taylor [88]. This model considers an infinite population subdivided into patches of N breeding spots. We suppose this population is non-overlapping (individuals die at the end of their first year), asexual, and haploid. The population's life cycle starts with each individual on a breeding spot interacting with another individual on the same patch, chosen randomly among all the other patch members, including

the actor itself. Without receiving or giving help, an individual will have M offspring. If they help the other individual, they will pay a cost by having $-C$ time less offspring offspring. In exchange, the recipient will have B more offspring. The recipient, and so its offspring, is related by a coefficient R to the helper. The parental (F0) generation dies, and the offspring disperse with probability d or stay on their natal nest with probability $1-d$. Finally, offspring compete for a breeding spot on the patch they are found. There are $B-C$ extra offspring, which will displace $(1-d)(B-C)$ local offspring if they compete on their natal patch with probability $1-d$. As those displaced offspring are born on the same patch as the helper, they are related to them by the relatedness coefficient R. I denote by y the level of help expressed by the actor, and z, the level of help expressed by the recipient. Hence, the fitness is equal to

$$W(y,z) = (M - Cy + Bz)\Bigg(\underbrace{\frac{d}{N(M+(B-C)\hat{z})}}_{\text{Dispersal}} + \underbrace{\frac{1-d}{Nd(M+(B-C)\hat{z}) + N(1-d)(M+(B-C)z)}}_{\text{Local}} \Bigg), \quad (1.5)$$

where $\hat{z}$ denotes the average helping rate in the entire population. Differentiating $W(y,z)$ with respect to genotypical value x, and evaluating the expression as $y = z = \hat{z}$, we obtain

$$\frac{dW}{dx} = \left(-C + B\frac{dz}{dx} - (1-d)^2(B-C)\frac{dz}{dx} \right) \frac{1}{N(M+(B-C)\hat{z})}. \quad (1.6)$$

This gives, using the relatedness coefficient $R = \frac{dz}{dx}$, and removing the right-

most positive scalar, assuming $B > C$,

$$\Delta W(\hat{z}) = \underbrace{-C}_{\text{Direct cost}} + \underbrace{BR}_{\text{Direct benefit}} - \underbrace{(1-d)^2(B-C)R}_{\text{Displaced local offspring}}. \tag{1.7}$$

The trait will spread if $\Delta W(\hat{z}) > 0$, which is equivalent to

$$\frac{B}{C} > \frac{1-(1-d)^2R}{R-(1-d)^2R} \tag{1.8}$$

The relatedness between the helper and its recipient, R is computed using the following recursion:

$$R = \frac{1}{N} + \frac{N-1}{N}(1-d)^2R \tag{1.9}$$

which can be rearranged to $NR(1-(1-d)^2) = 1-(1-d)^2R$. Using this formula in our fitness change, we obtain $\frac{B}{N} > C$, which shows that help can spread if the cost paid by the helper is compensated by giving benefit to the helper itself. In other words, the kin competition created by the population viscosity cancels the help benefits. Helping is only favored if the return expected by the actor exceeds the cost, i.e., the actor effectively helps itself with probability $1/N$.

In nature, a population can be grouped into classes, whether by sex, (male or female), or by age classes, (juveniles, adults). In that case, the benefits and costs related to help must consider the differences between the classes. Indeed, increasing offspring's survival may not have the same consequences as increasing an adult's survival. We need to consider the reproductive value of an individual in each class [24, 87]. The reproductive value of a class j is the probability that taking a gene from an individual in a distant future, this gene comes from the class j from now. Let's look at the methods presented

in [90]. Let's assume we have several classes. We first denote c_j as the class reproductive value of the class j. We also define by u_j, the frequency of the class j in the total population, and finally, ν_j, the reproductive value of an individual in the class j. It is commonly used to have $\nu_j = c_j/u_j$.

Let's assume that a class j individual produces a number w_{ij} of i-class individuals. The matrix $A = [w_{ij}]$ stores the number of individuals of the different classes produced by an individual from every class. In a monomorphic population (every individual bears the same phenotype, $\bar{z}$), the dominant eigenvalue, λ, of A gives the growth rate of the population. If $\lambda = 1$, then our population is at equilibrium. The population at equilibrium follows the class distribution given by the right eigenvector μ associated with λ. The left eigenvector ν gives the reproductive value of an individual in class j. In our monomorphic population, the average fitness is equal to the sum of the number of individuals u_i of every class, times the production of every class-j individual multiplied by the reproductive value of a class-j individual

$$W(y,z) = \sum_{i,j} \nu_i w_{ij}(y,z) u_j. \tag{1.10}$$

The inclusive fitness change associated with a change in the genotypic value x is then equal to

$$\frac{dW}{dx}|_{x=\hat{x}} = \sum_{i,j} \nu_i \frac{dw_{ij}}{dx} u_j = \nu \frac{dA}{dx} u, \tag{1.11}$$

where $\hat{x}$ denotes the genotypical value of the resident population. Vectors ν and u can change with $\hat{z}$, but they do not depend on x. Given that ν and μ are eigenvectors, the middle side of Equation (1.14) simplifies into the right side.

I now look at a concrete example of inclusive fitness change in a class-

structured population from [90]. Suppose we have a population divided into two classes: offspring and adults. Adults give birth to n offspring and survive at probability s after every time step. Offspring mature with probability m. The matrix A is

$$A = \begin{pmatrix} 0 & n \\ m & s \end{pmatrix} \tag{1.12}$$

I suppose now that an offspring can help its parent increase its fecundity by a factor B, it will decrease its maturation by a factor C. Let y be the offspring phenotype and z be the one from its parent. Let λ be the dominant eigenvalue of A, then λ corresponds to the growth rate of the population. We also have $u = (n, \lambda)^T$ and $\nu = (m, \lambda)$. An offspring helping will mature at rate $m(1 - sCy)$. This fitness depends on s as its parent needs to survive to be helped. The survival of an adult does not change with the behavior. A helped adult will produce $n(1 + snBz/\lambda)$ offspring, where s/λ gives the probability that an adult was already an adult at the time step before, and so give birth to offspring. The fitness matrix is then

$$A = \begin{pmatrix} 0 & n(1 + snBz/\lambda) \\ m(1 - sCy) & n \end{pmatrix} \tag{1.13}$$

The fitness change associated is

$$\frac{dW}{dx} = \nu \frac{dA}{dx} u = \begin{pmatrix} m & \lambda \end{pmatrix} \begin{pmatrix} 0 & n^2 sBR/\lambda \\ -smC & 0 \end{pmatrix} \begin{pmatrix} n \\ \lambda \end{pmatrix} = nsm(-\lambda C + BnR) \tag{1.14}$$

where $\frac{dy}{dx} = 1$, and $\frac{dz}{dx} = R$.

Equation (1.14) is positive when $C < BnR/\lambda$. An increase in the help level will then be selected if the cost is low and the benefit is high, but also if

the relatedness R is high, as expected from Hamilton's rule.

1.3 Convergence stability

Now that I have defined the inclusive fitness change and some of its applications, what can we say about this fitness change? Let's assume the genotypical value of the focal individual is x, its phenotypical value is $y(x)$, the phenotypical value of the recipient is $z(x)$, and the one of the resident population is $\hat{z}$, i.e., the phenotypical value of the general population. The fitness of the focal individual is then $W(y, z)$, and its fitness change is equal to $\Delta W(\hat{z})|_{y=z=\hat{z}}$. Three things can happen to the fitness change. The first case happens if $\Delta W(\hat{z})|_{y=z=\hat{z}} > 0$, then a higher level at which the behavior is expressed, y, will be selected. In the second case, a lower level is selected if $\Delta W(\hat{z})|_{y=z=\hat{z}} < 0$. Finally, if $\Delta W(\hat{z})|_{y=z=\hat{z}} = 0$, we reach an evolutionary equilibrium y^*, meaning that evolution does not change the behavior. However, this evolutionary equilibrium behavior may or may not be dynamically stable, meaning that a perturbation is corrected by selection, and the equilibrium state is restored. We then assess the stability of an equilibrium to see if another can replace this value of the behavior.

In evolutionary game theory, several stability concepts have been developed, such as evolutionary stability or convergence stability [81, 80]. In inclusive fitness models, a common assumption is weak selection, which assumes that the effect of change in fitness is small. This assumption allows us to determine the convergence stability of a strategy, but its evolutionary stability can only be assessed in certain specific cases.

I use the definitions and theorems from [86] to define the convergence stability I will use in my thesis. I assume the evolutionary equilibrium is y^*. I also

consider the function $W(y, z)$, which computes an individual's fitness given its own strategy y, the recipient strategy z, and the resident population's strategy $\hat{z}$.

The strategy y^* is convergence stable if for all resident $\hat{z}$ in the neighborhood of the equilibrium y^*, the mutant strategies that are better than $\hat{z}$ are only the ones who are closer to y^* than $\hat{z}$ is to y^*.

If W is twice differentiable, this condition can be summarized by:

$$\frac{\partial}{\partial \hat{z}} \Delta W(\hat{z}) \Big|_{\hat{z}=y^*} \leq 0, \tag{1.15}$$

where y^* satisfies $\Delta W(y^*) = 0$. This condition can tell that the local optimal strategy y^* can be reached, but it does not mean this strategy is evolutionary stable. The weak inequality is necessary for convergence stability, and the strict version is sufficient.

For an equilibrium to be evolutionary stable, we need the following conditions:

$$\frac{\partial^2 W}{\partial y^2}(y, z)|_{y=z=\hat{z}=y^*} < 0. \tag{1.16}$$

In general, this last condition may not be possible to obtain using See also [20, 19] for the formal analysis of these conditions.

I now show how an evolutionary equilibrium can be found in an inclusive fitness context. I present an example from [90], which is a simplification of [41] on the partial dispersal of offspring. I consider a population of adults living on patches with N breeding spots. I look at this population in discrete time steps. Each adult will give birth to a large number n of offspring. For a focal individual, I suppose that a fraction y of these offspring will disperse to a random patch and will survive dispersal at probability $1 - c$. I also

assume that the offspring itself control the dispersal of offspring. This focal individual's fitness (W) is given by the number of breeding spots occupied by its offspring after a time step. The average dispersal rate of other offspring on the same patch is denoted z, and the average dispersal rate of the entire population is $\hat{z}$. The fitness of our focal individual is thus

$$W(y,z) = \underbrace{(1-y)p(z)}_{\text{Local fitness}} + \underbrace{y(1-c)p(\hat{z})}_{\text{Exported fitness}}, \tag{1.17}$$

where $p(z) = \frac{1}{(1-z+\hat{z}(1-c))}$ gives the probability for an offspring to get a breeding spot on a patch where the dispersal rate is z.

The derivative of the fitness function, evaluated at $y = z = \hat{z}$, is

$$\frac{dW}{dx} = \frac{dW}{dy}\frac{dy}{dx} + \frac{dW}{dz}\frac{dz}{dx} = -p(\hat{z}) + (1-c)p(\hat{z}) + (1-\hat{z})p'\frac{dz}{dx}, \tag{1.18}$$

which simplifies to

$$\Delta W = \frac{1}{1-\hat{z}c}\left(-c + R\frac{1-\hat{z}}{1-\hat{z}c}\right), \tag{1.19}$$

where $R = \frac{dz}{dx}$ is the relatedness between the focal individual and a random offspring on the same patch.

Equation 1.19 gives the inclusive fitness change undergone by the focal individual who disperses one more offspring and retains one less. One more dispersing offspring returns $1-c$ to the fitness of the adult. However, as one less offspring remains, the adult loses 1 unit of fitness. The other offspring, related to R to the focal individual, has more opportunity to win a breeding site and gives $\frac{1-\hat{z}}{1-\hat{z}c}$, which is the probability for a local offspring to compete successfully for a local breeding site.

A higher dispersal rate will be selected if $\Delta W > 0$, which is equivalent to $R\frac{1-\hat{z}}{1-\hat{z}c} > c$. At evolutionary equilibrium, there is no inclusive fitness change, which then equals 0. This gives the following relation at evolutionary equilibrium $y = z = \hat{z} = y^*$:

$$R\frac{1-y^*}{1-y^*c} = c. \tag{1.20}$$

Note that the relatedness coefficient also depends on the dispersal rate y^*.

To find the dispersal rate at evolutionary equilibrium, y^*, we must compute the relatedness between the focal individual (offspring) and a random offspring on the natal patch. The relatedness coefficient between two individuals is equal to the ratio between the covariance of the genotypic value of the gene of interest of the two individuals and the covariance of the genotypic values of the gene of interest of the focal individual and itself [65, 68]. To compute these covariances, we define f as the inbreeding coefficient in the population (the probability that randomly chosen alleles at the locus in question are identical by descent) and g as the coefficient of kinship between two offspring born on the same patch. The values of these coefficients in the next generations are $f' = g$ and $g' = \frac{1}{N}\frac{1+f+2g}{4} + \frac{N-1}{N}(\frac{1-y^*}{1-y^*c})^2 g$. At equilibrium, we thus have $f = g = \frac{1}{4N-3-4(N-1)(\frac{1-y^*}{1-y^*c})^2}$. Now, I can compute the relatedness coefficient between two offspring born on the same patch using correspondences between covariances and f and g as defined above, such that

$$R = \frac{2g}{1+f} = \frac{1}{2N - 2(N-1)(\frac{1-y^*}{1-y^*c})^2}. \tag{1.21}$$

Solving $R\frac{1-y^*}{1-y^*c} = c$, we find

$$y^* = \frac{H + 1 - 2Nc}{H + 1 - 2Nc^2}, \tag{1.22}$$

with $H = \sqrt{1 + 4N(N-1)c^2}$. If we set $N = 1$, we have $y^* = \frac{1-c}{1-c^2}$. I will now show that y^* is convergence stable for $N = 1$. To do so, I compute the derivative of the inclusive fitness change with respect to y and evaluate this expression at $y = z = \hat{z} = y^*$

$$\frac{\partial}{\partial y}(\Delta W)\Big|_{y=z=\hat{z}=y^*} = \frac{1}{(1-cy^*)^2}\left(-c^2 + cR\frac{1-y^*}{1-cy^*} - R + cR\frac{1-y^*}{1-cy^*}\right). \tag{1.23}$$

Given $y^* = \frac{1-c}{1-c^2}$, we have

$$\frac{\partial}{\partial y}(\Delta W(\hat{z}))\Big|_{y=z=\hat{z}=y^*} = -\frac{1}{2(y^*)^2} - < 0. \tag{1.24}$$

The evolutionary equilibrium y^* is thus convergence stable.

I now look at the evolutionary equilibrium in a class-structured population. I introduce a new example presented by Taylor [90] and introduced by Hamilton [39] in which they look at the sex allocation by a female breeder. Sex allocation is defined as the allocation of resources to males or females by the parent. I consider a sexual population where N females breed on a patch. After mating, they disperse to another patch with probability μ and die during dispersal with probability k. Females then compete for a breeding spot on the patch they are found on.

I now suppose that each female gives birth to n sons and n daughters; their sons survive with probability y, and daughters survive with probability $1 - y$. We can see y as the male/female ratio. I assume this ratio is controlled by the mother, which is then the actor. As the actor is the mother and the recipients are her daughters and her sons, we must compute their fitness. The fitness of

a random female offspring is

$$W_f(y_f, z_f) = (1 - y_f)\left((1-\mu)p(z_f) + \mu(1-k)p(y^*)\right), \tag{1.25}$$

where y_f is the mother's phenotype, z_f is the average phenotype on her native patch, and y^* is the average sex ratio in the population. The probability that a female gets a breeding spot after dispersing is $p(z) = \frac{1}{n}(\frac{1}{(1-z)(1-\mu)+(1-y^*)\mu(1-k)})$.

Similarly, the fitness of a random male offspring is

$$W_m(y_m, z_m) = y_m \frac{1 - z_m}{z_m}((1-\mu)p(z_m) + \mu(1-k)p(y^*)), \tag{1.26}$$

where y_m is its mother's sex ratio, and z_m is the average sex ratio on his native patch. $\frac{1-z_m}{z_m}$ is the female/male mating ratio on his native patch.

As we have a class-structured population with female and male classes, I use the notation previously described to compute the fitness of an average individual, which equals $W = c_m W_m + c_f W_f$ at equilibrium. Again, here c_m is the class reproductive value for males, and c_f is the class reproductive value for females. If we consider a diploid species, we have $c_m = c_f$; if we have a haplodiploid species, we have $c_f = 2c_m$. At equilibrium, we have $\frac{dW}{dx} = 0$ which can be written

$$y^* F = (1 - y^*)M = 0, \tag{1.27}$$

where $y^* = y$ is the evolutionary equilibrium value,

$$M = c_m r_m - c_m R_m, \tag{1.28}$$

and

$$F = c_f r_f + c_m R_m - \left(\frac{1-\mu}{1-k\mu}\right)^2 (c_f R_f + c_m R_m). \tag{1.29}$$

The coefficient r_f is the relatedness between the mother and its female offspring, r_m is the relatedness between the mother and its male offspring, R_f is the relatedness between the mother and a random female offspring on the same patch and R_m is the relatedness between the mother and a random male on the same patch.

In equation 1.29, F gives the fitness change of having one daughter more breeding, M is the fitness change of having one more son breeding. Both terms take into account the extra fitness gained by the mother's offspring and the decreased fitness for other offspring on the same patch due to the increased sex-related kin competition.

In this specific example, it is difficult to determine the evolutionary stability of the equilibrium $x^* = y^*$. From the previous definition, if we want to check the evolutionary stability of y^*, we need to compute $\frac{\partial^2 W}{\partial y^2}$. In the case of class structured population, we obtain

$$\begin{aligned}\frac{\partial^2 W}{\partial y^2} &= \frac{\partial^2}{\partial y^2}(\nu A u) \\ &= \frac{\partial}{\partial y}\left(\frac{\partial \nu A u}{\partial y}\right) \\ &= \frac{\partial}{\partial y}\left(\nu \frac{\partial A}{\partial y} u\right) \\ &= \frac{\partial \nu}{\partial y}\frac{\partial A}{\partial y}u + \nu\frac{\partial^2 A}{\partial y^2}u + \nu\frac{\partial A}{\partial y}\frac{\partial u}{\partial y}.\end{aligned} \tag{1.30}$$

I used equation 1.11 to go from line 2 to line 3. Equation 1.11 cannot be used to simplify the last line. We thus need to determine the derivative of the eigenvectors ν and μ with respect to y. This makes it much more difficult to assess as we need to see the effect of selection on the reproductive value and the population distribution. In this thesis, I will then restrain myself to the convergence stability case.

1.4 Specific cooperative behaviors

Cooperative behaviors are common in many species, many environments, and in many different forms. For instance, you can find individuals helping to build the nest, defend against predators, or feed the offspring [3]. We also have behaviors that are not strictly speaking altruistic behaviors because either individuals do not provide active help or because this behavior is conditional. This is the case for delayed dispersal.

Delayed dispersal occurs when offspring remain on their breeding site rather than disperse immediately [35, 15]. In that case, they may lose the opportunity to breed as they require a breeding spot or a partner. Individuals who delay their dispersal can thus incur a cost of opportunity for their behavior. In exchange, by staying on their natal site, they maintain a group size that can protect against predators, increase the yield from foraging, and maybe even teach the offspring in the group [33, 34]. Note that these benefits do not need to be done actively. Indeed, a large group can protect better against predators without active protection [33, 34]. By delaying their dispersal, individuals can then bring some benefits to their relatives while paying a cost for not looking for a breeding spot. In this way, delayed dispersal may be considered cooperative behavior in the broad, Hamilton's sense.

Another behavior I present now is redirected helping. Some bird species lay eggs in brood during the breeding season. Before hatching, the eggs can be eaten by a predator or destroyed by bad weather. In that case, the breeder suffers brood failure and may be unable to re-breed after this loss. This breeder can decide to reallocate the parental resources it would have given to its offspring to the offspring of another breeder, which did not suffer brood failure.

This reallocation of parental effort is referred to as redirected helping [43, 83]. The breeder who decides to help will incur a cost because it will spend some resources for feeding offspring or defending the nest against predators. The help recipient will benefit as its offspring will be better fed or enjoy more protection. Again, this behavior may not be classified into cooperative behavior stricto sensu as it depends on the success or failure of the neighborhood. Still, it fits a broader definition as it incurs personal costs and benefits others.

Altruistic behaviors can take various forms and be exhibited in different environments. In future chapters, I will study these two examples more profoundly and examine their ecological relations with the environment.

1.5 Environment-population interactions

In some articles, the evolution of cooperative behaviors has been modeled but in a way that does not consider the environmental feedback. For example, in Kokko et al. [56], offspring can delay natal dispersal and help their dominant breeder. By staying and eventually helping, offspring can increase the group size by recruiting new members and providing survival and fecundity benefits. When offspring decide to delay dispersal, they lose the opportunity to look for a breeding spot. However, the ability of an offspring to find a breeding spot depends on a single parameter [56]. Delaying dispersal and helping offspring affects population saturation [57]. First, they change it in a short time scale. The offspring that do not disperse early do not compete directly for a breeding spot, and then the competition is decreased on a short time scale. They also increase the competition by recruiting new individuals who will compete for a breeding spot during their lifetime. Delaying dispersal and helping can then impact environmental features such as habitat saturation. Modeling ecological

feedback is thus necessary to understand the dynamics between a population and its environment.

Studying the evolution of social behaviors requires that we model the population, the environment, and the different feedback between them. Besides altruistic behavior, several interactions between individuals or with the environment occur. The interactions of altruism, competition, and reproduction must be considered when building a model.

Other model features also need to be taken into account. Spatial structures have different impacts on population dynamics [93, 42]. For instance, let's compare the effect of the cost of dispersal on the evolution of altruism when it provides survival benefits. If we use an infinite-island model, then a high cost of dispersal increases the range of cost/benefit, driving the emergence of help [91]. In a model using a stepping-stone spatial model, a high cost of dispersal decreases the range of cost/benefit, driving the emergence of help [47]. Spatial structure is thus an essential non-parametric feature that can impact the evolution of social behaviors.

1.6 Contribution of this thesis

In this introductory chapter, I presented different examples of social behaviors and mathematical tools used to study their evolution. I also introduced some specific behaviors that remained unexplored in relation to the environment.

In this thesis, I propose that group living features can challenge predictions on the evolution of altruistic behavior. I model the influence of the environment on population dynamics and social behaviors, whether through ecological feedback or the spatial structure of the population. In the following chapters of my thesis, I present three studies of the impact of group living features on the

evolution of altruistic behavior. Each chapter starts with a relevant literature review, followed by a presentation of the methods used, mainly introduced in this introduction.

In Chapter 2 of this thesis, I model the evolution of delayed natal dispersal in relation to habitat saturation and group size effect. Individuals delay natal dispersal for many reasons. There may be no place to disperse to; immediate dispersal or reproduction may be too costly; immediate dispersal may mean that the individual and their relatives miss the benefits of group living. Understanding the factors that lead to the evolution of delayed dispersal is important because delayed dispersal sets the stage for complex social groups and social behavior. Here, I study the evolution of delayed dispersal when the quality of the local environment is improved by greater numbers of individuals (e.g., safety in numbers). I assume that individuals who delay natal dispersal also expect to delay personal reproduction. In addition, I assume that improved environmental quality benefits manifest as changes to fecundity and survival. I am interested in how the changes in these life-history features affect delayed dispersal. I use a model that ties evolution to population dynamics. I also aim to understand the relationship between levels of delayed dispersal at evolutionary equilibrium and the probability of establishing as an independent breeder (a population-level feature) in response to changes in life-history details. My model emphasizes kin selection and considers a sexual organism, which allows us to study parent-offspring conflict over delayed dispersal. Using convergence stability, I show that at evolutionary equilibrium, fecundity and survival benefits of group size or quality promote higher levels of delayed dispersal over a more extensive set of life histories with one exception. The exception is for benefits of increased group size or quality reaped by the individuals who delay dispersal. The increased benefit does not change the life histories supporting

delay dispersal there. Next, in contrast to previous predictions, I find that a low probability of establishing in a new location is not always associated with a higher incidence of delayed dispersal. Finally, I find that increased personal benefits of delayed dispersal exacerbate the conflict between parents and their offspring. I discuss my findings in relation to previous theoretical and empirical work, especially work related to cooperative breeding.

In Chapter 3 of my thesis, I study the selective advantage of redirected help in a viscous population living on an infinite-island model. When a brood fails, the parent can decide to help a neighbor rear its offspring. This behavior is referred to as redirected helping and occurs in various species. The advantage of redirected helping may seem obvious, provided the individual whose brood fails helps a related neighbor: the helper at least gains indirect fitness by redirecting its parental effort. However, complications arise when considering a viscous population, where individuals remain on or close to their natal site. In such a population, individuals compete with their relatives, which dilutes the advantage of helping and may counteract it altogether. This raises the question, under what conditions can we expect redirected helping to emerge from a selfish population in a viscous population? I address this question with an inclusive fitness model that looks at the conditions for the emergence of redirected help using specific versions of Hamilton's rule described previously. This model allows overlapping generations and accounts for demographic stochasticity due to brood failure. In contrast to previous theoretical studies of species with overlapping generations, I find that helping (in this case, redirected helping) can be more strongly promoted when it provides survival rather than fecundity benefits. My result depends on the species' life history and environment. Most notably, the result is impacted by the risk of brood failure and the survival rate of breeders.

In the model of Chapter 3, I investigated how population viscosity can affect the emergence of redirected help by focusing on a population living on an infinite-island model. However, the dispersal pattern of individuals in an infinite-island model neutralizes the impact of population viscosity. In Chapter 4 of my thesis, I reestablish population viscosity by studying the evolution of redirected help using a one-dimensional stepping-stone model. In this new spatial structure, the dispersal pattern is much more constrained than the infinite-island spatial structure, reestablishing the population viscosity I am interested in. Using a Hamilton's rule argument for the emergence of redirected help, I find that dispersal acts in the opposite direction as in my previous model and that the different benefits redirected help can influence how this behavior will evolve. I discuss my results about the previous literature questioning the evolution of altruism in a viscous population.

In Chapter 5, I discuss my present results and suggest possible future work directions.

Chapter 2

Evolution of delayed dispersal with group size effect and population dynamics

2.1 Introduction

Delaying dispersal from one's natal site can be advantageous for many reasons. For example, if immediate dispersal is too costly, then by delaying dispersal, an individual may be able to exploit future opportunities [71, 82] or avoid immediate hardship [55]. Delayed dispersal also maintains the size of the natal group, which can sometimes confer group-size benefits on an individual and its relatives [40, 74].

Delayed dispersal is considered a precursor to the evolution of complex social systems [3]. These include cooperative breeding systems where some individuals delay dispersal and postpone or forego personal reproduction to help raise neighbors' offspring [35]. In fact, delayed dispersal is thought to

be the cornerstone that supports cooperative breeding [55, 98, 94]. Delayed dispersal can thus be studied to understand the link between group formation and environmental features [82, 54, 55, 15, 58].

For cooperative breeding systems in particular, the importance of the group size and group quality benefits of delayed dispersal is becoming increasingly recognized [56, 25, 52, 34, 33]. However, the theory connecting delayed dispersal and group benefits, in ways relevant to the evolution of cooperative breeding systems, is incomplete. In particular, previous theories assume either asexual reproduction or limited environmental feedback [56, 25]. Thus, we do not fully understand the evolution of delayed dispersal (and cooperation more generally) in a sexual and dynamic population. In turn, our lack of understanding means we do not fully grasp the consequences of the life-history changes that inevitably accompany group benefits [52]; nor do we fully grasp the extent to which group benefits change parent-offspring conflict over delayed dispersal [96].

In this paper, we fill gaps in the theory surrounding group size benefits and their connection to delayed dispersal. We build a sexual model that emphasizes the sub-social route to sociality, which occurs when parents and offspring remain in association through the delayed dispersal of the latter [3]. Our model assumes that social groups are made of one dominant breeding individual and non-breeding subordinates. We link population dynamics to the evolution of delayed dispersal to allow for eco-evolutionary feedback and assume that group size or quality benefits manifest as changes to fecundity and survival. We are interested in how these changes affect delayed dispersal. In particular, we investigate the relationship between the probability with which a subordinate will establish as an independent breeder and the delayed dispersal rate. Finally, we ask how parent-offspring (breeder-subordinate) conflict over delayed

dispersal changes with changing benefits of group living.

2.2 Model

2.2.1 Basic assumptions

We observe a population composed of breeders, subordinates, and floaters at discrete, evenly spaced points in time. Each breeder is the dominant individual in its breeding spot and is reproductively active. Subordinates live alongside a breeder on the same breeding spot and are sexually mature but not reproductively active. Each occupied breeding spot is thus composed of one breeder and several subordinates. Floaters do not live in a breeding spot but are looking for one. They occupy a habitat that is not suitable for reproduction. We want to consider group size effects in our model; however, explicitly tracking group size can make the model's state space unmanageable. To get around this challenge, we introduce breeding spot (i.e. group) quality and connect this quality to the dispersal tendencies of a group's members.

We allow breeding spots to be of two qualities: low-quality or high-quality. We separate breeders who live in a low-quality breeding spot from those who live in a high-quality one. We separate subordinates the same way, distinguishing between those living in a low-quality and a high-quality breeding spot, respectively. High quality is achieved and maintained when sufficient subordinates delay dispersal and remain on the breeding spot. If too few subordinates stay, then the quality of the breeding spot may be reduced.

We have five classes of individuals in the population, and we keep track of the number of individuals in each class. We use U_t to denote the number of floaters, $B_{0,t}$ for the number of breeders in the population who belong to a low-

quality group, $B_{1,t}$ for the number of breeders in the population who belong to a high-quality group, $A_{0,t}$ for the number of subordinates in the population who occupy a low-quality group, and $A_{1,t}$ for the number of subordinates in the population who occupy a high-quality group, where the subscript t tells us that population is censused at the beginning of time step t. All notation and parameters with their values are described in Table 2.1. Parameter values may vary depending on the life history of the species.

2.2.2 Population dynamics

Figure 2.1 presents the five life-history events that occur between census points. These events are described in order below.

(1) Early dispersal

At the beginning of this stage, one breeder and n_0 subordinates live on each low-quality breeding spot; one breeder and $n_1 \geq n_0$ subordinates live on each high-quality breeding spot. High-quality groups are thus larger than low-quality groups at the beginning of the time step. Each subordinate disperses (early) or delays dispersal as the stage progresses. The dispersal or delayed dispersal of each subordinate occurs independently but depends on the breeding spot's quality. If subordinates delay dispersal, they remain on the breeding spot; if they disperse, they become floaters. We use h_0 to denote the probability that a subordinate delays dispersal from a low-quality spot, and we use h_1 to denote the analogous probability for a high-quality breeding spot. Of course, $1 - h_0$ and $1 - h_1$ give the probabilities with which subordinates in low-quality and high-quality groups, respectively, become floaters. At the end of this stage, there are $h_0 n_0$ subordinates in a low-quality group and $h_1 n_1$

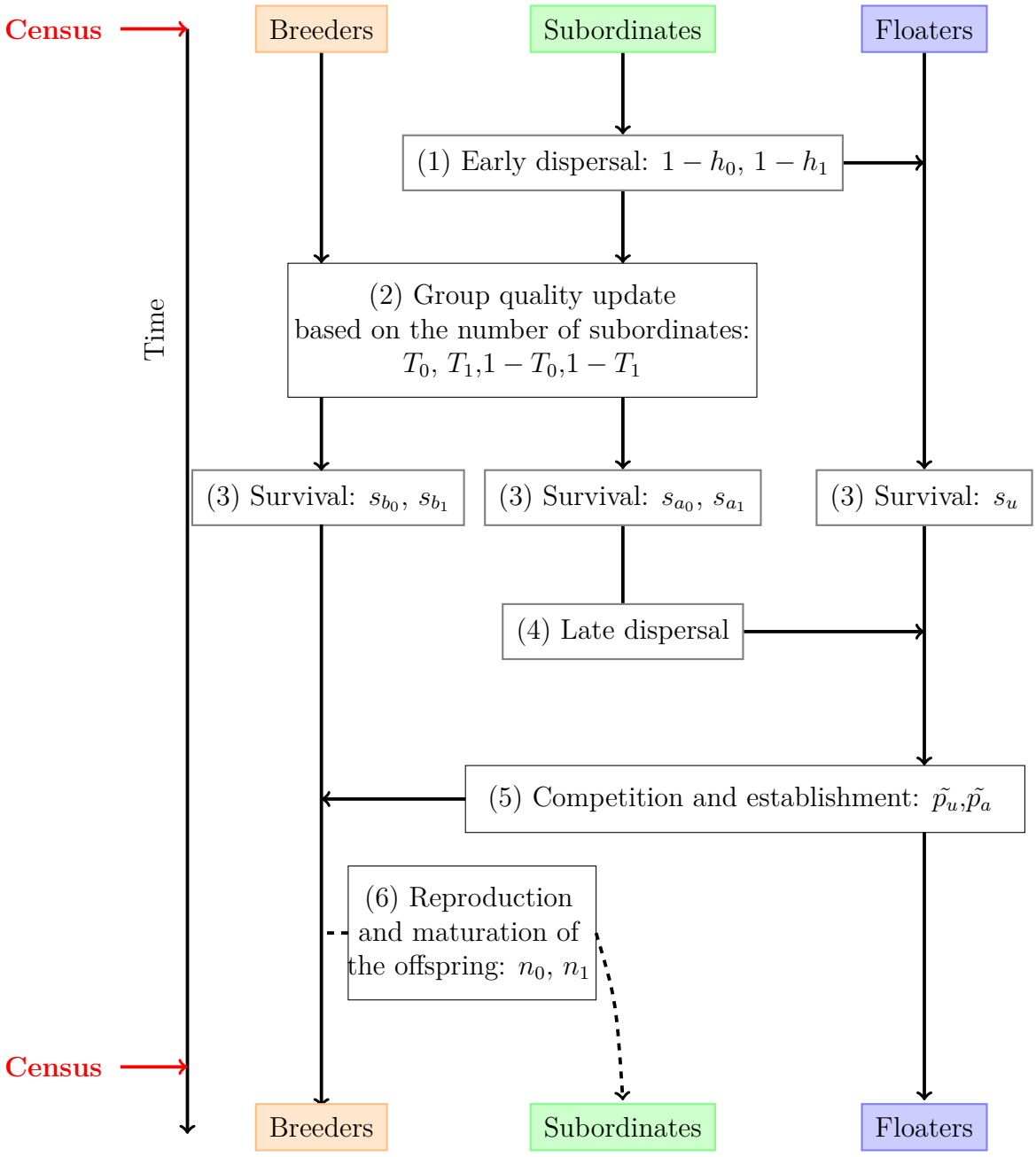

Figure 2.1: Life-history stages that occur during one time step.

subordinates in a high-quality group.

In general, we consider two scenarios. In the first scenario, delayed dispersal is controlled by the subordinate, meaning that it is determined by genes expressed by the subordinate itself. In the second scenario, delayed dispersal is under the breeder's control, meaning that it is determined by genes expressed by the breeder. We can study breeder-subordinate conflict over the decision to delay dispersal by considering breeder and subordinate control.

(2) Group quality updates

More subordinates on a breeding site increase the chance that the group will transition to or maintain a high-quality level. Conversely, fewer subordinates increase the chance that the group will transition to or maintain a low-quality level. To capture these effects, we introduce $T_0(h_0) = 1-e^{-n_0 h_0}$ as the probability that a low-quality group becomes high-quality and $1-T_1(h_1) = 1-e^{-n_1 h_1}$ as the probability that a high-quality group remains high-quality. We also introduce $1-T_0(h_0)$ as the probability that a low-quality group remains low-quality and $T_1(h_1)$ as the probability that a high-quality group becomes low-quality. In the long run, we expect a fraction $\frac{T_1(h_1)}{T_0(h_0)+T_1(h_1)}$ of the groups to be of low-quality and a fraction $\frac{T_0(h_0)}{T_0(h_0)+T_1(h_1)}$ of the groups to be of high-quality.

(3) Survival

At this stage of the life cycle, individuals suffer mortality. Floaters survive this stage at rate s_u. The survival rate among group members (i.e. non-floaters) depends on the quality of the group in which they are found. Breeders in a low-quality group survive this stage at rate s_{b_0}, while those in high-quality groups survive at s_{b_1}. We suppose $s_{b_1} \geq s_{b_0}$. Subordinates survive this stage

at rate $s_{a_0} = s_u$ in low-quality groups and at rate $s_{a_1} \geq s_{a_0}$ in high-quality groups. We treat this survival difference $s_{a_1} - s_{a_0}$ as indicative of a group size effect because it reflects the extent of the direct benefits reaped by a subordinate who delays dispersal. The difference for both $s_{b_1} - s_{b_0}$ also reflects a benefit awarded to the breeder thanks to the increased group size/quality. Similar effects have been ascribed to benefits associated with collective action in groups, group resource defense, group predator defense, and group foraging [79, 5, 58].

(4) Late dispersal of subordinates

Surviving subordinates disperse and become floaters. Note that they are slightly different than actual floaters for their ability to compete for a breeding spot, see next stage.

(5) Competition and establishment

Former subordinates and floaters attempt to establish as independent breeders. Because they have delayed dispersal, subordinates are at a disadvantage. Floaters become established with intrinsic probability p_u, while delayed dispersers become established with intrinsic probability $p_a < p_u$. In all cases, habitat saturation, represented by the total number of occupied breeding spots $(B_{0,t} + B_{1,t})$, reduces the probability of establishing. The actual probability with which a floater establishes in this time step is $\tilde{p_u} = \frac{p_u}{1+a(B_{0,t}+B_{1,t})}$, and is $\tilde{p_a} = \frac{p_a}{1+a(B_{0,t}+B_{1,t})}$ for late dispersers, where $a > 0$ is the strength of the habitat saturation effect. New breeders establish low-quality groups. Delayed dispersers and floaters who fail to secure a breeding spot remain floaters.

(6) Reproduction and maturation of offspring

Breeders, including the ones established in step (5), reproduce. Breeders are sexual hermaphrodites and reproduce as female and male. When a breeder reproduces through female function, it randomly chooses N potential mates, i.e., other breeders who could provide the paternal contribution to offspring. With probability $1-\phi$, the breeder selects one of its N potential mates to provide the paternal contribution for a given offspring; alternatively, with probability ϕ, the breeder provides the paternal contribution itself. We call ϕ the self-fertilization (selfing) rate based on this. Keeping in mind that a breeder can be chosen to be the mate of another individual, we conclude that the breeder of each low-quality group produces n_0 offspring through female function and expects to produce $\phi n_0 + (1-\phi)\frac{n_0 B_{0,t} + n_1 B_{1,t}}{B_{0,t}+B_{1,t}}$ through male function. Similarly, the breeder of each high-quality group produces n_1 offspring through female function and $\phi n_1 + (1-\phi)\frac{n_0 B_{0,t} + n_1 B_{1,t}}{B_{0,t}+B_{1,t}}$ through male function. All offspring produced through female function or through male function via selfing are found locally on the breeder's own breeding spot during this stage. All offspring produced through male function via outcrossing are found on the breeding spot(s) of the breeder's mate(s) during this stage.

After reproduction, offspring mature and become subordinates. Thus, at the end of the stage, there are n_0 subordinates in each low-quality breeding spot and n_1 subordinates in each high-quality breeding spot. We interpret the difference $n_1 - n_0$ as a fecundity benefit, again associated with group size [as described above 79, 5, 58].

2.2.3 Population dynamic features

The events described in the previous subsection lead to a mathematical description of how the distribution of individuals changes from one time step to the next. Let $V_t = (U_t, B_{0,t}, B_{1,t}, A_{0,t}, A_{1,t})^T$ be the distribution of individuals at time t. Then, $V_{t+1} = B(B_{0,t}, B_{1,t})V_t$, where the matrix $B(B_{0,t}, B_{1,t})$, given in Appendix A.1, captures the dynamics of the population. The dynamic equation admits a trivial equilibrium, $(0, 0, 0, 0, 0)^T$. We use the local stability analysis described in Appendix A.2 to find that the stability of the trivial equilibrium is determined by

$$R_0 = L_u L_b \Lambda \tag{2.1}$$

which is the expected number of subordinates a given subordinate produces throughout its lifetime. It is composed of $L_u = \frac{1}{(1-s_u(1-p_u))}$ which is the expected lifetime spent as a floater and $L_b = \frac{1}{((1-s_{b_0}(1-T_0))(1-s_{b_1}(1-T_1))-s_{b_0}s_{b_1}T_0T_1)}$ which gives the expected lifetime spent as a breeder, and finally Λ which summarizes the expected rate at which the average breeder produces subordinates of all types (given in Appendix A.2). When R_0 is less than 1, the trivial equilibrium is stable, and the population tends to extinction over time. When R_0 is greater than 1, the trivial equilibrium is unstable, and if there are constraints on the density of the habitat, i.e., $a > 0$, the population reaches a positive equilibrium $V^* = (U^*, B_0^*, B_1^*, A_0^*, A_1^*)$. Henceforth, we require R_0 to be greater than 1.

For later use, we introduce the probability with which a floater eventually

establishes as an independent breeder,

$$P^* = \frac{s_u \tilde{p_u}}{1 - s_u(1 - \tilde{p_u})}. \tag{2.2}$$

For short, we call this the probability of establishment. The star attached to P^* emphasizes the fact that $\tilde{p_u}$ is evaluated at equilibrium V^*. While P^* refers specifically to floaters, it also influences the probability with which a subordinate establishes as an independent breeder.

2.2.4 Evolution of delayed dispersal behavior

We study how selection changes the delayed dispersal strategies h_0 and h_1. Here, in the main text, we build an expression for the change in inclusive fitness [36] experienced by the individual who controls subordinate dispersal when the delayed dispersal rate is increased. Our approach follows [87], but in Appendix A.3, we show how our expressions can be built by the alternative method developed in [90].

To build an expression for the inclusive fitness change experienced by the focal individual, we must incorporate the reproductive values of different individuals. The reproductive value of an individual describes the asymptotic genetic contribution made by that individual to future generations [90, 23]. The reproductive value of each individual in a given class (breeder, floater, subordinate) is computed in Appendix A.3.

We describe the inclusive fitness change for the delay dispersal rate in low-quality groups h_0 and present the analogous expression for the delay dispersal rate in high-quality groups h_1. Fix attention on a subordinate in a low-quality

Table 2.1: Summary of notation used in the main text and, where appropriate, baseline value used.

Notation	Description	Baseline Value
s_u	survival of floaters	0.5
s_{b_0}	survival of breeders in a low-quality group	0.5
s_{b_1}	survival of breeders in a high-quality group	0.5
s_{a_0}	survival of subordinates in a low-quality group	0.5
s_{a_1}	survival of subordinates in a high-quality group	0.5
p_u	ability of floaters to find a breeding spot	0.5
p_a	ability of late dispersers to find a breeding spot	0.25
a	strength of the habitat saturation	2
ϕ	self fertilization rate	0.1
h_0	delayed dispersal rate in low-quality groups	
h_1	delayed dispersal rate in high-quality groups	
n_0	number of subordinates in low-quality groups	3
n_1	number of subordinates in high-quality groups	3
$T_0(h_0) = 1 - e^{-n_0 h_0}$	transition rate from low to high-quality groups	
$T_1(h_1) = e^{-n_1 h_1}$	transition rate from high to low-quality groups	
U_t	number of floaters at time t	
$B_{0,t}$	total number of low-quality breeding spots occupied by a breeder at time t	
$B_{1,t}$	total number of high-quality breeding spots occupied by a breeder at time t	
$A_{0,t}$	total number of subordinates in the population found on a low-quality breeding spot	
$A_{1,t}$	total number of subordinates in the population found on a high-quality breeding spot	

breeding spot. This subordinate is the actor. Suppose the individual who controls the actor's behavior decides to increase the likelihood with which the actor delays dispersal. We denote the increase as δh_0 (for h_1, we use δh_1), and we suppose it is positive and small. Below, we enumerate three consequences of the decision to increase the delayed dispersal behavior for the reproductive value of all individuals who are affected by the change in the actor's behavior and genetically related to the individual with control.

First, by being less likely to disperse early, the actor forfeits the reproductive value it would have accrued as a floater, denoted ν_u. Thus, it loses reproductive value in $\delta h_0 \nu_u$. Of course, there is still reproductive value to be added. By increasing the delayed dispersal rate by δh_0, the subordinate remaining in the group survives to compete in stage (5) with probability $(1 - T_0(h_0))s_{a_0} + T_0(h_0)s_{a_1}$. Given it survives to stage (4), the actor fails to secure a breeding spot with probability $(1 - \tilde{p_a})$. In this case, it becomes a floater and has reproductive value ν_u. Alternatively, the actor secures a breeding spot with probability $\tilde{p_a}$, and in this case, it becomes a breeder in a low-quality spot with reproductive value ν_{b_0}. We also credit the new breeder with the reproductive value of all the subordinate offspring $\nu_{sub,0}$, which is composed of the subordinates produced through its female function (local) and male functions (non-local) in low and high-quality groups. Assuming the population is at equilibrium and recognizing that the breeder donates only half of its genes to its offspring, while the other half comes from the other parent, we find

$$\nu_{sub,0} = \underbrace{\frac{n_0}{2}\nu_{a_0}}_{\text{female function}} + \underbrace{\phi\frac{n_0}{2}\nu_{a_0} + (1-\phi)\left[\frac{B_0^*}{B_0^* + B_1^*}\frac{n_0}{2}\nu_{a_0} + \frac{B_1^*}{B_0^* + B_1^*}\frac{n_1}{2}\nu_{a_1}\right]}_{\text{male function}}, \tag{2.3a}$$

where ν_{a_0} and ν_{a_1} are the reproductive values of a subordinate born on low-quality and high-quality patches, respectively (see Appendix A.3). Equation (2.3a) is the reproductive value of all the subordinates produced by a breeder on a low-quality breeding spot. The analogous expression for the reproductive value of all the subordinates produced by a breeder on a high-quality breeding spot is

$$\nu_{sub,1} = \frac{n_1}{2}\nu_{a_1} + \phi\frac{n_1}{2}\nu_{a_1} + (1-\phi)\left[\frac{B_0^*}{B_0^*+B_1^*}\frac{n_0}{2}\nu_{a_0} + \frac{B_1^*}{B_0^*+B_1^*}\frac{n_1}{2}\nu_{a_1}\right]. \quad (2.3b)$$

Putting our observations together, when the actor is born in a low-quality breeding spot, the decision to delay dispersal more readily changes the actor's reproductive value by

$$\Delta_{h_0} W_{\text{Actor}} = \delta h_0 \Big(\left((1-T_0(h_0))s_{a_0} + T_0(h_0)s_{a_1}\right)\left((1-\tilde{p_a})\nu_u + \tilde{p_a}(\nu_{b_0}+\nu_{sub,0})\right) - \underbrace{s_u((1-\tilde{p_u})\nu_u + \tilde{p_u}(\nu_{b_0}+\nu_{sub,0}))}_{=\nu_u} \Big). \quad (2.4a)$$

Had we focused on a subordinate in a high-quality spot,

$$\Delta_{h_1} W_{\text{Actor}} = \delta h_1 \Big(\left(T_1(h_1)s_{a_0} + (1-T_1(h_1))s_{a_1}\right)\left((1-\tilde{p_a})\nu_u + \tilde{p_a}(\nu_{b_0}+\nu_{sub,0})\right) - s_u((1-\tilde{p_u})\nu_u + \tilde{p_u}(\nu_{b_0}+\nu_{sub,0})) \Big), \quad (2.4b)$$

would have been the analogous expression.

Second, the decision to delay the dispersal of the actor also affects the breeder. The effects on a low-quality breeding spot are solely due to changes in the transition probability to a high-quality spot. As a result of this decision, the transition rate $T_0(h_0)$ changes by $T_0'(h_0)/n_0$. When such a change happens

and assuming the breeder survives, the breeder's reproductive value changes from $\nu_{b_0} + \nu_{sub,0}$ to $\nu_{b_1} + \nu_{sub,1}$, where ν_{b_1} is the reproductive value of a breeder on a high-quality breeding spot. It follows that the decision changes the reproductive value of the breeder with whom the subordinate actor is associated by

$$\Delta_{h_0} W_{\text{Breeder}} = \delta h_0 \frac{T_0'(h_0)}{n_0}(s_{b_1}(\nu_{b_1} + \nu_{sub,1}) - s_{b_0}(\nu_{b_0} + \nu_{sub,0})). \tag{2.5a}$$

The analogous expression for the change in a breeder on a high-quality breeding spot is

$$\Delta_{h_1} W_{\text{Breeder}} = \delta h_1 \frac{(1 - T_1(h_1))'}{n_1}(s_{b_1}(\nu_{b_1} + \nu_{sub,1}) - s_{b_0}(\nu_{b_0} + \nu_{sub,0})). \tag{2.5b}$$

Note we use $(1 - T_1(h_1))$ in the previous equation to denote the probability that a high-quality group remains a high-quality group.

Finally, the decision to delay dispersal more readily impacts the (approximately) $h_0 n_0$ subordinates who remain in the breeding spot, including the actor. Again, the transition to high-quality groups, $T_0(h_0)$ increases by $T_0'(h_0)/n_0$ as a result of this decision. In this case, the survival of every remaining subordinate changes from s_{a_0} to s_{a_1}, and we arrive at

$$\Delta_{h_0} W_{\text{Subs}} = \underbrace{\delta h_0 \, n_0 \, h_0 \frac{T_0'(h_0)}{n_0}(s_{a_1} - s_{a_0})}_{GS}[(1 - \tilde{p_a})\nu_u + \tilde{p_a}(\nu_{b_0} + \nu_{sub,0})], \tag{2.6a}$$

as the total change in reproductive value for remaining subordinates. The term GS denotes the group size effect for subordinates: an increase in delayed dispersal (δh_0) makes the group more likely to become of high-quality and ultimately leads to a survival benefit ($s_{a_1} - s_{a_0}$). This group size effect

is weighted by the reproductive value obtained as a late disperser (term in squared brackets). The analogous change for late dispersers in a high-quality breeding spot is

$$\Delta_{h_1} W_{\text{Subs}} = \delta h_1 \, n_1 \, h_1 \frac{(1 - T_1(h_1))'}{n_1} (s_{a_1} - s_{a_0})[(1 - \tilde{p_a})\nu_u + \tilde{p_a}(\nu_{b_0} + \nu_{sub,0})]. \tag{2.6b}$$

Again, we use $(1 - T_1(h_1))$ as the probability that a high-quality breeding spot remains high-quality.

We build an expression for the overall change of inclusive fitness experienced by the individual with control of the actor's behavior by taking (2.4), (2.5) and (2.6), weighting them by the appropriate relatedness coefficient, and summing. The result for the focal individual on a low-quality breeding spot is

$$\Delta_{h_0} W = R_{I,B_0} \cdot \Delta_{h_0} W_{\text{Breeder}} + \underbrace{\bar{R}_{I,A_0} \cdot \Delta_{h_0} W_{\text{Subs}}}_{j} + R_{I,A_0} \cdot \Delta_{h_0} W_{\text{Actor}}. \tag{2.7a}$$

Here R_{I,B_0} expresses the relatedness between the individual with control and the breeder ($I = A_0$ for subordinate control and $I = B_0$ for breeder control). The coefficient $\bar{R}_{I,A_0}$ expresses the relatedness between the individual with control and the average subordinate remaining in the low-quality breeding spot, including the actor. Finally, coefficient R_{I,A_0} is the relatedness between the individual in control and the subordinate actor. All relatedness coefficients are calculated in Appendix A.4.

Similarly, the inclusive fitness change for a subordinate on a high-quality patch is

$$\Delta_{h_1} W = R_{I,B_1} \cdot \Delta_{h_1} W_{\text{Breeder}} + \bar{R}_{I,A_1} \cdot \Delta_{h_1} W_{\text{Subs}} + R_{I,A_1} \cdot \Delta_{h_1} W_{\text{Actor}}, \tag{2.7b}$$

where similar to (2.7a), relatedness coefficients are computed between the individual in control I and the breeder or the subordinate(s) on a high-quality breeding spot.

Switching which individual controls the behavior, the breeder or the subordinate, only changes the relatedness coefficients that appear in the expression for the inclusive fitness change, R_{I,A_0}, R_{I,A_1}, R_{I,B_0}, R_{I,B_1}, $\bar{R}_{I,A_0}$, and $\bar{R}_{I,A_1}$. The other components of the inclusive fitness change remain the same, as the effect on fitness is identical. When the subordinate controls its behavior, $I = A_0$, we use $\bar{R}_{A_0,A_0}$ in the inclusive fitness expression. The definition of $\bar{R}_{A_0,A_0}$ means that term j in (2.7a) includes the personal benefits of increased group quality awarded to the actor. Thus, while the term j does include indirect benefits to kin, it also includes personal benefits associated with group size effect. To be clear, when the subordinate controls the behavior, the term j can be written as

$$\bar{R}_{A_0,A_0} \cdot \Delta_{h_0} W_{\text{Subs}} = \underbrace{\frac{1}{n_0} \cdot \Delta_{h_0} W_{\text{Subs}}}_{Direct} + \underbrace{\frac{n_0 - 1}{n_0}(\phi^2 + \phi(1-\phi) + (1-\phi)^2 \frac{N+1}{4N}) \cdot \Delta_{h_0} W_{\text{Subs}}}_{Indirect}. \quad (2.8)$$

When the breeder controls the behavior, we use $\bar{R}_{B_0,A_0}$. Thus, the term j covers only indirect benefits (from the breeder's perspective) awarded to the average subordinates remaining on the natal nest.

The sign of $\Delta_{h_0} W$ and $\Delta_{h_1} W$ gives us the direction of the evolution for h_0 and h_1 under the action of selection. If $\Delta_{h_0} W > 0$, then selection will favor an increase in the average tendency to delay dispersal h_0. If $\Delta_{h_0} W < 0$, then selection will favor a decrease in the average tendency to delay dispersal h_0. If

the inclusive fitness change is equal to 0, then selection does not act to change the associate delayed dispersal strategy. The same dynamics apply to h_1 and $\Delta_{h_1} W$.

When both $\Delta_{h_0} W = 0$ and $\Delta_{h_1} W = 0$, the system is at evolutionary equilibrium. We use h_0^*, h_1^* to denote this evolutionary equilibrium. We assess the stability of the evolutionary equilibrium numerically, when it exists, as described below. When it is stable, the evolutionary equilibrium represents the endpoint of the evolutionary process. In some cases, we expect the evolutionary process to lead us to the boundary of the trait space (the faces of the unit square $[0, 1]^2$). In these cases, the endpoint of evolution is not, strictly speaking, an evolutionary equilibrium.

2.2.5 Numerical methods

We use numerical simulations to obtain the endpoint of the evolution of the delayed dispersal strategies. Given delayed dispersal strategies h_0 and h_1, we compute $\Delta_{h_0} W$ and $\Delta_{h_1} W$, and then update using $h_0 + \delta h_0 \Delta_{h_0} W$ and $h_1 + \delta h_1 \Delta_{h_1} W$, respectively. If, at any time, either h_0 or h_1 or both is lower than 0 or greater than 1 (because the selection is directional), we replace this value with the one occurring at the closest border. We begin our simulation assuming a fully dispersing population ($h_0 = h_1 = 0$), and we continue until one of three things happens:

1. both $\Delta_{h_0} W$ and $\Delta_{h_1} W$ are within a tolerance of 0 in absolute values, in which case the evolutionary process has reached an internal evolutionary equilibrium;

2. both values of h_0 and h_1 remain the same after an iteration, in which case, the evolutionary process ended at one of the corners of the trait

space $[0, 1]^2$;

3. one delayed dispersal strategy, h_0 or h_1, has been repeated exactly. The other value's inclusive fitness change, $\Delta_{h_1} W$ or $\Delta_{h_0} W$, is within a tolerance of 0 in absolute value, indicating the process has settled on one of the faces of the trait space but not on a corner.

There is a fourth case for which we would stop the process, namely when a maximum number of iterations has been exceeded. We did not observe this case. Note that in the three cases, we observed, the endpoint of the evolution is stable in the sense that it is an attractor.

At the end of the process, the last value of h_0 and h_1 is reported. When reported values are both strictly greater than 0 and strictly less than 1, we have an evolutionary equilibrium (h_0^*, h_1^*) that is also an attractor (multidimensionally stable). We also use h_0^* and h_1^* for values returned by the simulation that occurs at the boundary. Overall, we use the simulations to study the role of various parameters that affect $\Delta_{h_0} W$ and $\Delta_{h_1} W$.

All simulations are performed using Julia (1.7.1), and results are visualized using Python (3.8).

All simulation code can be found on this link.

2.3 Results

2.3.1 At equilibrium, the number of delayed dispersers is independent of group quality

At evolutionary equilibrium, a class of solutions satisfying both $\Delta_{h_0} W = 0$ and $\Delta_{h_1} W = 0$ is captured by

$$h_0^* n_0 = h_1^* n_1. \tag{2.9}$$

No other classes of evolutionary equilibrium solutions exist if there are no survival benefits for delayed dispersers ($s_{a_0} = s_{a_1}$), i.e., no group size effect for subordinates' survival (see the stage (3) of the life cycle). If their survival increases with group size ($s_{a_0} < s_{a_1}$), there may be other classes of evolutionary equilibrium solutions, but extensive numerical simulations did not reveal them. It seems that equation (2.9) is the only evolutionary equilibrium solution that exists based on numerical simulations. This equation also holds whether the individual controlling the delayed dispersal behavior is the subordinate or the breeder. Thus, parent-offspring conflicts change the value of h_0^* and h_1^* but not the relation (2.9).

Biologically speaking, equation (2.9) tells us that after early dispersal, low and high-quality groups are of the same size. Any size difference between low and high-quality groups we might observe during the life cycle are temporary. Moreover, since $n_0 \leq n_1$, we have $h_0^* \geq h_1^*$, meaning that subordinates born in high-quality are never more likely to delay dispersal than those born in a low-quality breeding spot. Essentially, smaller groups (low-quality groups) retain more subordinates at evolutionary equilibrium. In addition, equation (2.9)

implies that the quality distribution of groups is independent of the current group quality. It follows that at evolutionary equilibrium, the fraction of high-quality groups is $T_0(h_0^*) = 1 - T_1(h_1^*)$ and the fraction of low-quality groups is $1 - T_0(h_0^*) = T_1(h_1^*)$.

We can also deduce from equation (2.9) that $h_0^* > 0$ is equivalent to $h_1^* > 0$. In other words, the conditions that lead to $h_0^* > 0$ also lead to $h_1^* > 0$. In turn, this also implies that the set of parameters that supports delayed dispersal at non-zero levels ($h_0, h_1 > 0$) is independent of survival benefits for subordinates. That said, we found the delayed dispersal strategies at evolutionary equilibrium higher with group size effects for subordinates (see section 3.4). As a final comment, since h_0^* and h_1^* are related by equation (2.9), we only report results for h_0^* below.

2.3.2 Selfing promotes delayed dispersal but can diminish group size benefits for subordinates

We are interested in how selfing ϕ, i.e., self fertilization, impacts the evolution of delayed dispersal. On the one hand, we expect selfing to increase relatedness, promoting cooperative behaviors like delayed dispersal [36]. On the other hand, increasing the selfing rate may decrease the reproductive value that subordinates achieve through male function, which scales the group size effect on subordinates' survival, making it smaller (see equations (2.3a) and (2.6)). Selfing may also change the cost of delaying dispersal paid by late dispersers, as described in equation (2.4). On balance, numerical results suggest that increasing the selfing rate ϕ increases the level of delayed dispersal h_0^* (Figure 2.2A). This finding is consistent with the interpretation of delayed dispersal as an act of altruism, as it brings direct costs to the subordinates while giving

benefits to the breeder.

In addition to increasing the level of delayed dispersal, we observe that selfing decreases the reproductive value of a remaining subordinate who survived to stage (3) in the life cycle (Figure 2.2B). Recall that this reproductive value scales the group size effect on subordinates. Therefore, while selfing leads to more philopatric individuals, it reduces their reproductive value and ultimately reduces the impact of group size survival benefits on the evolution of delayed dispersal.

The cost of delayed dispersal paid by the subordinates, $\Delta_{h_0} W_{\text{Actor}}$ in (2.4a) and $\Delta_{h_1} W_{\text{Actor}}$ in (2.4b), is also decreased when we increase the selfing rate (Figure 2.2C). Indeed, as ϕ increases the delayed dispersal rate h_0, the personal benefits of staying increase too. The cost of delayed dispersal is, therefore, decreased with a higher ϕ. Thus there is a positive feedback between h_0 and ϕ for the decrease of the cost of delaying dispersal.

Overall, despite diluting the group size effect for subordinates, an increased selfing rate lowers the cost of delayed dispersal and raises the relatedness between individuals, leading to an increase in the level of delayed dispersal. We can thus expect selfing to increase relatedness, and reduce the value of dispersers, leading to higher levels of delayed dispersal.

Two interesting features of our model are revealed when we increase either (a) the selfing rate or (b) the fecundity benefits of moving into a higher quality group, $n_1 - n_0$. First, when we make these increases, selection raises the delayed dispersal rate (Figure 2.2A). However, the same increases also lower the reproductive value achieved by a remaining subordinate (Figure 2.2B). The reproductive value of a philopatric subordinate is thus maximal when the delayed dispersal rate is minimal (which, in our case, is no delayed dispersal). Second, because this reproductive value scales the group size effect for subor-

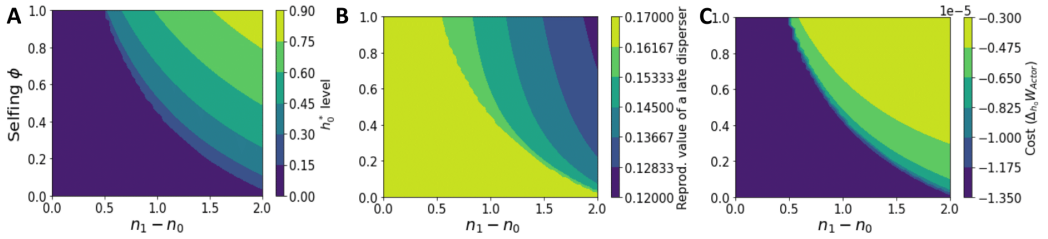

Figure 2.2: The impact of selfing rate ϕ and fecundity benefit $(n_1 - n_0)$ on (A) equilibrium level of delayed dispersal (h_0^*), (B) reproductive value of a delayed disperser $((1-\tilde{p}_a)\nu_u + \tilde{p}_a(\nu_{b_0} + \nu_{sub,0})$, see equation 6), and (C) the cost of delayed dispersal ($\Delta_{h_0} W_{\text{Self}} < 0$ is plotted). Numerical simulations assume $n_0 = 3$, $s_{b_0} = s_{b_1} = 0.5$, $s_u = s_{a_0} = 0.5$, $s_{a_1} = 0.6$, $p_u = 0.5$, $p_a = 0.25$, $a = 2$. Relatedness is calculated assuming a breeder only chooses one mate (see Appendix A.4). In all cases, we assume subordinate control of delay dispersal. Qualitatively similar results are found with breeder control.

dinates, this effect decreases with either selfing rate or fecundity benefits; it thus would be maximal when delayed dispersal is minimal if it were not for the fact that the increased group size effect is null at $h_0 = 0$ (see equation (2.6), GS). Thus, our second finding reveals a tragedy of sorts: the group size benefits for subordinates is unavailable exactly when it could have the highest impact.

2.3.3 Habitat saturation impact on delayed dispersal

The relationship between the delayed dispersal at evolutionary equilibrium and the probability of establishing as an independent breeder (equation (2.2)) depends on the species' life history. Figure 2.3 shows this relationship when we vary life history details captured by fecundity and survival. Note that we kept the strength of habitat saturation, a fixed to study the life history impact and not just habitat saturation effect. The qualitative nature of the relationship between habitat saturation and life history details changes depending on

whether fecundity or survival is varied. Nevertheless, the relationship reveals two consistent patterns. First, as we increase the benefits of delayed dispersal, we increase the evolutionary equilibrium rate of delayed dispersal. This is most evident when we consider the effect of increasing $n_1 - n_0$, $s_{b_1} - s_{b_0}$, and $s_{a_1} - s_{a_0}$ (Figure 2.3A, B, C). The pattern is also evident as we decrease n_0; in this case, the decrease leads to a greater benefit of delayed dispersal owing to the transition-rate model's geometry (Figure 2.3D). Second, whenever we change the life history in a way that leads to higher rates of production or survival, we lower the probability of establishment (Figure 2.3A, B, C).

We wish to emphasize that as we decrease the fecundity rate, n_0, we increase delayed dispersal and the probability of establishment (Figure 2.3A). In this case, we have a positive relationship between the probability of establishment and delayed dispersal. This pattern contrasts with other patterns that we found when varying survival (Figure 2.3B, C) and contrasts with reports of this relationship in the literature (17, 56; but see 57).

2.3.4 Breeder-subordinate conflict

We study the breeder-subordinate conflict by computing the difference between the levels of delayed dispersal at evolutionary equilibrium when the breeder controls the behavior and when the subordinate controls the behavior. In general, we expect this difference not to be negative. Indeed, it is more beneficial for the breeder than for the subordinate to delay the dispersal of the latter. This is in keeping with standard predictions of parent-offspring conflict given that delayed dispersal can be viewed as altruistic, [96]. In addition, the threshold at which individuals start to delay dispersal when under parental control is always lower than when under subordinate control. The distance

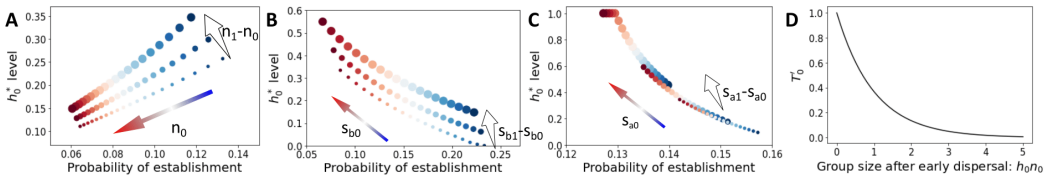

Figure 2.3: Relationship between equilibrium level of delayed dispersal (h_0^*) and probability of eventually establishing as an independent breeder (P^*, see equation (2.2)). Panel (A) shows the effect of varying fecundity n_0 between a minimum value of 3 (blue) to a maximum of 7 (red) and the effect of varying the benefit $n_1 - n_0$ from 3.42 (small circles) to 3.89 (medium circles) to 4.52 (large circles). Panel (B) shows the effect of varying breeder survival s_{b_0} from a minimum value of 0.3 (blue) to a maximum of 0.7 (red) and the effect of varying the benefit $s_{b_1} - s_{b_0}$ from 0.09 (small circles) to 0.12 (medium circles) to 0.15 (large circles). Panel (C) shows the effect of varying subordinate survival s_{a_0} from a minimum value of 0.5 (red) to a maximum of 0.7 (blue) and the effect of varying the benefit $s_{a_1} - s_{a_0}$ from 0.03 (small circles) to 0.07 (medium circles) to 0.11 (large circles). In (C), we assume assumes $n_1 = 5$ and $n_0 = 3$. Unless otherwise stated, simulations in (A)-(C) assume $p_u = 0.5$, $p_a = 0.25$, $a = 2$, $n_0 = n_1 = 3$, $s_{b_0} = 0.5$, $s_{b_1} = 0.5$, $s_u = s_{a_0}$, $s_{a_0} = 0.5$, $s_{a_1} = 0.5$, $\Phi = 0.1$. Relatedness is calculated assuming a breeder only chooses one mate (see Appendix A.4). Panel (D) shows the marginal effect of increased delayed dispersal on the probability with which low-quality groups transition to high-quality, T_0' depending on the group size $h_0 n_0$. In particular, as the fecundity parameter n_0 increases, the delayed dispersal's marginal effect is diminished.

between the thresholds is relevant to the results we present below.

As a check, we start our analysis of conflict by removing breeder benefits ($n_1 = n_0$ and $s_{b_1} = s_{b_0}$) and group size benefits for subordinates ($s_{a_0} = s_{a_1}$) while still group membership benefits ($s_u < s_{a_0}$). In this case, only direct benefits to the actor remain in the model, and our numerical simulation correctly predicts no breeder-subordinate conflict (see supplementary data). We investigate conflict by varying the selfing rate ϕ as an additional check. As ϕ increases, relatedness between breeders and subordinates increases, and conflict decreases (see supplementary data). In the limit, as ϕ goes to 1, conflict disappears, as expected.

Raising the benefits given to the breeder changes the level of breeder-subordinate conflict in a way that depends on the subordinates' survival benefits. The first noticeable change in conflict is an increase between the two thresholds described above. Between these thresholds, breeders want subordinates to stay more and more readily, but subordinates are unwilling (Figure 2.4Ai, ii, 2.4Bi, ii). The extent of the increase between the thresholds subordinates benefits from larger group size ($s_{a_1} > s_{a_0}$) even though the distance between the thresholds remains the same (colored vertical lines in Figure 2.4Aiii, iv, 2.4Biii, iv). Beyond the second threshold, both breeder and subordinate prefer non-zero levels of delayed dispersal, and the extent of the conflict either plateaus or decreases (Figure 2.4Ai, ii, 2.4Bi, ii). Importantly, the extent of the conflict beyond the second threshold tends to be higher when subordinates' survival increases with group size, with one exception (Figure 2.4Aiii, iv, 2.4Biii, iv). That exception occurs when the delayed dispersal level preferred by the breeder reaches its maximum value; in this case, conflict decreases as the subordinate's preference catches up to that of the breeder. Overall, increasing subordinates' survival in larger groups tends to exacerbate conflict as

benefits to the breeder increase.

Raising the group-size benefits given to subordinates has a non-monotonic effect on the breeder-subordinate conflict. In particular, the conflict first increases until reaching a maximal value and finally decreases for high values of benefits (Figure 2.4 Ci, Cii). This decrease is driven by the fact that delayed dispersal is increasing under the subordinate's control. In contrast, under the breeder's control, delayed dispersal has reached the upper bound of the behavior, $h_0 = 1$. While it is tempting to conclude that an increased survival for subordinates is tempering breeder-subordinate conflict, the decrease in conflict is because the breeder's preferred subordinate behavior is at its maximum level.

2.4 Discussion

We studied the formation and maintenance of groups that include a dominant breeder and non-breeding subordinates. We accomplished this by building a model for the evolution of a subordinate behaviors. These behaviors can be understood either as delayed dispersal or as delayed dispersal linked with helping. In both cases, the evolution of the behavior has implications for cooperative breeding. Delayed dispersal is considered an important precursor to the evolution of cooperative breeding systems [55]. Delayed dispersal linked to breeder-directed helping has been used repeatedly to explain the adaptive significance of cooperative breeding, especially in the face of ecological constraints [17, 70, 99].

Our first main result is that, at evolutionary equilibrium, the number of non-dispersers is independent of group quality. This is a consequence of the equilibrium dispersal rate from large high-quality groups being higher than

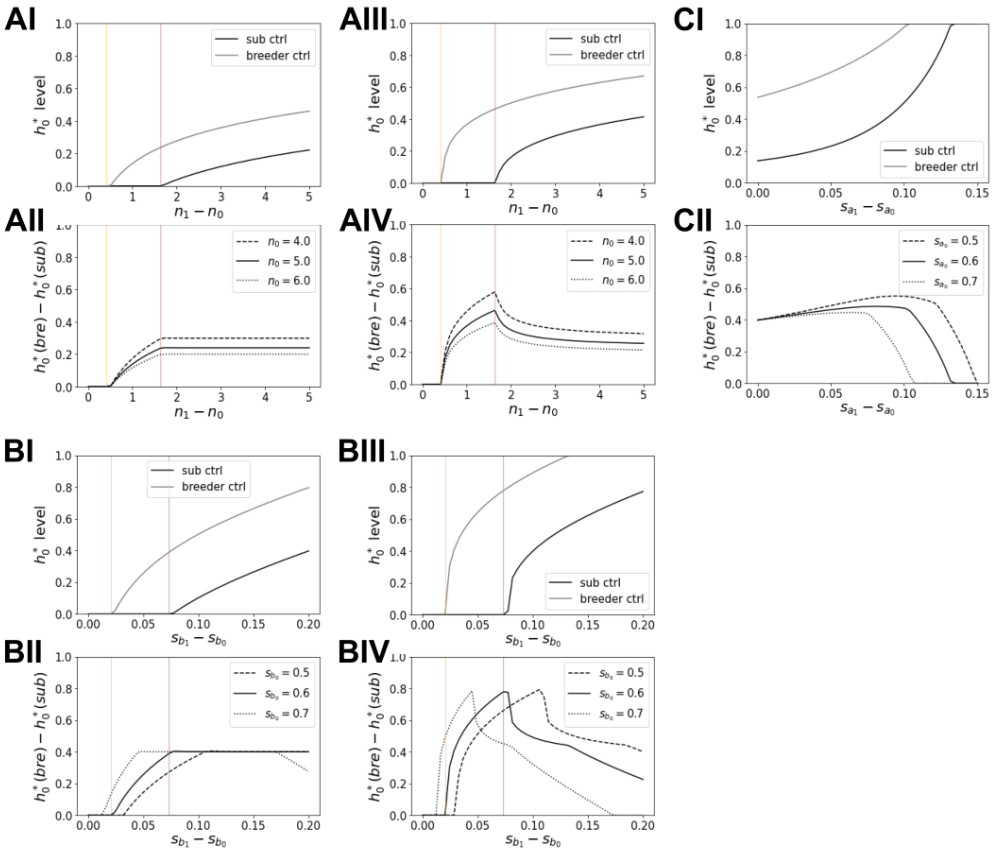

Figure 2.4: Equilibrium levels of delayed dispersal h_0^* and the extent of breeder-subordinate conflict over delayed dispersal represented by the difference of predictions under breeder exclusive control or under subordinate exclusive control $(h_0^*(bre) - h_0^*(sub))$. Panel (A) shows how the levels of delayed dispersal and the extent of the conflict change with fecundity benefit $n_1 - n_0$. Coloured vertical lines mark the location of the threshold value of $n_1 - n_0$ beyond which $h_0 = 0$ is no longer favoured. The left vertical line (orange) marks the threshold for the breeder control scenario and the right vertical line (brown) marks that for the subordinate control scenario. Vertical lines are in the same position in all of (A). Panel (B) shows how the levels of delayed dispersal and the extent of the conflict change with breeder survival benefit $s_{b_1} - s_{b_0}$. Coloured vertical lines mark the location of the threshold value of $s_{b_1} - s_{b_0}$ beyond which $h_0 = 0$ is no longer favoured. The left vertical line (orange) marks the threshold for the breeder control scenario and the right vertical line (brown) marks that for the subordinate control scenario. Vertical lines are in the same position in all of (B). Panel (C) shows how the levels of delayed dispersal and the extent of the conflict change with subordinate survival benefit $s_{a_1} - s_{a_0}$. Unless otherwise noted, $n_0 = n_1 = 3$, $s_{b_0} = s_{b_1} = 0.5$, $s_u = s_{a_1} = s_{a_0} = 0.5$, $\phi = 0.1$, $a = 2$, $p_a = 0.25$, $p_u = 0.5$. Relatedness was calculated assuming breeders have one mate (see Appendix A.4).

that from small low-quality groups. Similar density-dependent dispersal patterns have been found in cooperative breeders like meerkats (*S. surricata*) where individuals in small groups disperse less frequently than individuals in larger groups [61]. More generally, dispersal in other cooperative breeding systems may be positively or negatively related to group size depending on the benefits of cooperation and the cost of kin competition [9, 62].

Our first result reflects the conclusion of the "constant philopater hypothesis" proposed by [76]. Those authors used a classic island model to show that breeders of all qualities produce the same number of philopatric offspring, but breeders of higher quality produce more offspring that disperse. Our equation (2.9) shows that the number of delayed dispersers is independent of group quality and, since there is a higher fecundity in high-quality groups, there are more dispersers in high-quality groups. Therefore, our results extend the constant philopater hypothesis to a scenario where density is not regulated as strictly as in an island model, and dispersal (rather, delayed dispersal) influences the quality we assign to the breeder. Given that philopatry conditioned on quality impacts sex-ratio [8], then future work could extend the results of our study to sex-dependent helping in cooperative breeders.

We focus our work on the increase of the survival of an individual who delay dispersal. We treat these benefits as passive and connected to improved group size. We could have interpreted them as active benefits generated by an actor who delays dispersal and subsequently helps. Regardless of the interpretation, these group size benefits for subordinates, lead them to delay dispersal more readily but do not change the set of conditions under which groups are predicted to form. In other words, group size affecting the survival of subordinates leads to larger groups but does not make group formation more likely. This conclusion complements those arising from the simulation models

of [25]. There, the increased survival for subordinates in larger groups drives the formation of groups. Our work shows that kin selection can also drive the formation of groups via the sub-social route with a group size effect. In this context, delayed dispersal can be considered an act of altruism by the subordinate and directed toward the breeder. [25] correctly points out that group size effects set the stage for kin-selected active helping. Our work suggests group size effects can also open the door to increased benefits directed toward kin when those benefits are related to group size or quality.

When we interpret subordinate behavior in our model as delayed dispersal linked to active help, our predictions are consistent with recent empirical findings on group size effects in cooperative breeding systems. Specifically, it has been found that helpers in smaller groups work harder than those in larger groups, even after correcting for the load-lightening effect [26]. If we interpret subordinate behavior in our model as effort, then a similar idea comes through in equation (2.9): helpers in low-quality groups compensate by working harder.

We investigated the relationship between the probability with which a floater will eventually establish as an independent breeder and its tendency to delay dispersal. We found that a low probability of establishment is not always associated with a high level of delayed dispersal when the evolution of delayed dispersal itself is tied to population dynamics. In particular, we found that by decreasing the overall level of reproduction, we simultaneously decrease the competition among floaters and increase the reproductive benefits of delayed dispersal. Decreased competition among floaters leads to greater establishment probability, increased benefits incentivize delayed dispersal, and so a positive relationship emerges between the probability of establishment and delayed dispersal. Previous work has predicted a negative relationship between the probability of successful independent breeding and delayed dis-

persal [17, 56, 55]. However, that work treats the probability of success as an independent variable in the model that is not altered by population dynamic feedback. Overall, we see that a complete understanding of delayed dispersal and cooperative breeding requires consideration of population dynamic feedback. Pen and Weissing,[70] and Kokko et al. [57] have expressed similar sentiments. The latter found the survival of dispersers mediated a positive relationship with the probability of establishment and delayed dispersal when these were connected through population dynamics. Our work shows a similar effect but with reproduction.

We built a sexual model that allows us to study breeder-subordinate conflict over delayed dispersal. The scenario here is essentially parent-offspring conflict over altruism, even though we consider subordinate's behavior to provide passive benefits. In keeping with parent-offspring conflict theory [96], the breeder always wants the subordinate to delay dispersal more than the subordinate is willing to. The discrepancy between these perspectives is how we measure the intensity of the conflict. In this way, we study a battleground rather than a resolution model, as we observe and do not predict how the conflict can be solved, [29].

Previous work on natal dispersal showed a pattern of conflict that is different than the one we uncover. In those models, dispersal rather than philopatry is the altruistic act. Consequently, a parent in those model prefers a higher rate of dispersal (lower rate of philopatry) than preferred by an offspring [66, 85]. This previous work, however, does not account for the possibility for group size benefits and emphasizes a viscous population demographics and kin competition. In this way, previous work does not capture some important features that we emphasize in our model and that are thought to be important in family units where cooperative breeding is absent [58]. In particular, it misses the

particular parent-offspring conflicts that we identify here.

Our battleground model shows that, generally, group size effects exacerbates the conflict between the breeder and the subordinate. The results derive from the fact that the conflict is strongest when the subordinate is unwilling to delay dispersal, but some delay dispersal is optimal from the breeder's perspective. In this zone of conflict (between the thresholds we identify in the results), the breeder's perspective is more sensitive to group size effects than the subordinate's perspective. In fact, in many cases, the subordinate's perspective is insensitive to the large changes of benefits of delayed dispersal. We see signs of conflicts like the one predicted by our model in cooperative breeders like the white-fronted bee-eaters [*Merops bullockoides,* 18]. In this species, breeding parents disrupt their offspring's attempt at independent breeding to coerce the offspring into returning to their natal site and providing help. Similar patterns have been observed in pied babblers (*Turdoides bicolor*) where individuals can kidnap subordinates of other groups to increase their own group size [73], and in a cichlid fish (*Neolamprologus pulcher)* where dominant breeders harass non-contributing subordinates [102]. Given that this cichlid species' behavior is consistent with the group size effect hypothesis [26], it may provide a good system to test the effect of group size benefits on the extent of the breeders-subordinate conflict.

Our model of parent-offspring conflict can be used to contextualize reproductive skew within a social group. The conflict between the breeder and its offspring over delayed dispersal with group size benefits means the breeder will encourage offspring to delay dispersal more readily than the offspring would have otherwise. This encouragement can bring benefits for the offspring such as giving staying incentives by giving the offspring reproductive opportunities as in African lions (*Panthera leo*) [69], or dwarf mongooses, (*Helogale parvula*),

[11]. Other actions performed by the dominant breeder can be harmful for the offspring, such as reproduction suppression in naked mole rats. For instance, the breeder can delay the maturation of the subordinates. In the naked mole rats, the dominant breeder increases the subordinates' response to pup vocalizations [97] or inducing reproduction suppression [22].

The naked mole rat is a potential example of the kind of parent-offspring conflict we explore. This species lives in colonies where non-breeding subordinates help the dominant breeder (queen) reproduce. Only some offspring disperse; the rest are recruited as helpers. Dispersers have a specific morphology and behavior: they have a larger body and are more aggressive [67]. It is likely that the development of the disperser morph is a locus of conflict between the dominant breeder and its subordinates. The queen may prefer offspring develop as disperser morphs less readily than the offspring themselves would prefer. Perhaps, the breeder could advance its goal by manipulating the development of offspring through means that are similar to the one it uses when it manipulates the maturation and behavior of offspring already developed as subordinates [22].

Overall, group size effects plays a complex role in the evolution of delayed dispersal and cooperative breeding. This is especially true when evolution is tied to population dynamics and influenced by habitat saturation. Group size effects on subordinates' survival encourages delayed dispersal and promotes larger or higher-quality groups. In many cases, it exacerbates the conflict between the breeder and subordinate over the delayed dispersal. Our findings complement previous works and suggest new avenues for research.

Chapter 3

Selective advantages of redirected helping in a viscous population

3.1 Introduction

Cooperative breeding is a social system in which certain individuals invest their time and energy to care for offspring produced by others. Cooperative breeding —indeed, helpful behaviors more generally — has long puzzled researchers, but its adaptive significance can be understood in terms of benefits that come from reciprocity [95] or those arising via kin selection [38]. Although helping and cooperative breeding are well understood broadly, outstanding questions about their evolution remain.

One example of an open question centres around redirected help in cooperative breeding systems. Redirected helping occurs when an individual who has experienced brood failure redirects their parental effort to help a related

neighbor. This behavior is well documented in species such as the Long-tailed Tit (*Aegithalos caudatus*), [43], the Western Bluebird, (*Sialia mexicana*), [53], the Iberian Magpie (*Cyanopica cooki*) [13], among others. At first glance, redirecting helping looks like an evolutionary no-brainer: if an individual has little chance of personal (or direct) reproductive success in a given year, what downside could there be to attempting to make indirect gains through relatives? Of course, one obvious downside is the cost that would be incurred by helping [44].

Less obvious are the costs of helping in a viscous population. A population is considered viscous when individuals remain close to their natal site throughout their life [75]. Population viscosity imposes a genetic structure, meaning individuals interact and compete with their genetic relatives. Competition in a viscous population can be enough to cancel or at least reduce benefits arising from helping [101, 91]. In the same way, the success of redirected helping in a viscous population may be blunted by the increased local competition that it creates [78]. For instance, the benefits of redirected help, anticipated by the extra offspring it creates, may later be diminished if those extra offspring interfere with the success of related offspring produced by a different neighbor. Overall, it is unclear when redirected helping should be favored in viscous populations.

This paper tackles the problem of redirected helping in viscous populations. We build an inclusive-fitness model to outline the conditions under which this behavior is predicted to be advantageous. We use an infinite-patch (i.e., infinite-island) model where a breeder whose brood fails can decide to redirect parental effort to another breeder on the same patch. We assume redirected help brings survival or fecundity benefits to the recipient but costs the helper by decreasing its survival. In contrast with the results of previous studies,

we find that helping can, in some cases, be more strongly promoted when it provides survival rather than fecundity benefits. This result depends on the life history of the species and its environment; most notably, the result is impacted by the risk of brood failure and the survival rate of breeders. Our results provide new perspectives on cooperative breeding studies while opening new research areas.

3.2 Material and methods

3.2.1 Life cycle

We consider a large (ideally infinite) population of haploid asexual individuals. The population is subdivided into identical patches, each supporting n breeding spots occupied by a single adult (breeder).

We observe the population at discrete points in time (years). At the beginning of a given year, each individual produces a brood of N offspring, where N is very large. Each entire brood fails independently, with probability p. Failure could occur, for example, because of predation.

Each offspring produced by a successful breeder (probability $1-p$) becomes an adult with probability f (fledging success). It then disperses from its natal patch with probability d and survives dispersal with probability $(1-c)$, where c is the cost of dispersal. Given it survives, the dispersing offspring is on a new patch selected uniformly at random.

Each breeder suffers mortality after offspring dispersal. Breeders whose brood failed survive at rate s_0, while those with a successful brood survive at rate s_1. Survivors retain their patches, and offspring compete equally for a breeding spot vacated by adult breeders who died. As more offspring compete

than there are free breeding spots (N large), all breeding spots are occupied at the end of the time step.

When an individual's brood fails, it can decide to help another randomly chosen breeder on the same patch whose brood did not fail. Importantly, we assume that an individual's tendency to provide help when their brood fails does not impact the probability with which their brood fails. A helpful failed breeder (the actor) increases either the fledging success of the recipient's offspring by a small amount δf or the survival of the recipient itself by a small amount δs_1. However, by helping, the actor expends some energy, which translates to a small decrease in its own survival by δs_0. The fact that δf, δs_1, and δs_0 are small reflects an underlying assumption that the effect of selection is weak. This is a common assumption in inclusive fitness models [30, 86, 87, 100].

To connect to standard theory [38], we use $C = \delta s_0/s_0$ for the fitness cost paid by the actor (cost of helping), $B_s = \delta s_1/s_1$ (survival benefit) for the fitness benefit given to the actor's patchmate, and $B_f = \delta f/f$ (fecundity benefit) for the fitness benefit given to each of the patchmate's N offspring. We can interpret C, B_s, and B_f as selection coefficients from mathematical population genetics [see 12].

3.2.2 Inclusive fitness of redirected helping

We now build our inclusive fitness expression for the advantage of redirected help in an otherwise selfish population. In a later section, we will give a more mathematically detailed explanation for this inclusive fitness expression. That later section is aimed at readers with a technical interest in inclusive fitness modeling.

Table 3.1: Variables expression and definition

Expression	**Definition**
$P_{k,n} = \binom{n}{k} p^k (1-p)^{n-k}$	Probability that k broods fail on a patch with n breeders.
$h_k = \frac{(1-d)(n-k)}{(1-d)(n-k)+n(1-p)d(1-c))}$	Probability that a local offspring competes successfully on a patch where k breeders had brood failure
$\psi = \frac{p(1-s_0)+(1-p)(1-s_1)}{N(1-p)}$	Probability that an offspring finds a breeding spot, equivalent to its reproductive value
$\psi_k = \frac{k(1-s_0)+(n-k)(1-s_1)}{N(n-k)(1-d)+N(1-p)d(1-c)}$	Probability that an offspring competes successfully on a breeding spot where k breeders suffered brood failure, equivalent to its reproductive value on a patch where k breeders had brood failure
$\bar{r} = \frac{\alpha}{1-\beta}$	Coefficient of consanguinity (see B.2 for computation)
$R_s = \left(\bar{r} - \bar{r}\sum_{k=1}^{n} \frac{kP_{k,n}}{p(1-p^{n-1})} h_k\right) \Big/ \left(1 - \bar{r}\sum_{k=1}^{n} \frac{kP_{k,n}}{p(1-p^{n-1})} h_k\right)$	
$R_f = \frac{\left(\bar{r}d(1-c)\sum_{k=0}^{n} P_{k,n}\frac{\psi_k}{\psi} + \bar{r}(1-d)\sum_{k=1}^{n-1} \frac{kP_{k,n}}{p(1-p^{n-1})}\frac{\psi_k}{\psi}(1-h_k)\right)}{\left(1-\bar{r}\sum_{k=1}^{n} \frac{kP_{k,n}}{p(1-p^{n-1})} h_k\right)}$	

As mentioned, redirected helping decreases the survival of the helper (actor) and increases either the survival of another breeder (recipient) on the same patch or its fecundity. Fitness effects are always weighted by relatedness. The effects impacting the actor are weighted by relatedness to self equal to 1; those impacting a recipient on the same patch are weighted by R_s when considering survival benefits and R_f for fecundity benefits (see Table 3.1 for expression of relatedness coefficients). The relatedness coefficients weighting the impacts on a recipient on another patch equal 0. We use relatedness coefficients as [72] envisaged them: these coefficients measure relatedness in a way that reflects the scale of competition and the viscous nature of the population [72, 31, 78].

The costs and benefits of redirecting help are also weighted by the reproductive value of the individual whose fitness is changed. The reproductive value of an individual reflects its genetic contribution to future generations in the very distant future [23]. We set the reproductive value of a breeder at the beginning of the year to 1 without loss of generality. It follows that the reproductive value of an offspring is equal to the probability that it establishes itself as a breeder at the beginning of the next year, and we denote it as $\psi = \psi \cdot 1$. Similarly, the reproductive value of a breeder whose brood failed equals $s_0 = s_0 \cdot 1$, and that of a breeder whose brood succeeded is $s_1 = s_1 \cdot 1$. We express the reproductive value of an offspring in terms of the reproductive value of breeders as

$$\psi = \frac{p(1-s_0) + (1-p)(1-s_1)}{N(1-p)}. \tag{3.1}$$

We interpret equation (3.1) as the total reproductive value surrendered by adults who do not survive and divided by the total number of offspring.

After dispersal occurs, the ability of an offspring to obtain a breeding

spot depends on the number of breeders who experience brood failure on the patch it competes. We denote by ψ_k the reproductive value of an offspring competing on a patch with k brood failures after the dispersion of offspring occurs. Expression for ψ_k can be found in Table 3.1.

We are now able to state our version of Hamilton's rule for redirected help in a viscous population as

$$\text{Survival benefits: } -Cs_0 + R_s B_s s_1 > 0, \tag{3.2a}$$

$$\text{Fecundity benefits: } -Cs_0 + R_f B_f N\psi > 0. \tag{3.2b}$$

As presented, if conditions (3.2a) or (3.2b) hold, then redirected help is favored in the corresponding scenario. When the conditions are reversed, redirected help is disfavored. See appendix B.1 for detailed computations.

By setting (3.2a) or (3.2b) equal to 0 and solve for R_s and R_f, we can rearrange to $R_s^* = \frac{Cs_0}{s_1 B_s}$ and $R_f^* = \frac{Cs_0}{\psi N B_f}$. These two equations give us the critical survival and critical fecundity cost-benefit ratios; they set a maximum Cost:Benefit that supports redirected helping. As that critical ratio increases, redirected helping is favored more readily and for more cost-benefit ratios. As we change a given parameter, the cost-benefit ratio can increase and so the potential for the evolution of redirected help increases too, see Figure 3.1.

To compare the effect of each benefit, survival or fecundity, on the evolution of redirected helping in a selfish population, we compute the critical cost-benefit ratios R_s^* and R_f^* while varying different parameters such as the survival of the breeders, $s_0 = s_1 = s$, or the brood failure probability p.

All numerical computations and visualizations are performed using Julia (1.7.1) and can be found on this link.

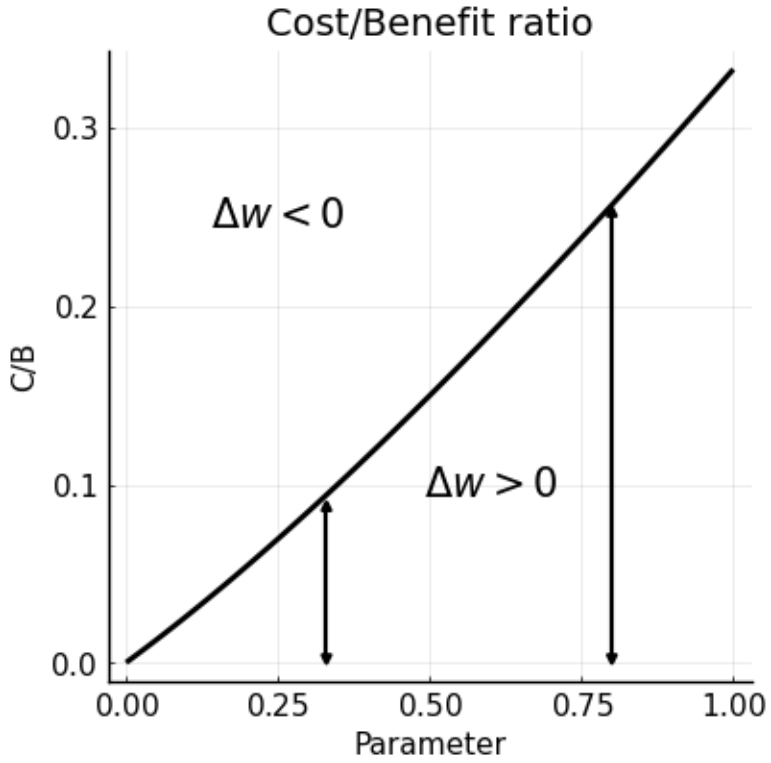

Figure 3.1: Variation of the critical Cost-Benefit ratio varying with a given parameter. As a given parameter changes, the critical cost-benefit ratio can also change, and the range of costs and benefits supporting redirected help changes, too. For example, when the critical cost-benefit ratio increases with a given parameter, redirected help is favored for a larger range of costs and benefits when the given parameter value is high.

3.2.3 Neighbor-modulated fitness and fitness change

In this subsection, we derive the inclusive fitness expressions in equations (3.2) using the neighbor-modulated fitness approach [90]. This approach is equivalent to the inclusive fitness method we developed in the previous section [92]. Consequently, the reader may skip this section without penalty.

We begin the neighbor-modulated fitness analysis by paying attention to an average breeder in the population. Let $z_\bullet$ be the probability that this focal individual helps given that its brood fails. Similarly, let $\bar{z}$ be the probability that the average (nonself) breeder on the same patch as the focal individual helps, given its brood fails. Finally, let z be the probability that the average breeder in the population helps given its brood fails.

The neighbor-modulated fitness of the focal individual, in the role of the recipient, is $w(z_\bullet, \bar{z})$. It is defined as the number of adult breeders in the next time step produced by a focal individual. As the notation suggests, neighbor-modulated fitness depends on the focal individual's phenotype and on the phenotype exhibited by the actor who influence the focal individuals. The focal individual's phenotype influences its survival; to capture this, we write $s_0 = s_0(z_\bullet)$. The actors in the neighborhood of the focal individual influence its survival and fecundity. Specifically, with probability $(1-p)P_{k,n-1}$ (Table 3.1), the focal individual is a successful breeder with exactly k brood failures on the same patch, so its survival is $s_1 = s_1(\frac{k\bar{z}}{n-k})$. Its fecundity is $f = f(\frac{k\bar{z}}{n-k})$. Given the life cycle described in a previous section, the neighbor-modulated

fitness of an individual is

$$w(z_\bullet, \bar{z}) = p(p^{n-1}s_0(0) + (1-p^{n-1})s_0(z_\bullet)) + (1-p)\sum_{k=0}^{n-1} P_{k,n-1} s_1\left(\frac{k\bar{z}}{n-k}\right) \tag{3.3}$$
$$+ (1-p)\sum_{k=0}^{n-1} P_{k,n-1} \underbrace{\frac{k(1-s_0(\bar{z})) + (n-k)(1-s_1\left(\frac{k\bar{z}}{n-k}\right))}{n-k}}_{\text{Number of local available breeding spots}} h_k(\bar{z})$$
$$+ (1-p)\sum_{k=0}^{n-1} P_{k,n-1} f\left(\frac{k\bar{z}}{n-k}\right) \sum_{i=0}^{n} P_{i,n} \underbrace{\frac{i(1-s_0(z)) + (n-i)(1-s_1(\frac{iz}{n-i}))}{\sum_{l=0}^{n} P_{l,n}(n-l) f(\frac{l\bar{z}}{n-l})}}_{\text{Number of non-local available breeding spots}} \cdot (1-h_i(\bar{z})),$$

where

$$h_k(\bar{z}) = \frac{(1-d) f(\frac{k\bar{z}}{n-k})(n-k)}{(1-d) f(\frac{k\bar{z}}{n-k})(n-k) + d(1 - c\sum_{l=0}^{n} P_{l,n}(n-l) f(\frac{l\bar{z}}{(n-l)}))}$$

now replaces h_k from Table 3.1. The argument in h_k denotes the average help received by the helped individual. This fitness expression combines both survival and fecundity effects. In addition, when $z_\bullet = \bar{z} = z$, this expression is equal to 1 as expected.

Four terms comprise the neighbor modulated fitness in Equation (3.3). The first term captures the survival of the focal individual when its brood fails. It also accounts for the fact that if all broods on the patch fail, no neighbor receives help. The second term describes the expected survival of the focal individual in the case where its brood does not fail. Expected survival in the second term is calculated by conditioning on how many of the $n-1$ other broods on the patch fail. The third and fourth terms capture the expected fecundity of the focal individual. The third term represents success through offspring that do not disperse and is calculated by conditioning on how many of the broods on this patch fail. The fourth term represents success through offspring that disperse and is calculated by conditioning on the number of

local broods that fail and the number of broods that fail where the dispersing offspring compete.

We now want to establish when help is advantageous in a population of selfish individuals. To do this, we define $x_\bullet$ as the genotypic value of the focal individual at the locus controlling redirected helping. We then assume that the phenotype of the focal individual is a function of $x_\bullet$ (i.e., $z_\bullet = z_\bullet(x_\bullet)$), and we do the same for the average non-self neighbor on the patch (i.e., $\bar{z} = \bar{z}(x_\bullet)$). Finally, we differentiate w with respect to $x_\bullet$ and evaluate it at $z_\bullet = \bar{z} = z = 0$ to obtain

$$\Delta w = \frac{\partial w}{\partial z_\bullet}\frac{dz_\bullet}{dx_\bullet} + \frac{\partial w}{\partial \bar{z}}\frac{d\bar{z}}{dx_\bullet}\Big]_{z_\bullet=\bar{z}=z=0} = \frac{\partial w}{\partial z_\bullet}1 + \frac{\partial w}{\partial \bar{z}}\bar{r}\Big]_{z_\bullet=\bar{z}=z=0}. \tag{3.4}$$

Where $\bar{r}$ is the coefficient of consanguinity between the focal individual and an average (non-self) breeder on the same patch. Note that we now use $\bar{r}$ to quantify relatedness. When Δw is positive, redirected helping is favored in a selfish population ($z = 0$) and when it is negative, redirected helping is disfavored.

Carrying the operation in equation (3.4) to equation (3.3), we obtain

$$\begin{aligned}
\Delta w = {} & s_0'(0)p(1-p^{n-1}) + s_1'(0)(1-p)\sum_{k=0}^{n-1} P_{k,n-1}\frac{k}{n-k} \\
& - s_0'\bar{r}(1-p)\sum_{k=0}^{n-1}\frac{k}{n-k}h_k(0) - s_1'\bar{r}(1-p)\sum_{k=0}^{n-1}\frac{k}{n-k}h_k(0) \\
& + \bar{r}(1-p)\sum_{k=0}^{n-1} P_{k,n-1}\frac{k(1-s_0(0)) + (n-k)(1-s_1(0))}{n-k}\frac{k}{n-k}h_k'(0) \\
& + f'(0)\bar{r}(1 \\
& - p)\sum_{k=0}^{n-1}\sum_{i=0}^{n} P_{k,n-1}\frac{k}{n-k}P_{i,n}\frac{i(1-s_0(0)) + (n-i)(1-s_1(0))}{f(0)n(1-p)}(1-h_i(0)).
\end{aligned} \tag{3.5}$$

Which can be reduced to

$$\begin{aligned}\Delta w/p = {} & s_0'(0)\sum_{k=1}^{n-1} P_{k-1,n-1}(1-\bar{r}h_k(0)) \\ & + s_1'(0)\bar{r}\sum_{k=1}^{n-1} P_{k-1,n-1}(1-h_k(0)) \\ & + f'(0)\bar{r}\sum_{k=1}^{n-1} P_{k-1,n-1}\Big(\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{f(0)(n-k)}h_k(0)(1 \\ & - h_k(0)) \\ & + \sum_{i=0}^{n} P_{i,n}\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{f(0)n(1-p)}(1-h_i(0))\Big).\end{aligned} \tag{3.6}$$

Note that survival and fecundity changes introduced previously correspond to terms in the previous equation in the following way: $\delta s_0 = s_0'(0)$, $\delta s_1 = s_1'(0)$ and $\delta f = f'(0)$. If δf is equal to 0, then equation (3.6) has the same sign as the left-hand side of the condition (3.2a) (survival benefits). If δs is equal to 0, then equation (3.6) has the same sign as the left-hand side of the condition (3.2b) (fecundity benefits).

To interpret (3.6), we focus on a helpful individual (the actor) established on a patch and in an otherwise selfish population. As suggested by the left-hand side of (3.6), we assume that the actor's brood fails, so it expresses its helpful tendency. The first term, on the right hand, reflects the reduced survival of the actor and the resulting reduction in competition experienced by all the offspring produced by the actor's neighbors. From a technical perspective, the actor reduces its survival, ultimately changing its reproductive value by $s_0'(0) \times 1$. Because the total reproductive value of all individuals in the population is fixed, the change in the actor's reproductive value must be compensated exactly by a change in the reproductive value of another (or others) in the opposite direction [31, 78]. If we assume that a total of k broods on the actor's patch failed (with probability $P_{k-1,n-1}$), then compensation is made

by one of the actor's patchmates with probability $h_k(0)$. Thus, the compensatory change alters the actor's inclusive by $-\bar{r}h_k(0)s_0'(0)$. Biologically, since the helper will survive at a lower rate, its breeding spot will become available at a higher probability. It follows that offspring, especially those born on this patch, will have another opportunity to win a breeding spot, increasing their fitness.

We interpret the second term on the right-hand side of (3.6) like the first. This term reflects the increased survival experienced by the actor's average neighbor and the resulting increase in competition experienced by all offspring produced by these neighbors. Here, the actor changes the reproductive value of a neighbor by $s_1'(0)$ and is related to that neighbor by $\bar{r}$. Again, assuming that a total of k broods on the actor's patch failed, the change in reproductive value is compensated by another of the actor's neighbors with probability $h_k(0)$. This ultimately changes the actor's inclusive fitness by $-\bar{r}h_k(0)s_1'(0)$. Biologically, the increased survival of breeders who succeeded on this patch decreases the probability that their breeding spot will become available for competing offspring, leading to a reduction in the offspring's fitness.

We interpret the third term in (3.6) as the net effect of increased fledging success, $f'(0)$, for offspring competing for a breeding spot on the local patch with probability $h_k(0)$, first term, and dispersing to compete on another patch with i brood failure among the n breeding spots with probability $1 - h_i(0)$, second term. This accounts for the increased fledging success as a benefit and increased competition on the local patch ($-h_k(0)$). Indeed, as more offspring become adults, more individuals compete for a breeding spot, leading to a decrease of the fitness of offspring. The increased competition on non-local patches is not considered as it impacts unrelated individuals, i.e coefficient of consanguinity equal to 0.

3.3 Results

3.3.1 Low brood failure probability $p \approx 0$

We first compute the critical cost-benefit ratio under survival or fecundity benefits only, assuming brood failure probability, p, is small. Biologically, small brood failure probability means the focal actor is the only individual suffering brood failure. We also look at the backward migration rate, $m = 1 - h_0$, which gives the fraction of non-local competitors on a given patch when the brood failure probability is low. This rate gives an idea of how well mixed the population is.

As one should expect, the critical cost-benefit ratios respond to model parameter changes. The critical cost-benefit ratios increase as the patch size, n, decreases (Figure 3.2 top and middle row left to right panels). This means that helping emerges more readily when the patch size is small. A decrease in the backward migration rate, m, has the same effect: it raises the critical cost-benefit ratios, and in so doing, it promotes helping (Figure 3.2 top and middle rows). As we change adult survival, s, the critical cost-benefit ratios vary depending on the nature of the benefits. Specifically, the critical cost-benefit ratio associated with survival benefits increases with the probability of survival, s (Figure 3.2 top row). However, the cost-benefit ratio associated with fecundity benefits responds non-monotonically, often decreasing with s (Figure 3.2 Middle row). Thus, increasing the probability of adult survival promotes helping with survival benefits and often hinders helping with fecundity benefits.

The effects of the parameters on relatedness explain most but not all of these results. As we decrease the patch size, n, decrease the backward migra-

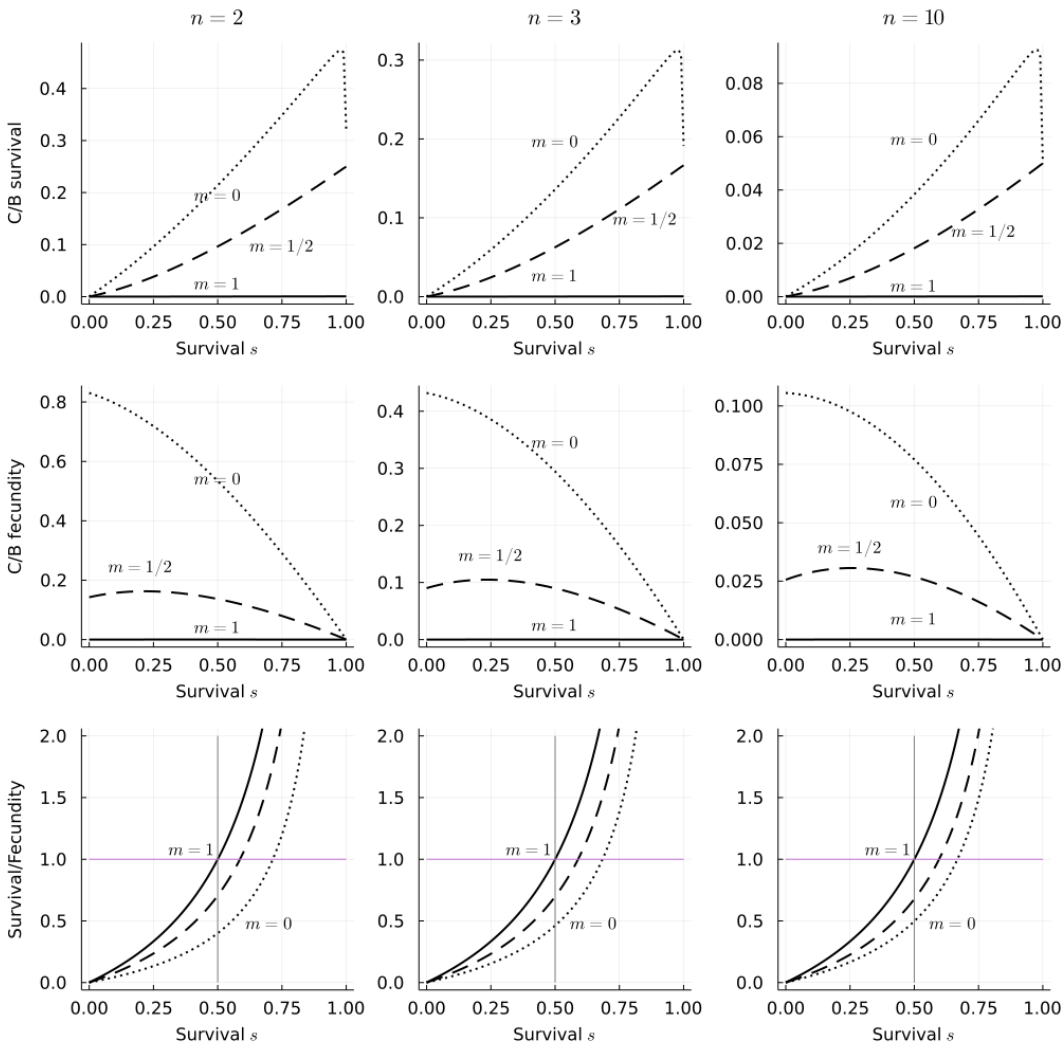

Figure 3.2: Panel Top row: Critical cost-benefit ratio associated with survival benefits (y-axis) depending on the survival of breeders (x-axis), backward migration rate, m, and patch size, n. Dotted lines, $m \approx 0$, dashed lines, $m \approx 1/2$, solid lines, $m \approx 1$. Left panel, $n = 2$, middle panel, $n = 3$, right panel, $n = 10$. As a recall, the higher the line, the easier for redirected help to emerge. Middle row: Critical cost-benefit ratio associated with fecundity benefits. Parameters and legend are identical as in top row panels. Bottom panels: ratio of the cost-benefits associated with survival benefits over fecundity benefits. Dotted lines, $m \approx 0$, dashed lines, $m \approx 1/2$, solid lines, $m \approx 1$. Left panel, $n = 2$, middle panel, $n = 3$, right panel, $n = 10$. The purple line represents an equal value of both critical cost-benefit ratios. Over the purple line, survival is a better driver for redirected help; under it, fecundity is a better driver. Grey line: $s = 0.5$. Other parameters used: $p \approx 0$.

tion rate, m, and increase the generation overlap through s, the relatedness increases. Naively, helping is promoted by changes in these parameters because of greater relatedness [38].

In the case where helping provides fecundity benefits, we must consider both relatedness and the reproductive value of offspring. As we increase s, relatedness increases, but the reproductive value of offspring decreases (See Table 3.1, ψ, for the expression of the reproductive value). For lower values of s, the decrease in reproductive value is outweighed by the increase in relatedness; in these cases, the response of the critical cost-benefit ratio is understood as a relatedness effect. For higher values of s, the decrease in reproductive value exceeds the increase in relatedness; in these cases (which predominate our results), the response is understood as being driven by the reproductive value of offspring.

We can directly compare the critical cost-benefits ratios under survival or fecundity benefits to determine which is more effective at promoting redirected helping. For small p, the comparison is made using

$$\frac{sR_s^*}{\psi N R_f^*} \sim \frac{s}{1-s}\left(\frac{n(n-1+m)}{(n-1+m)^2+(1-m)n^2}\right), \tag{3.7}$$

where R_s^* and R_f^* are the critical costs:benefits ratios, i.e., the relatedness coefficients that satisfy $-Cs_0 + R_s B_s s_1 = 0$ and $-Cs_0 + R_f B_f N\psi = 0$, respectively. When equation (3.7) exceeds 1, survival benefits promote helping more readily than fecundity benefits; the reverse is true when the expression is less than 1. From equation (3.7), it is clear that raising adult survival probability strengthens the ability of survival benefits to promote helping compared to fecundity benefits (see also Figure 3.2 bottom row). This result differs from one reported by [91], who found that fecundity benefits are always more effective

than survival benefits at promoting help. We come back to this point in the discussion.

We know that we can increase s to a point where survival benefits are better at promoting helping than fecundity benefits. Our question now is, where does this point lie?

We know that the term in brackets in (3.7) lies between 0 and 1. Thus, any survival probability for which survival benefits are better is no less than the point $s = 1/2$. We move this point ever closer to $s = 1/2$ as we increase the backward migration rate, m (Figure 3.2 bottom row). The value of s beyond which survival benefits become better at promoting helping also respond to changes in patch size n. The impact of the patch size is not monotonic in general, but in the vast majority of cases of the results, a larger patch size leads to moving the tipping point closer to $s = 1/2$ (Figure 3.2 bottom row).

3.3.2 Larger brood failure probability $p > 0$

In the larger brood failure probability, critical cost-benefit ratios were again positive almost everywhere in the space of model parameters (Figures 3.3 and 3.5). Thus, there is almost always scope for the advantage of redirected helping when the number of brood failures varies among patches. Population viscosity and the local competition that results do not eliminate the possibility that redirected helping is advantageous.

All else being equal, we found that decreasing patch size n promotes the advantage of redirected helping in the variable-failures scenario (Figures 3.3 and 3.5). Generically, reduced mixing among patches (i.e. lower backward migration rates m) also promotes redirected helping.

The qualitative effect of increasing the brood-failure probability, p, de-

pends on the nature of the benefits conferred by help. With survival benefits, increasing p inhibits redirected helping in some cases and promotes it in others (Figure 3.3). Specifically, increasing p tends to inhibit redirected helping on smaller patches when there is less mixing among patches or both. Conversely, increasing p tends to promote redirected helping when patches are larger and mixing among patches is more extensive (m large).

When helping confers fecundity benefits, the qualitative effect of p promotes redirected helping (Figure 3.5). We conjecture that this qualitative agreement arises (at least in part) because some portion of fecundity benefits can be exported to other patches. That is to say, some fraction of the extra offspring resulting from help leave their natal patch and experience the average environment.

For the survival-benefits model, increasing s promotes redirected helping when the brood-failure probability p is smaller and inhibits helping when p is larger (Figure 3.3). For the fecundity-benefits model, increasing survival probability s uniformly inhibits redirected helping (Figure 3.5).

As in the case where p was closer to 0, we can compare how well each benefit drives redirected helping by comparing their respective critical cost-benefit ratios. As expected from our previous results, we see that survival benefits drive the evolution of help better than fecundity benefits when the survival is high and the brood failure probability is low (Figure 3.6). We also observe that as we decrease the backward migration rate m, we increase the range of brood failure and survival probabilities for which survival benefits are a better driver of helping than fecundity benefits (Figure 3.6). The set of parameters for which survival benefits are a better driver of helping than fecundity benefits becomes less sensitive to changes in the patch size for low values of the backward migration rate (Figure 3.6).

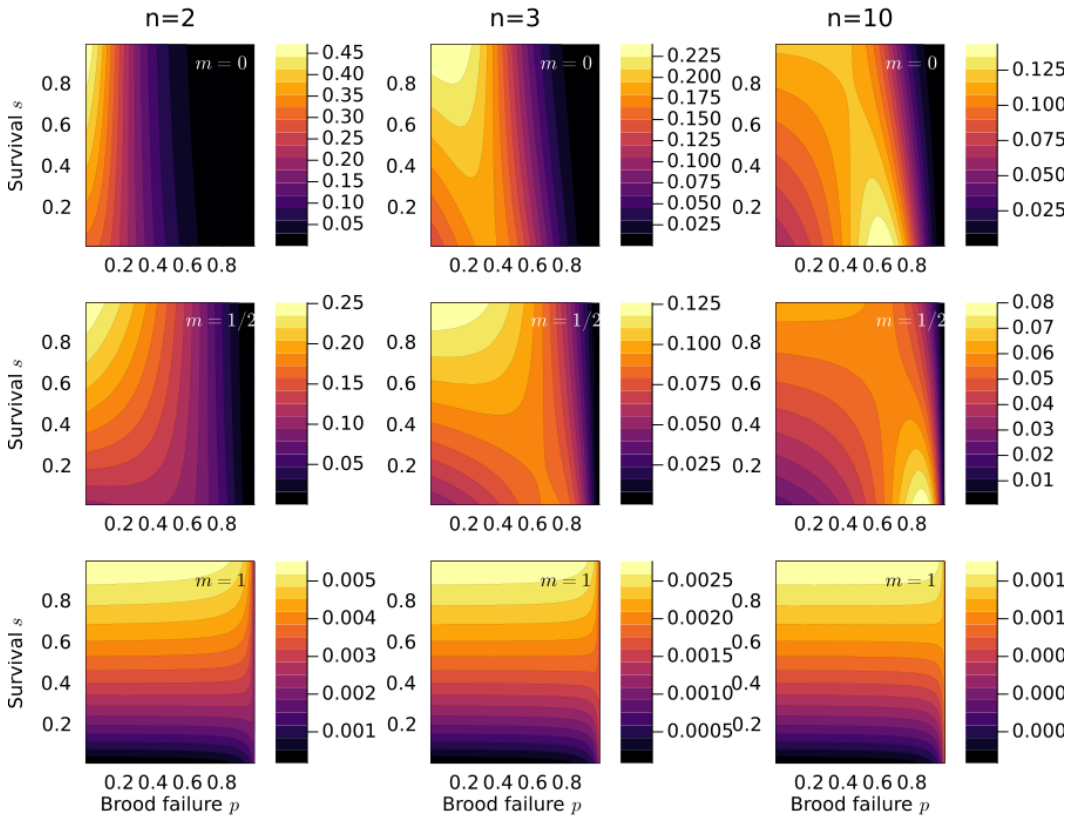

Figure 3.3: Contour plots of the critical cost-benefit ratio associated with survival benefits depending on the survival (y-axis), brood failure probability (x-axis), patch size n, and backward migration rate m. Left column, $n = 2$, middle column, $n = 3$, right column, $n = 10$. Top row, $m \approx 0$, middle row, $m = 1/2$, bottom row, $m \approx 1$. The color bar remains the same by column. A higher value on the color bar means that redirected help can emerge more easily.

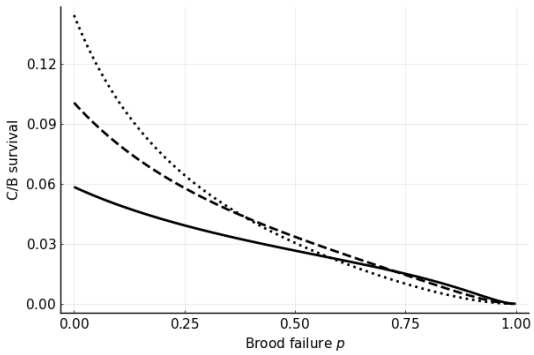

Figure 3.4: Critical cost-benefit ratio associated with survival benefits for different patch sizes: dotted lines, $n = 2$, dashed lines, $n = 3$, solid lines: $n = 10$. Adult survival equals $s = 0.5$, and backward migration rate $m \approx 0$. Recall that a high value on the y-axis means that redirected help is more easily favored.

3.4 Discussion

We derive a version of Hamilton's rule to capture the advantage of redirected helping in a viscous population. We found that redirected helping can emerge when it confers fecundity and survival benefits respectively. Life history features that increase relatedness among neighbors also promote the emergence of redirected helping. The relatedness measure we used accounted for local competition among kin [72, 78]. Therefore, our conclusions automatically account for the inclusive fitness losses that hinder helping in other model scenarios [e.g. 88]. Life history features that increase the reproductive value of offspring also promote the emergence of redirected helping when that helping is associated with fecundity benefits. Our conclusions align with previous accounts of kin selection and reproductive skew [49, 1]. In that theory, the recipients become a more attractive target for helping as they become more valuable. Here, the same is true for offspring in our model as the brood failure goes up and adult's survival goes down.

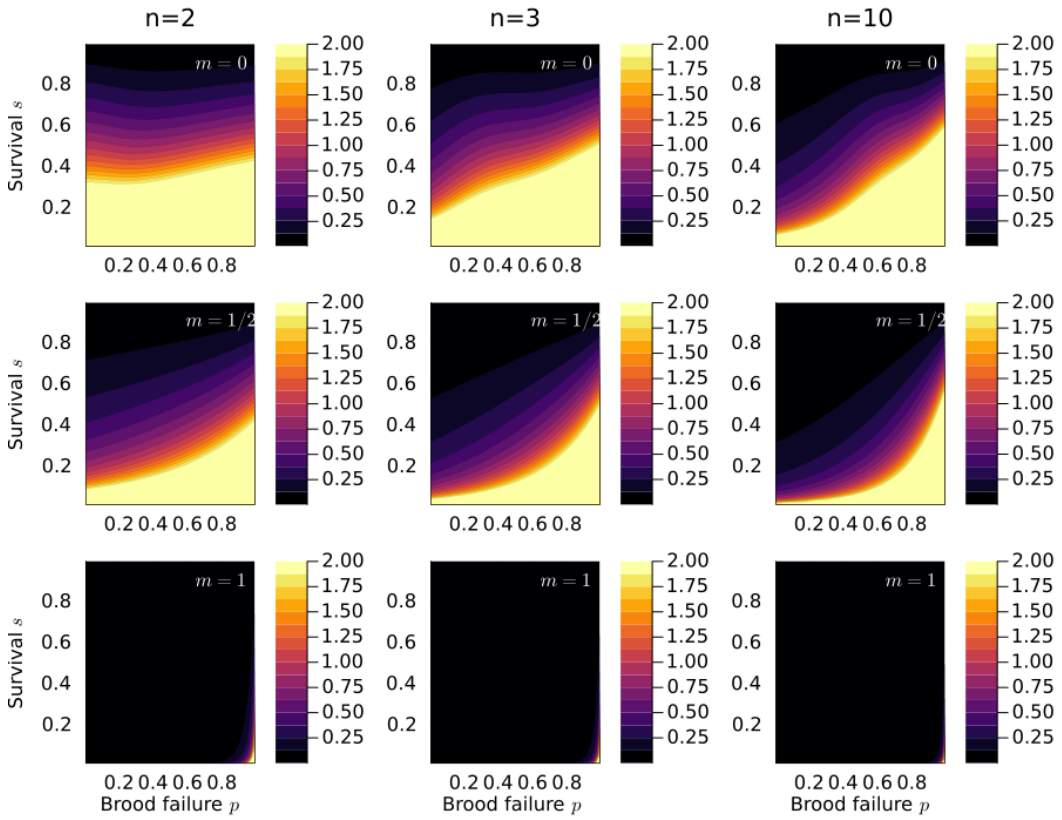

Figure 3.5: Contour plots of the critical cost-benefit ratio associated with fecundity benefits depending on the survival (y-axis), brood failure probability (x-axis), patch size n, and backward migration rate m. Left column, $n = 2$, middle column, $n = 3$, right column, $n = 10$. Top row, $m \approx 0$, middle row, $m = 1/2$, bottom row, $m \approx 1$. The color bar remains the same by column. A higher value on the color bar means that redirected help can emerge more easily. We capped the value of the critical cost-benefit ratio to 2.

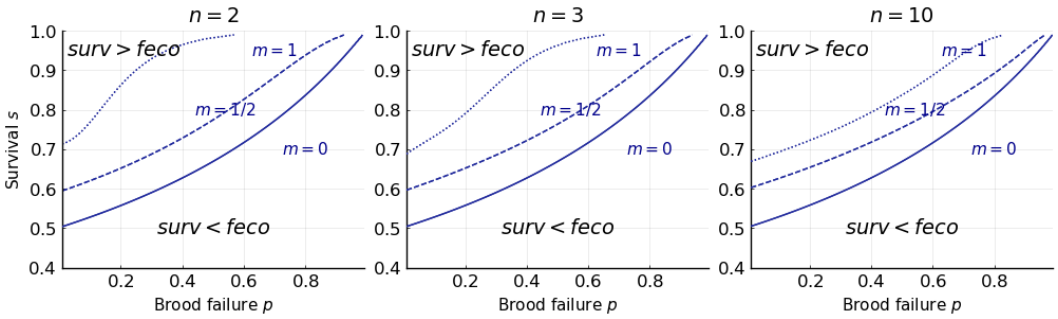

Figure 3.6: Equal level of the critical cost-benefit ratios associated with survival and fecundity ($sR_s = N\psi R_f$) depending on breeder's survival (y-axis), brood failure probability (x-axis), patch size, n, and backward migration rate. When a point (p, s) is higher than a curve, then survival benefits are more effective in promoting helping than fecundity benefits for this set of parameters (surv>feco). The inverse is true when the point is located below the curve. Left column, $n = 2$, middle column, $n = 3$, right column, $n = 10$. Dotted lines, $m = 0$, dashed lines, $m = 1/2$, solid lines, $m = 1$.

We found that survival benefits can promote redirected helping better than fecundity benefits. This result contrasts with [48], where authors found that fecundity benefits are always a better driver for the evolution of helping than survival benefit. To understand the difference between our model and the one presented in one must first understand what drives the results in these articles.

The results of [48, 91] can be understood by considering that the total reproductive value of a population is fixed. So, if the reproductive value of an individual increases – say because it receives help– then the reproductive value of other individuals decreases to compensate. Conversely, if the reproductive value of an individual decreases – say because it provides help– then the reproductive value of other individuals increases to compensate. When the actor's relatives make the compensation, it appears on the actor's inclusive fitness balance sheet [78, 32]. In the case of [48, 91], a portion of the net benefits created when helping affect fecundity is exported. The same cannot

be said when helping affects the survival of adults. Thus, the compensation associated with fecundity benefits is less of an inclusive fitness liability for the actor.

One might think that the best evolutionary case for helping occurs when (a) the compensatory losses of reproductive value that follow the benefits of helping are imposed disproportionately on non-relatives, while (b) the compensatory gains that follow the cost of helping are shared disproportionately with relatives. In other words, one might think that the scenario most conducive to helping is that benefits are paid in fecundity and costs are paid in survival. However, as [48, 91] argued, personal survival effects can be converted into personal fecundity effects and vice versa in their model. Thus, their conclusion that helping is promoted more strongly with fecundity benefits cannot be changed by adjusting the component of fitness affected by helping itself.

In our model, personal survival effects cannot be converted into personal fecundity effects as the helper is a failed breeder and so has no fecundity to convert to, at least immediately. The cost has to be paid in survival, so we cannot always restrict our discussion to only one fitness component, unlike [48, 91]. In our model, all else being equal, helping will be more promoted if compensatory reductions in reproductive value are exported, but all else is not always equal. With redirected helping, we have an extra degree of freedom in the form of relative reproductive value of offspring and adults, which means that survival benefits can outperform fecundity benefits even though we cannot export compensatory costs to non-relatives.

Our results offer a new perspective on the findings of a recent meta-analysis of data from 23 cooperatively breeding bird species, including ones that engage in redirected help [14]. This meta-analysis concluded that help promotes breeder longevity by allowing the breeder to reduce its investment in offspring,

i.e. load-lightening. By contrast, our results show that greater longevity of breeders promotes helping: greater breeder longevity increases both relatedness and breeder reproductive value, raising the inclusive-fitness benefits of helping. In this way, our findings add to the long-standing theory that casts cooperative breeding as an adaptive response to the ecological constraints imposed by long-lived breeders [17, 6]. Though we emphasize a different causal connection between helping and breeder longevity than previous authors [14], our different perspectives are not mutually exclusive. In fact, we expect that helping increases and responds to changes in the lifespan of a breeder in a way that creates positive feedback between high survival and helping. This co-evolution of helping behavior and load-lightening would be an interesting direction to explore.

Our work has demonstrated that the survival benefits of redirected helping can promote helping more strongly than fecundity benefits. The clear long-term perspective provided by reproductive value was key to understanding the contrast between benefits achieved through survival versus fecundity. In our model, the fecundity benefits of help may be low relative to survival benefits simply because the long-term outlook for offspring is poor. In this way, our work underscores the importance of a long-term perspective when studying redirected helping and, more generally, cooperative breeding. With that in mind, revisiting studies that emphasize this short/long-term perspective may be fruitful. One example here is the evolution of redirected helping in Long-tailed Tits (*Aegithalos caudatus*)[45]. Long-tailed Tits who experience brood failure redirect their parental care to related neighbors by feeding the offspring produced by those neighbors [53]. In this way, the helpers increase fledging success, corresponding to fecundity benefits in our model. However, the long-term effects of helping benefits must be considered to make the correspondence

as close as possible. While the impact of helping on the survival of fledging or breeders has been studied before — and has been studied longitudinally — it has mostly been studied using what we would call a short-term perspective (43, 63; but see 64). Granted, there are constraints on pursuing what theorists would call long-term studies, but feasible solutions could include creating a matrix population model that describes the life history of the species of interest. Such models provide information on the reproductive value of individuals and can project the consequences of helping far into the future [7]. These models can also help us disentangle the complicated long-term, indirect effects of helping, e.g., an effect mediated by habitat availability and resource competition.

Our model has ignored a prominent feature in the biology of redirected helping, namely kin recognition. In the Long-tailed Tits, recipients of help are identified by kin recognition mechanisms [43]. Although we have neglected kin recognition, we expect a more complicated model incorporating this feature will make the same qualitative prediction. Adding kin recognition may change where the critical cost:benefit thresholds are, making them less restrictive as seen in other work [16]. However, one must be careful about considering the geometry of the returns before committing to such conclusions [21].

In summary, we used an inclusive fitness model to show that redirected helping arises when helping either increases fecundity or the survival of relatives. Our model captures the demographic stochasticity associated with brood failure in social groups of finite size. Our key findings contrast with previous theories by demonstrating that the survival benefits of helping can be a potent driver of helping. We also demonstrated that various life-history and environmental features mediate this potent driver. Our study of redirected helping provides a new perspective on cooperative breeding and points to new

field and theoretical studies.

Chapter 4

Evolution of redirected helping in a population over a stepping stone model

4.1 Introduction

When a parent's brood of offspring fails, it may redirect its efforts to help a neighbor raise that neighbor's offspring. This phenomenon is known as "redirected help" and occurs often in nature [43, 13]. It may seem that redirected helping confers an obvious selective advantage: an individual who misses an opportunity to accrue fitness through their own offspring may still accrue inclusive fitness [38] by redirecting its help, specifically toward related neighbors. However, redirected helping faces evolutionary obstacles when individuals compete with their relatives in addition to helping them. Competition among relatives is the hallmark of a "viscous population", i.e., a population in which dispersal is limited. Population viscosity imposes indirect costs on the

helper that may be sufficient to cancel out any benefits that help generates for itself [88, 89].

We have recently investigated the evolution of redirected helping in a viscous population and found that its selective advantage is certainly not guaranteed in Chapter 3. We found that the interaction between selective advantage and population viscosity changes depending on whether redirected help brings survival or fecundity benefits to a neighbor. In particular, we found that redirected helping can more readily overcome the evolutionary obstacles associated with a viscous population when helping provides survival benefits, as compared to fecundity benefits. This finding challenged the established idea that fecundity benefits always promote the evolution of helping more effectively than survival benefits [48, 91].

Our recent findings in Chapter 3 are based on an infinite-island model. In such a model, space is ignored, and individuals are arranged into islands. Importantly, dispersal from an island in an infinite-island model scatters individuals so far that they are guaranteed not to compete with their kin. In this way, dispersal in an infinite-island model provides a strong antidote to population viscosity. However, individuals who disperse may still compete with kin because dispersal occurs locally or over relatively short distances. Thus, dispersal may not counterbalance population viscosity to the extent we think it does based on infinite-island models alone. If we want to understand how population viscosity affects the evolution of redirected helping, we need models that capture space in an explicit way.

In this paper, we use a one-dimensional stepping-stone model to study how spatial structuring of the environment can affect the evolution of redirected help. By constraining dispersal to neighboring sites, we restore a portion of the population viscosity lost in our previous model due to the infinite-island

spatial structure. Looking at the influence of the environment, we ask how the environmental parameters can select the type of benefits driving the emergence of redirected help.

To answer our questions, we build a kin selection argument for the advantage of redirected helping that recognizes that both help and competition occur among relatives [90]. We find that the incorporation of explicit spatial structure leads to different population dynamics and impacts the evolution of redirected help. We also show that different benefits can be selected to drive the emergence of help in different environments.

4.2 Methods

4.2.1 Life cycle

We consider a population of haploid asexual individuals living on breeding spots that are, themselves, arranged on a one-dimensional stepping-stone model. We keep the dimension of our lattice to a minimum to constrain the dispersal. Each breeding spot supports exactly one individual. Each individual at spot i has 2 neighbors at sites $i-1$ and $i+1$ where $i \in \mathbb{Z}$.

We observe the population at discrete time steps (e.g., every year). At the beginning of every time step, each individual produces a brood of N offspring, where N is large. Each brood fails independently with probability p; this can happen, for example, because of predation. In the case of a failure, the individual has no offspring during this step. Each offspring from a successful brood matures independently (fledging) with probability F and disperses to one of the two neighboring breeding spots with probability d; half disperse to the left and the other half to the right. Offspring can also disperse to an

infinitely far breeding spot with probability μ. We suppose μ is very small, meaning that terms in $O(\mu)$ are negligible but not terms in $O(\sqrt{\mu})$. Dispersing offspring die with probability c during dispersal. Non-dispersing offspring (probability $1-d$) remain in their natal breeding spot.

Owners of breeding spots experience mortality after the offspring dispersal phase. An owner who fails its brood survives at s_0, while a successful breeder survives at s_1. If the owner survives, it remains in its breeding spot; otherwise, the breeding spot becomes vacant. Offspring on a vacant breeding spot, after dispersing or not, can compete for its ownership. They compete equally, and the breeding spot is occupied by one offspring, who is taken randomly among the ones found on the breeding spot. Offspring die if they do not successfully compete for a breeding spot.

4.2.2 Neighbor-modulated fitness

When the individual at site i suffers brood failure, it decides to help one of its two direct neighbors with probability z_i. This help is provided if at least one of its two neighbors experiences brood failure. If only one neighbor suffers brood failure, all the help is directed to it. If both neighbors suffer brood failure, help is shared equally between the two. If no neighbors suffer brood failure, no help is given.

Help can increase the survival of the neighbors by a small amount or the probability at which offspring becomes mature/fledges by a small amount (fecundity/fledging benefits). However, by helping, the helper decreases its own survival by a small amount. We then denote the survival rates by $s_0 = s_0(z_i)$, $s_1 = s_1(z_i)$ and the fledging success by $F = F(z_i)$. These small changes in survival or fecundity reflect a weak selection assumption, commonly used in

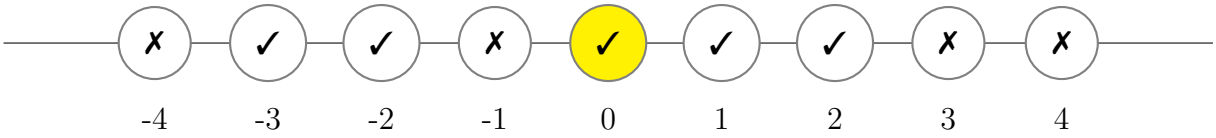

Figure 4.1: Example of brood successes and failures of the neighboring breeding spot (white circles) around a focal individual at breeding site 0 (yellow circle). A ✗ represents a brood failure, while a ✓ represents a brood success.

inclusive fitness works [30, 86, 100]. We will compare the impact of the survival and fecundity benefits on the evolution of redirected help.

We use a neighbor-modulated fitness approach to evaluate the evolution of redirected help in a selfish population [90]. First, we focus on a random individual at the beginning of a time step (focal individual) and assume, without loss of generality, that it is located at the breeding spot $i = 0$. We use z_0 to denote the probability that the focal individual attempts to help its neighbors at sites -1 or 1 when it experiences brood failure. We use z_i to denote the probability that the individual at breeding spot i will help its neighbor at step $i-1$ or $i+1$ (given that its brood fails). We use the vector $\vec{z} = (...z_{-1}, z_0, z_1, ...)$ to store the helping rates of each individual in the population.

The neighbor-modulated fitness of an individual, $w(\vec{z})$, is measured as the number of breeding spots the focal individual and its direct descendant occupy after one time step. Thus, $w(\vec{z})$ captures both the survival of the focal individual and the capacity of its offspring to compete successfully for breeding spots.

When computing the focal individual's neighbor-modulated fitness, we must consider its neighbors' brood successes and failures. For instance, if we consider the case in Figure 4.1 associated with survival benefits, we observe that the neighbor-modulated fitness of the focal individual is equal to

$$\underbrace{s_1(z_{-1}/2)}_{\text{Survival}} + \underbrace{(1-s_1(z_{-1}/2))\frac{1-d}{1-d/2(1+c)}}_{\text{Competition on local breeding spot}} + \underbrace{(1-s_0(z_{-1}))\frac{1}{2}}_{\text{Competition on breeding spot -1}} + \underbrace{(1-s_1(0))\frac{d/2(1-c)}{1-cd}}_{\text{Competition on breeding spot +1}} + \underbrace{O(\mu)}_{\text{Competition far away, negligible}}, \tag{4.1}$$

when considering survival benefits. If we consider fecundity benefits, the neighbor-fitness of the focal individual is

$$s_1(0) + (1-s_1(0))\frac{(1-d)F(z_{-1}/2)}{(1-d)F(z_{-1}/2) + d/2(1-c)F(0)} + (1-s_0(z_{-1}))\frac{1}{2} + (1-s_0)\frac{d/2(1-c)F(z_{-1}/2)}{d/2(1-c)F(z_{-1}/2) + (1-d)F(0) + d/2(1-c)F(z_3)} + O(\mu). \tag{4.2}$$

The terms in (4.1) and (4.2) include the survival of the focal individual itself (first term), the probability one of its offspring competes successfully on its breeding site (if the focal individual does not survive), (second term), the ability of the offspring to compete successfully on a neighbors breeding spot (-1 and +1), (third and fourth terms) and the ability for its offspring to compete successfully on a breeding spot far away (fifth term). This last term is negligible as we suppose μ is small.

We determine the focal individual's overall neighbor-modulated fitness by computing a weighted average over every possible neighborhood using the frequency of the neighborhoods as weights (e.g. the case in Figure 4.1 is weighted by $p^4(1-p)^5$, which is its probability of occurring). This averaging scheme is appropriate because neighborhood frequency can also be interpreted as class reproductive value (see [87, 77]).

4.2.3 Neighbor-modulated fitness change

We suppose redirected help z_i is determined at a single locus. Recall that our population is haploid and asexual. Let x_0 denote the focal individual's genotypic value at this locus. Following [90], we treat each z_i as a function of x_0. We compute the change in the neighbor-modulated fitness of the focal individual by differentiating $w(\vec{z})$ with respect to x_0 as $\frac{dw}{dx_0}$. We then evaluate the derivative in a selfish population ($z_i = 0, \forall i \in \mathbb{Z}$) to obtain

$$\sum_{i=-\infty}^{\infty} \frac{\partial w}{\partial z_i} \frac{dz_i}{dx_0}\bigg|_{\vec{z}=\vec{0}}. \tag{4.3}$$

We then replace $\frac{dz_i}{dx_0}$ by r_i the coefficient of consanguinity between the focal individual and its neighbor i steps to obtain,

$$\Delta w = \sum_{i=-\infty}^{\infty} \frac{\partial w}{\partial z_i} r_i. \tag{4.4}$$

Computations of r_i coefficients can be found in Section 4.2.4. Remember that the survival and fledging success are measured in a selfish population $\vec{z} = \vec{0}$. When (4.4) is positive, redirected help is favored and emerges. When (4.4) is negative, redirected help is disfavored, and does not emerge.

While $w(\vec{z})$ looks at how an individual is affected by its neighbors (recipient-centered), we can interpret (4.4) as the effects an actor has on its neighbors (actor-centered) [92]. The change in perspective means that (4.4) can be understood in the way Hamilton [38] originally envisioned his theory.

I now define $C = s_0'(0)/s_0 < 0$ as the survival cost of helping, $B_s = s_1'(0)/s_1 > 0$ as the increase in the survival for the helped breeder, and $B_f = F'(0)/F(0)$ as the increase in fecundity/fledging success for the helped breeder.

As an example of neighbor-modulated fitness change, when we look at the

case of Figure 4.1, we have

$$r_1\left(\frac{s_1'(0)}{2}\left(1-\frac{1-d}{1-d+d/2(1-c)}\right)-\frac{s_0'(0)}{2}\right)=\frac{s_1}{2}r_1\left(B_s\left(1-\frac{1-d}{1-d+d/2(1-c)}\right)-Cs_0\right) \tag{4.5a}$$

for survival benefits and

$$\begin{aligned}&-r_1Cs_0\\&+r_1B_fF(0)\left((1-s_0)\frac{1-d}{1-d/2(1+c)}+(1-s_1)\frac{d/2(1-c)}{1-d/2(1+c)}\right.\\&\left.-(1-s_0)\left(\frac{1-d}{1-d/2(1+c)}\right)^2-(1-s_1)\left(\frac{d/2(1-c)}{1-d/2(1+c)}\right)^2\right)\\&-r_3F(0)(1-s_0)\left(\frac{d/2(1-c)}{1-d/2(1+c)}\right)^2\end{aligned} \tag{4.5b}$$

for fecundity benefits. In (4.4), $C=s_0'(0)/s_0<0$ is the cost of helping for the helper, $B_s=s_1'(0)/s_1>0$ is the increase in survival for the helped breeder, and B_f is the increase in fecundity/fledging success for the helped breeder. When (4.4) is positive, redirected help is favored and emerges. When (4.4) is negative, redirected help is disfavored and does not emerge.

4.2.4 Coefficients of consanguinity

Neighbor-modulated fitness computations depend on the coefficients of consanguinity r_i, between the focal individual and a neighbor i steps away. We define the coefficient of consanguinity (CC) between two individuals as the probability that the allele carried by one and the allele carried by the other are identical by descent [65]. We use the method developed in [84] to compute the coefficients between a focal individual and its i step neighbor [see also 51]. We assume a selfish population $z_i=0,\forall i\in\mathbb{Z}$ as we look at the emergence of

help in a selfish population. The assumption is justified because changes in neighbor-modulated fitness represented by Equation (4.4) are estimated to be first-order in the strength of selection [86].

We start by defining ρ_0 as the probability that a breeding spot is retained by its current owner or obtained by one of the current owner's offspring. We use ρ_1 to denote the probability that a breeding spot is obtained by an individual born one site away, an offspring born on the breeding spot on the left or the right. The quantities ρ_0 and ρ_1 depend on the different parameters used, such as brood failure probability, p, dispersal rates d, and μ, etc. We observe that each coefficient r_i satisfies $r_i = A(r_{i-2} + r_{i+2}) + B(r_{i-1} + r_{i+1}) + Dr_i$, where $A = \rho_1^2$, $B = 2\rho_0\rho_1$ and $D = \rho_0^2 + 2\rho_1^2$. Coefficients r_i satisfy the recursive equation

$$\begin{pmatrix} r_{i+2} \\ r_{i+1} \\ r_i \\ r_{i-1} \end{pmatrix} = \frac{1}{A} \begin{pmatrix} -B & 1-D & -B & A \\ A & 0 & 0 & 0 \\ 0 & A & 0 & 0 \\ 0 & 0 & A & 0 \end{pmatrix} \begin{pmatrix} r_{i+1} \\ r_i \\ r_{i-1} \\ r_{i-2} \end{pmatrix} \tag{4.6}$$

subject to $r_0 = 1$ as an individual is surely identical by descent to itself and $r_i = r_{-i}$ as the population disperses symmetrically.

The solutions of (4.6) are linear combinations of the eigenvectors of the matrix, which has form $v_k = \lambda^k$ where λ is an eigenvalue and k is the index of the coefficient of consanguinity. In the limit as $\mu \to 0$, to first order in $\sqrt{\mu}$, we know that there are two eigenvalues whose modules are between 0 and 1, as we want coefficients of consanguinity to be bounded between 0 and 1, we only consider these two eigenvalues only, l_1 and l_2. Finally, given that $r_0 = 1$

and that we must have $r_1 = r_{-1}$, we obtain the following coefficients

$$\begin{aligned} r_0 =& 1 \\ r_1 =& c_1 l_1 + c_2 l_2 \\ r_2 =& c_1 l_1^2 + c_2 l_2^2 \\ r_3 =& c_1 l_1^3 + c_2 l_2^3, \end{aligned}$$

where $c_1 = \frac{l_1 - l_1 l_2^2}{l_1^2 l_2 - l_1 l_2^2 + l_1 - l_2}$ and $c_2 = 1 - c_1$.

4.3 Results

4.3.1 Small brood failure probability p

When the brood failure probability is almost zero, we can approximate the neighbor-modulated fitness associated with survival and fecundity benefits. For survival benefits, we have

$$\Delta w(\vec{z}) = -C s_0 (1 - r_1) + B_s s_1 (r_1 - r_2) \frac{d/2(1-c)}{1 - d/2(1+c)}, \tag{4.7}$$

and

$$\begin{aligned} \Delta w(\vec{z}) = & -C s_0 (1 - r_1) + B_f F r_1 \Bigg[(1 - s_0) \frac{1-d}{1 - d/2(1+c)} \\ & - (1 - s_0) \left(\frac{1-d}{1 - d/2(1+c)} \right)^2 + (1 - s_1) \frac{d/2(1-c)}{1 - cd} - (1 - s_1) \left(\frac{d/2(1-c)}{1 - cd} \right)^2 \Bigg] \\ & - r_2 B_f F (1 - s_1) \left(\frac{d/2(1-c)(1-d)}{(1 - d/2(1-c))^2} + \frac{1-d}{1-cd} \frac{d/2(1-c)}{1-cd} \right) \\ & - r_3 B_f F (1 - s_1) \left(\frac{d/2(1-c)}{1-cd} \right)^2, \end{aligned} \tag{4.8}$$

for fecundity benefits. Again, we have $s_0 = s_0(0)$, $s_1 = s_1(0)$, and $F = F(0)$

By reorganizing each of these two expressions and setting $s_0 = s_1 = s$, we can express the cost-benefit ratio associated with survival and fecundity benefits

$$\frac{-C}{B_s} = \left[(r_1 - r_2)\frac{d/2(1-c)}{1-d/2(1+c)}\right]/(1-r_1) \tag{4.9}$$

for survival benefits and

$$\begin{aligned}\frac{-C}{B_f} = \Bigg[Fr_1\Bigg((1-s_0)\frac{1-d}{1-d/2(1+c)} - (1-s_0)\left(\frac{1-d}{1-d/2(1+c)}\right)^2 \\ + (1-s_1)\frac{d/2(1-c)}{1-cd} - (1-s_1)\left(\frac{d/2(1-c)}{1-cd}\right)^2\Bigg) \\ - r_2F(1-s_1)\left(\frac{d/2(1-c)(1-d)}{(1-d/2(1-c))^2} + \frac{1-d}{1-cd}\frac{d/2(1-c)}{1-cd}\right) \\ - r_3F(1-s_1)\left(\frac{d/2(1-c)}{1-cd}\right)^2\Bigg]/s_0(1-r_1),\end{aligned} \tag{4.10}$$

for fecundity benefits. Note that these equations represent Hamilton's rule [38] for redirected help in our model. Coefficients r_i are computed assuming p close to 0 and then well approximate the values found in [91, 48].

When expressions (4.9) and (4.10) are set equal to 0, they put a maximum cost-benefit ratio that can support the emergence of redirected help. As the critical ratio increases, redirected helping is favored more readily, i.e. across a greater range of parameters. As the critical cost-benefit ratio decreases, redirected help is favored for less cost-benefit ratio and is thus favored less readily. To understand the impact of each parameter on the emergence of redirected helping, we vary that parameter and observe the response of the critical cost-benefit ratio.

When we decrease the cost of dispersal c, we increase the critical cost-benefit ratio, making it easier for help to emerge (Figure 4.2 top panels). Increasing the dispersal rate d increases the critical cost-benefit ratio (Figure

4.2 bottom panels). A high dispersal rate and low cost of dispersal then promote the emergence of help.

Increasing the cost of dispersal or decreasing the dispersal rate increases the coefficient of consanguinity (relatedness) between individuals. By increasing c or decreasing d, we naively expect an increase in the redirected help rate as individuals are more inclined to help a more related individual [37]. We observe the opposite, however. The kin competition between offspring can explain this relation. By decreasing dispersal d and increasing the cost of dispersal c, we increase the competition among relatives when they compete for a breeding spot. Indeed, when d goes to 0, all offspring of the same breeder will compete only for a single breeding spot. When we increase the dispersal rate and decrease the cost of dispersal, offspring can compete for more breeding sites, which reduces the kin competition [88].

We observe that the type of benefits redirected help confers also impacts the emergence of redirected help. The critical cost-benefit ratio associated with survival benefits increases with survival (Figures 4.2A, left column). Conversely, the critical cost-benefit ratio associated with fecundity benefits decreases with survival (Figures 4.2B, middle column). Fecundity benefits are a better promoter for redirected helping for low survival values, while survival benefits drive the emergence of redirected helping for high survival values better. Ultimately, survival benefits drive the emergence of help better than fecundity benefits for high survival values (Figures 4.2C, right column).

4.3.2 Larger brood failure probability p

As brood failure becomes more frequent, variation in the composition of the focal individual's neighborhood becomes more important to predictions made

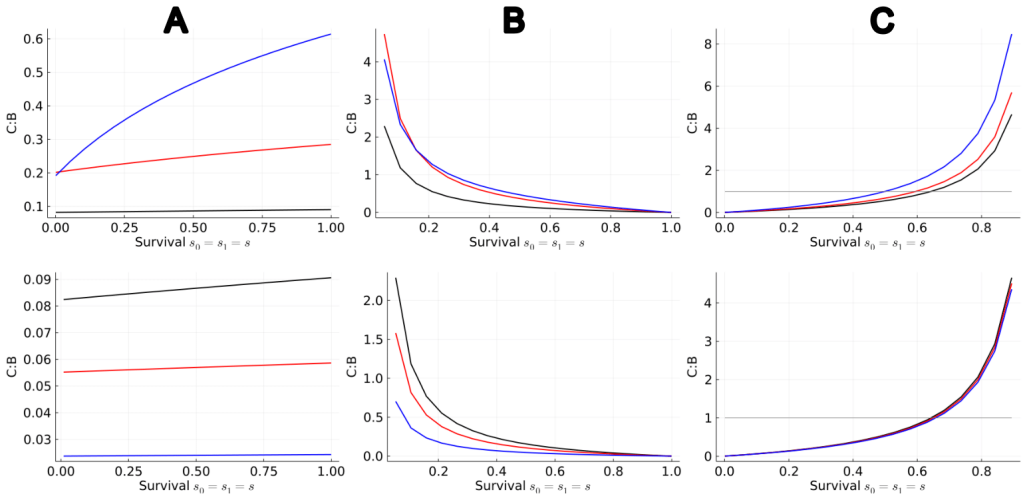

Figure 4.2: A (left column): Critical cost-benefit ratio associated with survival benefits depending on the survival $s_0 = s_1 = s$ on the x-axis. B (middle column): Critical cost-benefit ratio associated with fecundity benefits for various values depending on the survival $s_0 = s_1 = s$ on the x-axis. C (right column): ratio of the critical cost-benefit ratio associated with survival benefits over the critical cost-benefit ratio associated with fecundity benefits depending on the survival $s_0 = s_1 = s$ on the x-axis. The grey curve in C shows a survival/fecundity ratio equal to 1: no benefits are a better driver for the emergence of help than the other. Top row: Curves are plotted with various values of the cost of dispersal c: Black curves: $c = 0.2$, red curves: $c = 0.5$, blue curves: $c = 0.8$. Here $d = 0.2$. Bottom row: Curves are plotted with various values of the dispersal rate d: Black curves: $d = 0.2$, red curves: $d = 0.5$, blue curves: $d = 0.8$. Here, $c = 0.2$. Other parameters: $p = 10^{-5}$, $\mu = 10^{-6}$.

by our model. Thus, for more appreciable rates of brood failure (larger p), we must account for which of the focal individual's nearby neighbors experience brood failure and which do not. The variation we describe here is a form of demographic stochasticity [sensu 77]. We investigate the effects of this demographic stochasticity by observing the impact changing model parameters (now including p) has on the neighbor-modulated fitness change. We examine the emergence of redirected help by examining the neighbor-modulated fitness change associated with survival or fecundity benefits (4.4). We vary several parameters: the survival of breeders, $s_0 = s_1 = s$, the brood failure probability p, the dispersal rate d, and finally, the cost of dispersal c.

Looking at how the brood failure rate, p, impacts the fitness change, we observe several things. When p gets larger, the inclusive fitness change becomes negative for survival and fecundity benefits (Figure 4.3). For low values of p, redirected help can be promoted, but for higher values of p, redirected help may need greater benefits or lower costs to be promoted.

A decrease in the inclusive fitness change with increased p can be partially explained by the reduction of the coefficient of consanguinity with higher p. In fact, as p increases, coefficients r_i decrease. Indeed, when p is high, the individuals colonizing each breeding spot will become less related to the neighboring breeders. Helping a neighbor will become less valuable if individuals are more susceptible to brood failure (high p).

When dispersal d increases or the cost of dispersal c decreases, the maximal values of p that promote redirected help increase (Figure 4.3). For high values of d and low values of c, redirected help can emerge in an environment with higher brood failure probabilities. This pattern is similar to what we found with $p \approx 0$: high dispersal rate/low cost of dispersal decreases the fitness change and thus prevents the emergence of redirected helping.

Again, we can compare how survival and fecundity benefits can drive the emergence of redirected help. If brood failure probability is too high, redirected help does not evolve (Figure 4.4). When brood failure probability is sufficiently low, fecundity benefits will drive the emergence of help for low survival (Figures 4.4). At the same time, survival benefits will become a better driver than fecundity for higher survival (Figure 4.4). Changing the cost of dispersal does not change the range of survival values for which survival benefits promote the emergence of helping better than fecundity benefits (Figure 4.4 top panels). Increasing the dispersal rate, however, can make survival benefits a better promoter than fecundity benefits, even for low survival (Figure 4.4 bottom panels).

4.4 Discussion

We used a neighbor-modulated fitness approach to investigate how population viscosity can impact the evolution of redirected help. We assume our population inhabits a one-dimensional stepping-stone lattice, contrasting with our previous model in Chapter 3. We found that high relatedness via, for example, a low natal dispersal does not always promote redirected help. Even though the relatedness increases, competition among kin can diminish the net benefit of helping.

In fact, the benefits of help can be exactly compensated by kin competition [88, 89]. Several theories have been proposed to bypass this compensation. Adding generation overlap [48], dispersal of small groups rather than individuals (budding) [27], changing the timing of altruism [59], and conditional behaviors [16] have been proposed as different solutions to the evolution of altruism in a viscous population. Our model of redirected help assumes over-

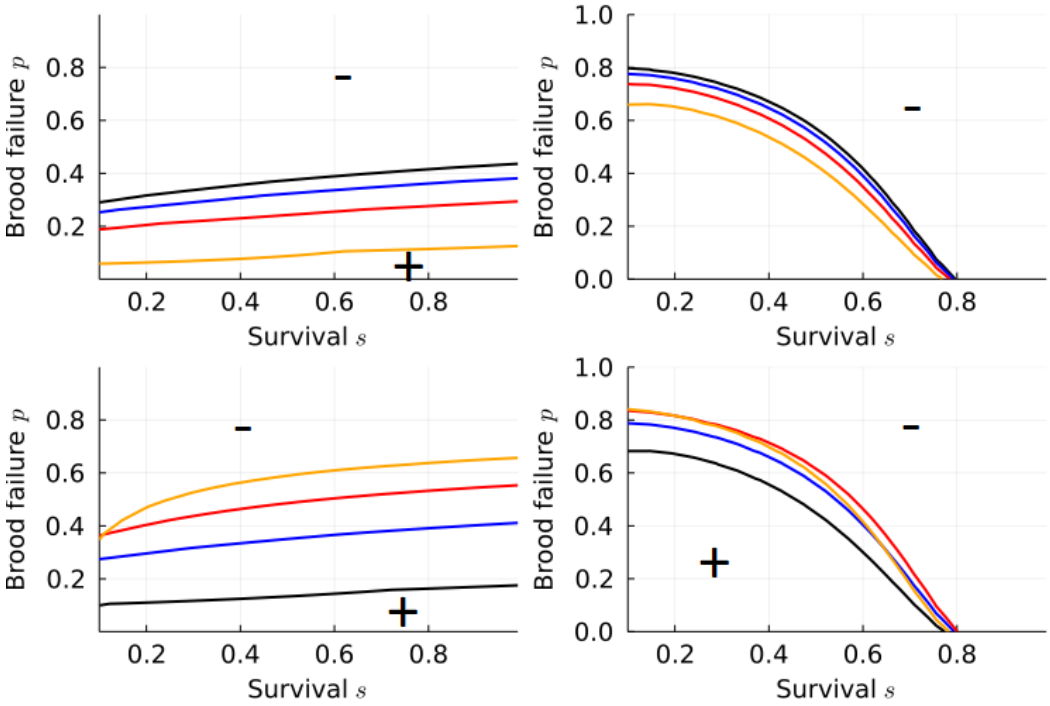

Figure 4.3: Neighbor-modulated fitness change nullclines ($\Delta w = 0$) associated with survival benefits (left panels) and fecundity benefits (right panels) when the brood failure probability p is larger than 0 for various values of the cost of dispersal c (top panels) and various values of the dispersal rate d (bottom panels). On the x-axis, survival $s_0 = s_1 = s$, and on the y-axis, brood failure probability p. Top panels: Black curves: $c = 0.1$, blue curves: $c = 0.3$, red curves: $c = 0.5$, orange curve: $c = 0.7$. Bottom panels: Black curves: $d = 0.3$, blue curves: $d = 0.5$, red curves: $d = 0.7$, orange curve: $d = 0.9$. A $+$ is intended to remind us that redirected help is favored under each curve, and a $-$ shows that redirected help is not favored in the region over each curve. Other parameters, unless changed: $c = 0.2$, $d = 0.2$, $\mu = 10^{-6}$, $C = 1$, $B_s = B_f = 10$.

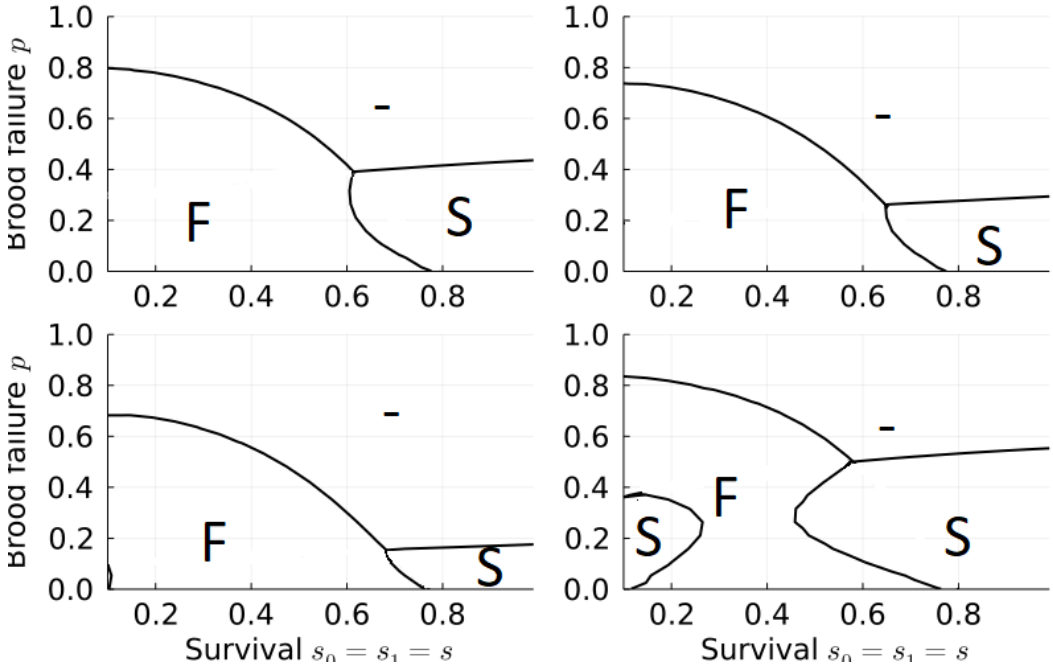

Figure 4.4: Difference of neighbor-modulated fitness change associated with survival and fecundity benefits for brood failure probability p (y-axis), survival s (x-axis), various values for the cost of dispersal (top panels), or dispersal rate d (bottom panels). Top panels: cost of dispersal c: left panel: $c = 0.1$, right panel: $c = 0.5$. Bottom panel: dispersal rate d. Left panel: $d = 0.3$, right panel: $d = 0.7$. A "-" sign denotes regions of the parameter space where redirected help is not emerging. F: regions of the parameter space where fecundity benefits drive the emergence of redirected help better than survival benefits. S: regions of the parameter space where survival benefits drive the emergence of redirected help better than fecundity benefits. Other parameters (unless changed): $c = 0.2$,$d = 0.2$, $\mu = 10^{-6}$, $C = 1$, $B_s = B_f = 10$.

lapping generations. Life history is also constrained in our model, as helpers do not have any fecundity because of brood failure. This brood failure implies that helpers pay a cost of survival to help their relatives. Consequently, overlapping generations play a complex role in our model. Increasing generation overlap increases survival benefits and the cost of helping but decreases fecundity benefits. Brood failure makes helping with fecundity benefits worthwhile, even if the generation overlap is low. This result contrasts with previous studies, where low survival hinders help emergence [48, 91]. In our model, altruism can then evolve thanks to the overlapping generations and the constraints of our life cycle.

In a previous study, costly dispersal hindered the emergence of help, regardless of the benefits provided, [48]. Even if they looked at the evolution of helping and we looked at the evolution of redirected helping, we observed similar results in our model of redirected helping. This is due to the similar spatial structure in the two models, where, once born, offspring disperse to the same breeding spots. However, the evolutionary consequences of the offspring dispersal can change if we use different details of the spatial structure incorporated into a model. In our previous model of redirected help, in which we used an infinite-island model, we found that when dispersal is rare (or costly), redirected help is promoted (see Chapter 3), which is the opposite result to what we found using a stepping stone model. In both models, more dispersing individuals diminishes the kin competition and lowers the relatedness between individuals. In the stepping stone model, the reduction of the kin competition compensates for the lower relatedness, which makes helping emerge more easily when offspring are more likely to disperse. However, this compensation is not strong enough in the infinite-island model, causing helping to emerge more easily when offspring disperse less.

Dispersal in nature can occur over relatively short or long distances. It is often thought to incur a cost for the dispersers due to the predation or lack of resources during the dispersal [2]. If we want to study the impact of dispersal on different behaviors, we need to consider how much individuals disperse and how they disperse. Some individuals will disperse close to their natal site; some will die during the process. It then may be compelling to separate local and long-distance/costly dispersal as they have different consequences on the life history of a species. For example, long-tailed tits disperse to a few hundred meters from their natal site but also create flocks before looking for a breeding site [43]. Thus, tong-tailed tits population shows population viscosity as individuals disperse to relatively short distances. Given this local dispersal, a (two-dimensional) stepping-stone spatial structure would be more relevant than an infinite-island model. However, a spatial structure that considers the formation of flocks followed by the local dispersal of individuals could lead to a more well-mixed population and show different impacts of the dispersal rate.

We modeled the evolution of redirected help in a viscous population on a one-dimensional stepping stone following our model using an infinite-island model. We highlight the impact of the spatial structure in the evolution of altruistic behaviors. Spatial structures affect the population's life histories by impacting the population's dispersal, mating, and helping. Our models show the need to consider the impact of different spatial structures when studying social behaviors such as altruism, competition, or reproduction.

Chapter 5

Conclusion

5.1 Summary

I studied the evolution of different altruistic behaviors using three models. I showed that group living features, such as the benefits of altruism, habitat saturation, or spatial structure, can challenge previous theories and results for the evolution of altruistic behaviors. My work emphasizes the need to model environmental feedback when studying the evolution of social behaviors. In addition, I highlighted the impact of spatial structure on ecological relations between individuals and their environment.

In Chapter 2 of this thesis, I modeled the evolution of delayed natal dispersal in relation to habitat saturation and group size effects. I studied the evolution of delayed dispersal when the quality of the local environment is improved by greater numbers of individuals (e.g., safety in numbers). I assumed that individuals who delay natal dispersal also expect to delay personal reproduction. In addition, I assumed that improved environmental quality benefits manifest as changes to fecundity and survival. I focused my interest on how the

changes in these life-history features affect delayed dispersal. I also aimed to understand the relationship between levels of delayed dispersal and the probability of establishing as an independent breeder (a population-level feature) in response to changes in life-history details. I considered a sexual organism, which allows me to study parent-offspring conflict over delayed dispersal. At evolutionary equilibrium, fecundity and survival benefits of group size or quality promote higher levels of delayed dispersal over a larger set of life histories with one exception. The exception is for benefits of increased group size or quality reaped by the individuals who delay dispersal. The increased benefit does not change the life histories supporting delay dispersal there. In contrast to previous predictions, I found that a low probability of establishing in a new location is not always associated with a higher incidence of delayed dispersal. Previous literature expected a positive link between habitat saturation and delayed dispersal [71]. My dynamical model of the evolution of delayed dispersal with ecological feedback demonstrates that this relation is not as simple. Finally, I found that increased personal benefits of delayed dispersal exacerbate the conflict between parents and their offspring. This result contrasts with my own predictions. We should expect to observe more altruistic behavior if we have more benefits. In fact, this benefit is available when group formation has already occurred, which requires offspring to delay their dispersal. This benefit is thus circumstantial and can drive a higher dispersal rate.

Overall, my model of delayed dispersal confirms but also shows new predictions about the evolution of delayed dispersal in a population subject to habitat saturation.

In Chapter 3 of my thesis, I studied the selective advantage of redirected help in a viscous population living on an infinite island model. When a brood fails, the parent can decide to help a neighbor rear its offspring. This behavior

is referred to as redirected helping and occurs in various species. In a viscous population, the obvious advantages of redirected helping can be diluted by the competition among kin. I addressed the evolution of redirected helping in a viscous population using an inclusive fitness model that allows overlapping generations and accounts for demographic stochasticity due to brood failure. In contrast to previous theoretical studies of species with overlapping generations, I found that helping (in this case, redirected helping) can be more strongly promoted when it provides survival rather than fecundity benefits. This result depends on the species' life history and environmental parameters, such as survival or brood failure probability. I obtained this result due to the specific life history of the population, as helpers cannot reproduce. Such life history features can thus change predictions and lead to unexpected results. Altruistic behaviors can occur in very various life histories. My results show that the diversity of these life histories makes it challenging to draw general rules about the evolution of altruistic behaviors.

In the model of Chapter 3, I investigated how population viscosity can affect the evolution of redirected help by focusing on a population living on an infinite-island model. However, the dispersal pattern of individuals in an infinite-island model reduces the impact of population viscosity. In Chapter 4 of my thesis, I strengthened population viscosity by studying the evolution of redirected help using a one-dimensional stepping-stone model. In this new spatial structure, the dispersal pattern is much more constrained than the infinite-island spatial structure, strenghtening the population viscosity I was interested in. Using a different model, I wanted to study how changing the spatial structure can affect the evolution of altruistic behavior. In this new spatial structure, dispersal acts in the opposite direction as in our previous model. This difference is inherent to the spatial structures used in these mod-

els. I also found similarities between the results. For instance, in both models, the different benefits from redirected help can influence how this behavior will evolve. Different spatial structures can then affect how altruistic behaviors and social behaviors, in general, can evolve.

5.2 Future directions

Throughout this thesis, I described potential future directions that would be interesting to study.

Following Chapter 1, given individuals delay their natal dispersal, a natural behavior that can arise following this group formation is the evolution of help. Helping behavior is similar to delayed dispersal as it incurs a cost to the helper and provides survival or fecundity benefits to the helped individual. However, the coevolution of delayed dispersal and help could lead to different group sizes and qualities, creating discrepancies between groups. The coevolution of these two behaviors has been studied before [25] but in a way that does not consider a sexual population, which prevents social interactions such as sexual reproduction, which can shape evolution differently. Studying this coevolution in a dynamic population context could provide results and answers on the history of altruistic behaviors [60, 10, 79]. Given the coevolution of these two behaviors, we could also look at the interactions between help and habitat saturation. From our unexpected results of Chapter 2, we could also expect some additional results regarding the evolution of help and ecological feedback. These results would provide the necessary or sufficient environmental conditions for the evolution of helping in groups formed after delayed dispersal [60, 10, 79].

Following Chapters 3 and 4, a potential new project would be to study the

evolution of redirected helping and load-lightening. Load-lightening happens when an individual decreases the parental resources it provides its offspring [46, 50]. This behavior appears naturally because a breeder may want to favor their fitness. From Bourke's definition [3], load-lightening is thus a selfish behavior. This behavior also occurs in the Long-tailed Tits [43], which is a species which performs redirected helping. It appears then that potentially, some individuals can behave selfishly toward their own offspring but altruistically toward the offspring of others, which seems paradoxical. Exploring the evolutionary history of these two behaviors could provide us with solutions to this apparent paradox. We could also extend this model to help in general. Given ecological feedback such as competition among kin or related, we may also observe the evolution and coevolution of each behavior in a different environmental/ecological context. Using examples from the field, such as the Long-tailed tits, we could compare the results with data and test the predictions.

In addition to the coevolution of redirected helping and load-lightening, studying the conflict over parental resource allocation would be very interesting. Offspring always want more resources than their parents or a helper would give them [96]. This type of conflict is similar to the one we observe in Chapter 2. Measuring the extent of this conflict in the context of coevolution of redirected help and load-lightening would provide resourceful information on the difference between conflict and coevolution of behaviors. A resolution model of these conflicts could lead to a different evolution of these two behaviors compared to the coevolution model. We could thus investigate how these behaviors and the conflicts evolve with respect to each other. Should we expect to have a reduction of conflict as social behavior evolves? Or should we expect social behaviors to increase the conflict? This direction could then

provide philosophical insight on the nature of altruism. Is altruism a reduction of conflict, or does altruism actually increase this conflict? Ironically, we may also observe that conflict over parental resources could lead to the evolution of altruistic behaviors.

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Appendix A

Appendix Chapter 2

A.1 Matrix B

Life-history details provided in the main text, see subsection 2.2.2, tell us that the distribution of individuals at time step $t+1$, $V_{t+1} = (U_{t+1}, B_{0,t+1}, B_{1,t+1}, A_{0,t+1}, A_{1,t+1})^T$ is equal to $B(B_0, B_1)V_t$, where B is the matrix

$$\begin{pmatrix} s_u(1-\tilde{p_u}) & 0 & 0 & (1-h_0)s_u(1-\tilde{p_u}) & (1-h_1)s_u(1-\tilde{p_u}) \\ & & & +h_0S(A_0)(1-\tilde{p_a}) & +h_1(S(A_1)(1-\tilde{p_a}) \\ s_u\tilde{p_u} & (1-T_0(h_0))s_{b_0} & T_1(h_1)s_{b_0} & (1-h_0)s_u\tilde{p_u} + h_0S(A_0)\tilde{p_a} & (1-h_1)s_u\tilde{p_u} + h_1S(A_1)\tilde{p_a} \\ 0 & T_0(h_0)s_{b_1} & (1-T_1(h_1))s_{b_1} & 0 & 0 \\ s_u\tilde{p_u}n_0 & (1-T_0(h_0))s_{b_0}n_0 & T_1(h_1)s_{b_0}n_0 & (1-h_0)s_u\tilde{p_u}n_0 + h_0S(A_0)\tilde{p_a}n_0 & (1-h_1)s_u\tilde{p_u}n_0 + h_1S(A_1)\tilde{p_a}n_0 \\ 0 & T_0(h_0)s_{b_1}n_1 & (1-T_1(h_1))s_{b_1}n_1 & 0 & 0 \end{pmatrix}. \quad \text{(A.1.1)}$$

In the previous line, $S(A_0) = T_0(h_0)s_{a_1} + (1-T_0(h_0))s_{a_0}$ and $S(A_1) = T_1(h_1)s_{a_0} + (1-T_1(h_1))s_{a_1}$. Note that $B(B_{0,t}, B_{1,t})$ depends on $\tilde{p_u}$ and $\tilde{p_a}$, and expressions for those are given in the main text.

A.2 Stability of the trivial equilibrium

The population has a trivial equilibrium $V_t = (0, 0, 0, 0, 0)^T$. To determine the stability of this equilibrium, we compute the dominant eigenvalue of the next-generation matrix [4] evaluated at the trivial equilibrium. The next generation matrix is the product of two matrices. The first is F, a 5×5 matrix whose top three rows consist only of 0 and whose bottom two rows are those from the matrix $B(B_{0,t}, B_{1,t})$, (A.1.1). The second is $(I - S)^{-1}$,which involves the 5×5 identity matrix I and the 5×5 matrix S. Here, S contains those elements of $B(B_{0,t}, B_{1,t})$, (A.1.1) not already contained in F. We write the next generation matrix as $G = F(I - S)^{-1}$. The dominant eigenvalue of G is $R_0 = L_u L_b \Lambda$ in equation (2.1), where

$$\Lambda = (1 - s_{b_1}(1 - T_1))((p_u - p_a)s_u + p_a)h_0 n_0 S(A_0) + ((p_u - p_a)s_u + p_a)s_{b_1} n_1 h_1 T_0 S(A_1)$$
$$+p_u(((1 - h_0)(T_1 - 1)n_0 + T_0 n_1(1 - h_1))s_{b_1} + n_0(1 - h_0))s_u$$

and L_u and L_b are given in the main text. When R_0 is greater than 1, the population reaches a positive and stable equilibrium $V^* = (U^*, B_0^*, B_1^*, A_0^*, A_1^*)$ whose value and stability are computed numerically. This new equilibrium is also the dominant right eigenvector of the matrix $B(B_0^*, B_1^*)$, (A.1.1), for the dominant eigenvalue 1.

A.3 Evolution of h_0 and h_1

In this appendix, we derive expressions for the action of selection on h_0 and h_1. We use the approach outlined in [90], which requires us to begin our derivation by identifying recipients of social behavior, i.e., those individuals

whose fitness is affected by the delayed dispersal of others. In our model, there are five classes of recipient: floaters (class 1), breeders in a low-quality spot (class 2), breeders in a high-quality spot (class 3), subordinates in a low-quality spot (class 4), subordinates in a high-quality spot (class 5). Fix attention on a class-2 recipient and let $\bar{\eta}_0^{(2)}$ denote the probability that the average offspring produced by this individual delays dispersal as a subordinate. Fix attention on a class-3 recipient and let $\bar{\eta}_1^{(3)}$ denote the probability that the average offspring produced by this individual delays dispersal as a subordinate. Fix attention on a class-4 recipient, let $\eta_0^{(4)}$ denote the probability that this individual delays dispersal, and let $\bar{\eta}_0^{(4)}$ denote the probability that the average offspring produced by this individual's parent delays dispersal as a subordinate (including the actor itself). Finally, fix attention on a class-5 recipient, let $\eta_1^{(5)}$ denote the probability that this individual delays dispersal, and let $\bar{\eta}_1^{(5)}$ denote the probability that the average offspring produced by this individual's parent delayed dispersal as a subordinate (including the actor itself).

Let m_{ij} denote the expected number of class-i individuals produced by a focal class-j individual in the previous time step, weighted by genetic contribution. We store these quantities in the matrix $M = [m_{ij}]$, where M is given in Table A.3.1. If $\bar{\eta}_0^{(2)} = \eta_0^{(4)} = \bar{\eta}_0^{(4)} = h_0$ and $\bar{\eta}_1^{(3)} = \eta_1^{(5)} = \bar{\eta}_1^{(5)} = h_1$, then $MV^* = V^*$, where V^* is given in the previous Appendix. This implies that, in the absence of selection, the largest eigenvalue of M is unity and is associated with the right eigenvector V^*. In the absence of selection, the largest eigenvalue of M is also associated with a left eigenvector $\nu = (\nu_u, \nu_{b_0}, \nu_{b_1}, \nu_{A_0}, \nu_{A_1})$. This left eigenvector stores the individual reproductive value of each class of individual in the order introduced above. We determine ν by numerically solving the equation $\nu = \nu M$ with M evaluated at $\bar{\eta}_0^{(2)} = \eta_0^{(4)} = \bar{\eta}_0^{(4)} = h_0$ and $\bar{\eta}_1^{(3)} = \eta_1^{(5)} = \bar{\eta}_1^{(5)} = h_1$.

Matrix M and matrix B from the previous section of the appendix are related but are not identical. Matrix B tracks the number of individuals, while matrix M tracks the flow of genes. This means we have to carefully account for male and female functions when deriving M. It also means the same level of detail is not needed for matrix B.

To determine the effect of selection on h_0, we introduce $g_0^{(j)}$ as the genotypic value of a class-j recipient ($j = 2, 4$) at the locus controlling delayed dispersal behavior of individuals born in a low-quality group. We treat $\eta_0^{(j)}$ and $\bar{\eta}_0^{(j)}$ as functions of corresponding $g_0^{(j)}$, then compute the change in fitness by computing the derivative of the matrix M with respect to the genotypic value of each recipient $g_0^{(j)}$, and multiply it by the left and right eigenvectors to obtain

$$\nu \frac{dM}{dg_0^{(2)}} V^* + \nu \frac{dM}{dg_0^{(4)}} V^* = \nu \left(\frac{\partial M}{\partial \bar{\eta}_0^{(2)}} \frac{d\bar{\eta}_0^{(2)}}{dg_0^{(2)}} \right) V^* + \nu \left(\frac{\partial M}{\partial \eta_0^{(4)}} \frac{d\eta_0^{(4)}}{dg_0^{(4)}} + \frac{\partial M}{\partial \bar{\eta}_0^{(4)}} \frac{d\bar{\eta}_0^{(4)}}{dg_0^{(4)}} \right) V^* \tag{A.3.1}$$

where it is understood that expressions are evaluated at $\bar{\eta}_0^{(2)} = \eta_0^{(4)} = \bar{\eta}_0^{(4)} = h_0$ and $\eta_1^{(3)} = \eta_1^{(5)} = \bar{\eta}_1^{(5)} = h_1$. Finally, we replace the ordinary derivatives in the previous line with corresponding relatedness coefficients to obtain

$$\nu \left(\frac{\partial M}{\partial \bar{\eta}_0^{(2)}} R_{I,B_0} \right) V^* + \nu \left(\frac{\partial M}{\partial \eta_0^{(4)}} R_{I,A_0} \right) V^* + \nu \left(\frac{\partial M}{\partial \bar{\eta}_0^{(4)}} \bar{R}_{I,A_0} \right) V^*, \tag{A.3.2}$$

which can be rewritten as

$$\nu \left(\frac{\partial M}{\partial \bar{\eta}_0^{(2)}} \right) V^* R_{I,B_0} + \nu \left(\frac{\partial M}{\partial \eta_0^{(4)}} \right) V^* R_{I,A_0} + \nu \left(\frac{\partial M}{\partial \bar{\eta}_0^{(4)}} \right) V^* \bar{R}_{I,A_0}, \tag{A.3.3}$$

where R_{I,B_0} is the relatedness between the individual controlling the delayed dispersal behavior on a low-quality breeding spot (breeder or subordinate)

and the breeder on the same low-quality breeding spot (the breeder itself or the breeder of the subordinate). $R_{I,B_0} = 1$ if the breeder controls the behavior, and $R_{I,B_0} = R_{A_0,B_0}$ if the subordinate controls it. R_{I,A_0} is the relatedness between the individual controlling the delayed dispersal behavior on a low-quality breeding spot and the subordinate who delays dispersal. If the subordinate itself controls the behavior, $R_{I,A_0} = 1$; when the breeder controls it, $R_{I,A_0} = R_{B_0,A_0}$. Finally, $\bar{R}_{I,A_0}$ is the relatedness between the individual controlling the delayed dispersal behavior on a low-quality breeding spot and the average delayed disperser on the same breeding spot. $\bar{R}_{I,A_0}$ is equal to R_{B_0,A_0} when the breeder controls the behavior and to $\bar{R}_{A_0,A_0}$ if the controller is the subordinate. Those relatedness coefficients are the slopes of the actor's phenotype $\eta_0^{(j)}$ on the recipient's genic value $g_0^{(j)}$ and can be replaced by statistical regression coefficients computed in the following Appendix.

This expression gives the change in fitness detected by the different recipients when the genotypic value of the delayed dispersal behavior of individuals born in a low-quality breeding spot is changed. In other words, equation (A.3.3) gives the inclusive fitness change for the delayed dispersal behavior h_0 which can be rewritten as:

$$\Delta_{h_0} W_{\text{Breeder}} \cdot R_{I,B_0} + \Delta_{h_0} W_{\text{Subs}} \cdot \bar{R}_{I,A_0} + \Delta_{h_0} W_{\text{Actor}} \cdot R_{I,A_0}. \qquad \text{(A.3.4)}$$

which is equation (2.7a) in the main text

To determine the effect of selection on h_1, we carry out a process similar to the one we use for h_0 evolution. We introduce $g_1^{(j)}$ as the genotypic value of a class-j recipient ($j = 3, 5$) at the locus controlling the delayed dispersal behavior of individuals born in a high-quality group. We treat $\eta_1^{(j)}$ and $\bar{\eta}_1^{(j)}$ as functions of corresponding $g_1^{(j)}$, then compute the change in fitness by com-

puting the derivative of the matrix M with respect to the genotypic value of each recipient $g_1^{(j)}$, and multiply it by the left and right eigenvectors

$$\nu\frac{dM}{dg_1^{(3)}}V^* + \nu\frac{dM}{dg_1^{(5)}}V^* = \nu\left(\frac{\partial M}{\partial\bar{\eta}_1^{(3)}}\frac{d\bar{\eta}_1^{(3)}}{dg_1^{(3)}}\right)V^* + \nu\left(\frac{\partial M}{\partial\eta_1^{(5)}}\frac{d\eta_1^{(5)}}{dg_1^{(5)}} + \frac{\partial M}{\partial\bar{\eta}_1^{(5)}}\frac{d\bar{\eta}_1^{(5)}}{dg_1^{(5)}}\right)V^* \tag{A.3.5}$$

where expressions are evaluated at $\bar{\eta}_1^{(3)} = \eta_1^{(5)} = \bar{\eta}_1^{(5)} = h_1$ and $\eta_0^{(2)} = \eta_0^{(4)} = \bar{\eta}_0^{(4)} = h_0$. Finally, we replace the ordinary derivatives in the previous line with corresponding relatedness coefficients to obtain

$$\nu\left(\frac{\partial M}{\partial\bar{\eta}_1^{(3)}}R_{I,B_1}\right)V^* + \nu\left(\frac{\partial M}{\partial\eta_1^{(5)}}R_{I,A_1}\right)V^* + \nu\left(\frac{\partial M}{\partial\bar{\eta}_1^{(5)}}\bar{R}_{I,A_1}\right)V^*, \tag{A.3.6}$$

Which can be rewritten as

$$\nu\left(\frac{\partial M}{\partial\bar{\eta}_1^{(3)}}\right)V^*R_{I,B_1} + \nu\left(\frac{\partial M}{\partial\eta_1^{(5)}}\right)V^*R_{I,A_1} + \nu\left(\frac{\partial M}{\partial\bar{\eta}_1^{(5)}}\right)V^*\bar{R}_{I,A_1}, \tag{A.3.7}$$

where R_{I,B_1} is the relatedness between the individual controlling the delayed dispersal behavior on a high-quality breeding spot (breeder or subordinate) and the breeder on the same high-quality breeding spot (the breeder itself or the breeder of the subordinate). $R_{I,B_1} = 1$ if the breeder controls the behavior, and $R_{I,B_1} = R_{A_1,B_1}$ if the subordinate controls it. R_{I,A_1} is the relatedness between the individual controlling the delayed dispersal behavior on a high-quality breeding spot and the subordinate who delays dispersal. If the subordinate itself controls the behavior, $R_{I,A_1} = 1$; when the breeder controls it, $R_{I,A_1} = R_{B_1,A_1}$. Finally, $\bar{R}_{I,A_1}$ is the relatedness between the individual controlling the delayed dispersal behavior on a high-quality breeding spot and the average subordinate on the same breeding spot. $\bar{R}_{I,A_1}$ is equal to R_{B_1,A_1} when the breeder controls the behavior and to $\bar{R}_{A_1,A_1}$ if the controller is the

subordinate. Those relatedness coefficients are the slopes of the actor's phenotype $\eta_1^{(j)}$ on the recipient's genic value $g_1^{(j)}$ and can be replaced by statistical regression coefficients computed in the following Appendix. As well as (A.3.3), (A.3.7) gives the change in fitness perceived by the different recipients when the genotypic value of the delayed dispersal behavior of individuals born in high-quality breeding spots is changed. In other words, equation (A.3.7) is the inclusive fitness change for h_1.

Equations (A.3.3) and (A.3.7) compute the effect of selection on the genotypic value of a subordinate living on a low-quality and high-quality, respectively. They thus correspond to the inclusive fitness change of increasing the delayed dispersal rates (2.7a) and (2.7b) we found in the main text.

Table A.3.1: Matrix M whose i,jth entry stores the expected number of class-i individuals produced by a class-j individual, weighted by genetic contribution. To keep notation compact we introduce $\theta_0 = (1-\phi)\frac{B_0^*}{B_0^*+B_1^*}$, $\theta_1 = (1-\phi)\frac{B_1^*}{B_0^*+B_1^*}$, $\psi_0 = 1+\phi+\theta_0$, and $\psi_1 = 1+\phi+\theta_1$. The values θ_0 and θ_1 represent reproductive success through male function, while ψ_0 and ψ_1 represent reproductive success through female function. The terms $S^{(4)}(A_0) = (1-T_0(\bar{\eta}_0^{(4)})s_{a_0} + T_0(\bar{\eta}_0^{(4)})s_{a_1}$ and $S^{(5)}(A_1) = (1-T_1(\bar{\eta}_1^{(5)})s_{a_1} + T_1(\bar{\eta}_1^{(5)})s_{a_0}$ denote the survival rates of a subordinate born on a low-quality and high-quality breeding spot, respectively.

$$M = \begin{pmatrix}
s_u(1-\tilde{p_u}) & 0 & 0 & (1-\eta_0^{(4)})s_u(1-\tilde{p_u}) & (1-\eta_1^{(5)})s_u(1-\tilde{p_u}) \\
 & & & +\eta_0^{(4)}S^{(4)}(A_0)(1-\tilde{p_a}) & +\eta_1^{(5)}S^{(5)}(A_1)(1-\tilde{p_a}) \\
s_u\tilde{p_u} & (1-T_0(\bar{\eta}_0^{(2)}))s_{b_0} & T_1(\bar{\eta}_1^{(3)})s_{b_0} & (1-\eta_0^{(4)})s_u\tilde{p_u} & (1-\eta_1^{(5)})s_u\tilde{p_u} \\
 & & & +\eta_0^{(4)}S^{(4)}(A_0)\tilde{p_a} & +\eta_1^{(5)}S(A_1)\tilde{p_a} \\
0 & T_0(\bar{\eta}_0^{(2)})s_{b_1} & (1-T_1(\bar{\eta}_1^{(3)}))s_{b_1} & 0 & 0 \\
s_u\tilde{p_u}\frac{n_0}{2}\psi_0 & \frac{n_0}{2}((1-T_0(\bar{\eta}_0^{(2)}))s_{b_0}\psi_0 & \frac{n_0}{2}(T_1(\bar{\eta}_1^{(3)})s_{b_0}\psi_0 & (1-\eta_0^{(4)})s_u\tilde{p_u}\frac{n_0}{2}\psi_0 & (1-\eta_1^{(5)})s_u\tilde{p_u}\frac{n_0}{2}\psi_0 \\
 & +T_0(\bar{\eta}_0^{(2)})s_{b_1}\theta_0) & +(1-T_1(\bar{\eta}_1^{(3)}))s_{b_1}\theta_0) & +\eta_0^{(4)}S^{(4)}(A_0)\tilde{p_a}\frac{n_0}{2}\psi_0 & +\eta_1^{(5)}S^{(5)}(A_1)\tilde{p_a}\frac{n_0}{2}\psi_0 \\
s_u\tilde{p_u}\frac{n_1}{2}\theta_1 & \frac{n_1}{2}(T_0(\bar{\eta}_0^{(2)})s_{b_1}\psi_1 & \frac{n_1}{2}((1-T_1(\bar{\eta}_1^{(3)}))s_{b_1}\psi_1 & (1-\eta_0^{(4)})s_u\tilde{p_u}\frac{n_1}{2}\theta_1 + & (1-\eta_1^{(5)})s_u\tilde{p_u}\frac{n_1}{2} \\
 & +(1-T_0(\bar{\eta}_0^{(2)}))s_{b_0}\theta_1) & +T_1(\bar{\eta}_1^{(3)})s_{b_0}\theta_1) & \eta_0^{(4)}S^{(4)}(A_0)\tilde{p_a}\frac{n_0}{2}\theta_1 & +\eta_1^{(5)}S^{(5)}(A_1)\tilde{p_a}\frac{n_1}{2}\theta_1
\end{pmatrix}$$

A.4 Relatedness

We computed the relatedness between the actor and its relatives using the method developed in Michod and Hamilton [65]. In general, the relatedness between individuals I and J is defined as

$$R_{I,J} = \frac{Q_{I,J}}{Q_{I,I}},$$

where $Q_{I,J}$ is defined as the probability of identity by descent between an allele chosen uniformly at random from individual I and another chosen independently at random from individual J. For $Q_{I,I}$, the choice is made with replacement. For a breeder and its subordinate, the probability of identity by descent is $(1+\phi)(1+f)/4$, where f is the probability of identity by descent between the two homologous alleles carried by the same individual. The probability that two alleles chosen from the same individual are identical by descent is $(1+f)/2$. Thus, the relatedness between a subordinate and its breeder is

$$R_{B_0,A_0} = R_{B_1,A_1} = R_{A_0,B_0} = R_{A_1,B_1} = \frac{1+\phi}{2}.$$

Using the same method, we compute the relatedness between a subordinate and the average subordinate on the breeding spot. As mentioned in the main text, the average subordinate includes the actor itself as it affects its own fitness. The probability that two alleles chosen from the same individual are identical by descent remains $(1+f)/2$. The probability of identity by descent between two subordinates born on the same patch is,

$$\left(\frac{1}{n_0} + \frac{n_0-1}{n_0}\left(\phi^2 + \phi(1-\phi) + (1-\phi)^2\frac{N+1}{4N}\right)\right)(1+f)/2,$$

where N denotes the number of mates an individual has. Note that, on average, two individuals taken randomly in the population have a probability of identity by descent equal to 0 relative to the average population. It means that if two outcrossed subordinates do not share the same male parent, they share only 1/4 of their genes through their female parent. The relatedness between two subordinates born in the same low-quality breeding spot is thus

$$\bar{R}_{A_0,A_0} = \frac{1}{n_0} + \frac{n_0 - 1}{n_0}\left(\phi^2 + \phi(1-\phi) + (1-\phi)^2\frac{N+1}{4N}\right),$$

Finally, the same process leads to the relatedness between two subordinates born in the same high-quality breeding spot,

$$\bar{R}_{A_1,A_1} = \frac{1}{n_1} + \frac{n_1 - 1}{n_1}\left(\phi^2 + \phi(1-\phi) + (1-\phi)^2\frac{N+1}{4N}\right).$$

A.5 Numerical simulations

As described in the main text, we use numerical approximations to estimate $\Delta_{h_0}W$ and $\Delta_{h_1}W$. To estimate the different parts of the equations, (A.3.3), and (A.3.7), we use finite difference approximations on the mutant matrix M. We start by estimating $\frac{\partial M}{\partial \bar{\eta}_0^{(2)}}$ using the matrix M evaluated when the delayed dispersal of a subordinate, $\bar{\eta}_0^{(2)}$, is increased and subtract this matrix with the matrix M evaluated when $\bar{\eta}_0^{(2)}$ is decreased:

$$\frac{\partial M}{\partial \bar{\eta}_0^{(2)}} \approx M|_{\bar{\eta}_0^{(2)}=h_0+\delta h_0,\eta_0^{(4)}=\bar{\eta}_0^{(4)}=h_0,\bar{\eta}_1^{(3)}=\eta_1^{(5)}=\bar{\eta}_1^{(5)}=h_1} - M|_{\bar{\eta}_0^{(2)}=h_0-\delta h_0,\eta_0^{(4)}=\bar{\eta}_0^{(4)}=h_0-\delta h_0,\bar{\eta}_1^{(3)}=\eta_1^{(5)}=\bar{\eta}_1^{(5)}=h_1}.$$

We approximate $\frac{\partial M}{\partial \eta_0^{(4)}}$, and $\frac{\partial M}{\partial \bar{\eta}_0^{(4)}}$, (A.3.3), using the same process but changing $\eta_0^{(4)}$ and $\bar{\eta}_0^{(4)}$. We multiply $\frac{\partial M}{\partial \bar{\eta}_0^{(2)}}$, $\frac{\partial M}{\partial \eta_0^{(4)}}$, and $\frac{\partial M}{\partial \bar{\eta}_0^{(4)}}$ by the dominant left and right

eigenvectors of M evaluated at $\eta_0^{(2)} = \eta_0^{(4)} = \bar{\eta}_0^{(4)} = h_0$ and $\bar{\eta}_1^{(3)} = \eta_1^{(5)} = \bar{\eta}_1^{(5)} = h_1$, ν, and μ and by the corresponding relatedness coefficients (see equation (A.3.3) and Appendix A.4 for the relatedness coefficients). Summing all these quantities, we find the approximation of the inclusive fitness change $\Delta_{h_0} W$. The same method is used to compute the approximation of the inclusive fitness change $\Delta_{h_1} W$.

We then update the values of h_0 and h_1 based on the computed values of $\Delta_{h_0} W$ and $\Delta_{h_1} W$. A positive computed value results in an increase of the delayed dispersal strategy, whereas a negative computed value results in a decrease. We repeat the process using the updated values of h_0 and h_1 until the algorithm stops by one of the three events described in the main text.

A.6 Supplementary data

The parent-offspring conflict over delay dispersal varies with selfing rate ϕ and fecundity benefits $n_1 - n_0$. As ϕ gets to 1, the population is clonal so relatedness is equal to 1, the difference between subordinate and breeder preferred level of delay dispersal is then null (Figures A.6.1, A.6.2).

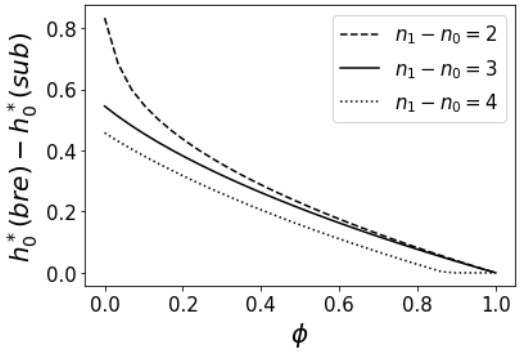

Figure A.6.1: Extent of breeder-subordinate conflict over delayed dispersal represented by the difference of predictions under breeder exclusive control or under subordinate exclusive control ($h_0^*(bre) - h_0^*(sub)$). The conflict varies with selfing rate ϕ and fecundity benefits $n_1 - n_0$. Unless otherwise noted, $s_{b_0} = s_{b_1} = 0.5$, $s_u = s_{a_0} = 0.5$ $s_{a_1} = 0.6$, $a = 2$, $p_a = 0.25$, $p_u = 0.5$

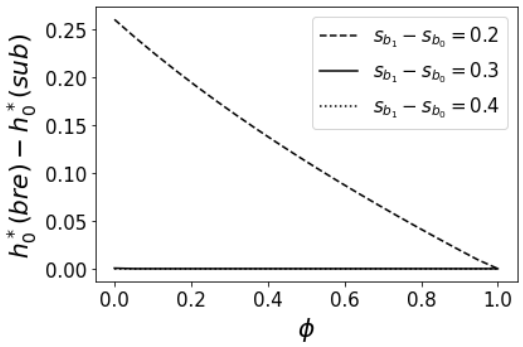

Figure A.6.2: Extent of breeder-subordinate conflict over delayed dispersal represented by the difference of predictions under breeder exclusive control or under subordinate exclusive control ($h_0^*(bre) - h_0^*(sub)$). The conflict varies with selfing rate ϕ and survival benefits $s_{b_1} - s_{b_0}$. Unless otherwise noted, $n_0 = n_1 = 3$, $s_u = s_{a_0} = 0.5$, $s_{a_1} = 0.6$, $a = 2$, $p_a = 0.25$, $p_u = 0.5$

Appendix B

Appendix Chapter 3

B.1 Inclusive fitness change

Let's fix attention on an actor who has experienced brood failure and who has at least one patchmate who has not. In this case,

$$\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})} = \frac{P_{k-1,n-1}}{1-p^{n-1}} \tag{B.1.1}$$

gives the probability that the actor inhabits a patch with exactly $1 \leq k \leq n-1$ brood failures, including its own.

The actor increases its level of phenotypic expression (i.e., its tendency toward redirected helping) by a small amount $\delta > 0$, and three primary inclusive-fitness changes result from this increase. First, the actor changes its survival by $\delta s_0'(0)$. Second, it changes the survival of a patchmate by $\delta s_1'(0)$. Third, the actor changes the fecundity of its patchmate in the amount $Nf'(0)\delta$ (e.g., the actor improves the fledging success of the N offspring produced by its patchmate). The expression

$$(1-d)\frac{1}{f(0)}\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\left[\,(n-k)(1-d)+d(1-c)n(1-p)\,\right]} + d(1-c)\sum_{i=0}^{n} P_{i,n}\frac{1}{f(0)}\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{N\left[\,(n-i)(1-d)+d(1-c)n(1-p)\,\right]} \quad \text{(B.1.2)}$$

translates improved fecundity into competitive success. Putting these observations together gives us

$$\begin{aligned}&\delta s_0'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}} + \delta\bar{r}s_1'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}\\ &+\delta\bar{r}Nf'(0)(1-d)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}\frac{1}{f(0)}\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\left[\,(n-k)(1-d)+d(1-c)n(1-p)\,\right]}\\ &+\delta\bar{r}Nf'(0)d(1-c)\sum_{i=0}^{n}P_{i,n}\frac{1}{f(0)}\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{N\left[\,(n-i)(1-d)+d(1-c)n(1-p)\,\right]}\end{aligned} \quad \text{(B.1.3)}$$

as the primary inclusive-fitness change that results from the actor's change in phenotype. As an aside, one can more easily make sense of the previous line by recognizing that $\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}$ is equal to 1.

The primary fitness changes in (B.1.3) must be compensated by changes in the opposite direction by other individuals in the population [78]. Compensation associated with the first three terms is made by individuals who carry alleles that are identical-by-descent to the one carried by the actor with probability $h_k\bar{r}$. By contrast, compensation associated with last term (gains made by offspring who disperse) is made by individuals whose alleles are not identical-by-descent to the one carried by the actor. It follows that

$$\delta s_0'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}(1-\bar{r}h_k)+\delta s_1'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}(\bar{r}-\bar{r}h_k)$$
$$+\delta N f'(0)(1-d)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}\frac{1}{f(0)}\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\,[\,(n-k)(1-d)+d(1-c)n(1-p)\,]}(\bar{r}-\bar{r}h_k)$$
$$+\delta N f'(0)d(1-c)\sum_{i=0}^{n}P_{i,n}\frac{1}{f(0)}\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{N\,[\,(n-i)(1-d)+d(1-c)n(1-p)\,]}\bar{r} \tag{B.1.4}$$

as the overall inclusive-fitness change experienced by the actor. We do not change the sign of the overall inclusive-fitness change when we multiply by $p(1-p^{n-1})/\delta$ (the numerator, here, gives probability that an individual experiences brood failure and at least one of its $n-1$ neighbors does not). By carrying out this multiplication, though, we arrive at the expression in (3.6) derived with the neighbor-modulated fitness approach. Thus, the inclusive-fitness argument and the neighbor-modulated fitness argument agree on the direction in which selection acts on redirected helping.

We now return our attention to the expression in (B.1.2). We weigh this expression by

$$\frac{P_{k,n}(n-k)}{n(1-p)}=P_{k,n-1}, \tag{B.1.5}$$

which is the probability an offspring is born on a patch with $0\leq k\leq n-1$ brood failures, and sum over k to obtain,

$$(1-d)\frac{1}{f(0)}\sum_{k=0}^{n-1}\frac{P_{k,n}(n-k)}{n(1-p)}\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\,[\,(n-k)(1-d)+d(1-c)n(1-p)\,]}$$
$$+d(1-c)\frac{1}{f(0)}\sum_{i=0}^{n}P_{i,n}\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{N\,[\,(n-i)(1-d)+d(1-c)n(1-p)\,]}. \tag{B.1.6}$$

To simplify the previously line, we collect the terms in $(1-s_0(0))$ separately

from those in $(1 - s_1(0))$ to obtain

$$\frac{1 - s_0(0)}{Nf(0)} \left(\sum_{k=0}^{n-1} P_{k,n} \frac{k}{n(1-p)} h_k + \sum_{i=0}^{n} P_{i,n} \frac{i}{n(1-p)} (1 - h_i) \right) + \frac{1 - s_1(0)}{Nf(0)} \left(\sum_{k=0}^{n-1} P_{k,n} \frac{n-k}{n(1-p)} h_k + \sum_{i=0}^{n} P_{i,n} \frac{(n-i)}{n(1-p)} (1 - h_i) \right). \quad \text{(B.1.7)}$$

We recognize that we can extend the sum in k to $k = n$ because $h_n = 0$. We also recognize that we can exchange the index i for the index k. Thus, the previous expression can be simplified further as

$$\frac{1 - s_0(0)}{Nf(0)n(1-p)} \sum_{k=0}^{n} k P_{k,n} + \frac{1 - s_1(0)}{Nf(0)n(1-p)} \sum_{k=0}^{n} (n-k) P_{k,n} = \frac{1}{f(0)} \psi \quad \text{(B.1.8)}$$

where

$$\psi = \frac{p(1 - s_0(0)) + (1-p)(1 - s_1(0))}{N(1-p)}. \quad \text{(B.1.9)}$$

The quantity ψ is understood as either the reproductive value of offspring at birth or equivalently as the establishment probability for the variable brood-failure model. Under the former interpretation, we set the reproductive value of an individual at the very beginning of the year equal to 1, without loss of generality. Then, the numerator of ψ represents the per-capita reproductive value forfeited by individuals who die in that year. The forfeiture is divided among the annual per-capita reproductive success — the denominator of ψ — to arrive at the reproductive value of an offspring at birth.

Inspired by our new insight about reproductive value, we re-write the overall inclusive-fitness effect of the actor's phenotypic change in (B.1.4) as

$$\delta s_0'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}(1-\bar{r}h_k)+\delta s_1'(0)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}(\bar{r}-\bar{r}h_k)$$

$$+\delta N\psi f'(0)(1-d)\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}\frac{1}{f(0)}\underbrace{\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\,[\,(n-k)(1-d)+d(1-c)n(1-p)\,]\psi}}_{\text{term i}}(\bar{r}-\bar{r}h_k)$$

$$+\,\delta N\psi f'(0)d(1-c)\sum_{i=0}^{n}P_{i,n}\frac{1}{f(0)}\underbrace{\frac{i(1-s_0(0))+(n-i)(1-s_1(0))}{N\,[\,(n-i)(1-d)+d(1-c)n(1-p)\,]\psi}}_{\text{term ii}}\bar{r} \tag{B.1.10}$$

and interpret terms i and ii as elements of a decomposition of offspring reproductive value at birth. Going a step further, we recognize that the inclusive-fitness effect has the same sign as

$$-s_0(0)C+s_1(0)B_sR_s+B_fN\psi R_f \tag{B.1.11}$$

where $C=-s_0'(0)/s_0(0)$, $B_s=s_1'(0)/s_1(0)$, and $B_f=f'(0)/f(0)$, and

$$R_s=\frac{\bar{r}-\bar{r}\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}h_k}{1-\bar{r}\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}h_k}=\frac{\bar{r}-\bar{r}\sum_{k=1}^{n-1}\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})}h_k}{1-\bar{r}\sum_{k=1}^{n-1}\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})}h_k} \tag{B.1.12}$$

as well as

$$\begin{aligned}R_f&=\frac{d(1-c)\bar{r}\sum_{k=0}^{n}P_{k,n}\frac{\psi_k'}{\psi}+(1-d)\bar{r}\sum_{k=1}^{n-1}\frac{P_{k-1,n-1}}{1-p^{n-1}}\frac{\psi_k'}{\psi}(1-h_k)}{1-\bar{r}\sum_{k=1}^{n-1}\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})}h_k}\\&=\frac{d(1-c)\bar{r}\sum_{k=0}^{n}P_{k,n}\frac{\psi_k'}{\psi}+(1-d)\bar{r}\sum_{k=1}^{n-1}\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})}\frac{\psi_k'}{\psi}(1-h_k)}{1-\bar{r}\sum_{k=1}^{n-1}\frac{k}{n}\frac{P_{k,n}}{p(1-p^{n-1})}h_k}\end{aligned} \tag{B.1.13}$$

with ψ_k' standing in for $\frac{k(1-s_0(0))+(n-k)(1-s_1(0))}{N\,[\,(n-k)(1-d)+d(1-c)n(1-p)\,]}$.

B.2 Coefficient of consanguinity

We define the coefficient of consanguinity (CC) between two individuals as the probability that the allele carried by one and the allele carried by the other are identical by descent [65]. We compute the CC between a focal individual and its average non-self patch mate in a selfish (selectively neutral) population using recursion. Again, note that weak selection justifies this neutral approximation.

Let $\bar{r}$ be the CC between a focal individual and its non-self patch mate. The CC between a focal individual and the average breeder on its patch (self-included) where k brood failures happened at the last time step is $\bar{r}_\bullet = \frac{1}{n-k} + \frac{n-k-1}{n-k}\bar{r}$. By choosing two random individuals on the same patch, given the different events happening during a time step, we can express the equilibrium value of $\bar{r}_\bullet$ as

$$\begin{aligned}\bar{r}_\bullet = \frac{1}{n-k} + \frac{n-k-1}{n-k}\sum_{k=0}^{n} P_{k,n}\Bigg[&\frac{n-k}{n}\frac{n-k-1}{n-1}\Bigg(s_1^2\bar{r} \\ &+2s_1(1-s_1)h_k\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big)+(1-s_1)^2h_k^2\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big)\Bigg) \\ &+2\frac{n-k}{n}\frac{k}{n-1}\Bigg(s_0s_1\bar{r}+s_0(1-s_1)h_k\bar{r}+s_1(1-s_0)h_k\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big) \\ &\qquad+(1-s_0)(1-s_1)h_k^2\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big)\Bigg) \\ &+\frac{k}{n}\frac{k-1}{n-1}\Bigg(s_0^2\bar{r}+2s_0(1-s_0)h_k\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big) \\ &\qquad+(1-s_0)^2h_k^2\Big(\frac{n-k-1}{n-k}\bar{r}+\frac{1}{n-k}\Big)\Bigg)\Bigg],\end{aligned} \tag{B.2.1}$$

where h_k is given in Table 3.1 of the main text. Simplifying the notations, we

have $\bar{r}_\bullet = \frac{1}{n-k} + \frac{n-k-1}{n-k}(\alpha + \bar{r}\beta)$, where

$$\alpha = \sum_{k=0}^{n-1} P_{k,n} \frac{h_k}{n-k}\Big[\frac{n-k}{n}\frac{n-k-1}{n-1}(2s_1(1-s_1)+(1-s_1)^2h_k) + 2\frac{n-k}{n}\frac{k}{n-1}(s_1(1-s_0)+(1-s_1)(1-s_0)h_k) + \frac{k}{n}\frac{k-1}{n-1}(1-s_0)^2h_k\Big],$$

and

$$\beta = \sum_{k=0}^{n-1} P_{k,n}\Big[\frac{n-k}{n}\frac{n-k-1}{n-1}(s_1^2 + \frac{n-k-1}{n-k}h_k(2s_1(1-s_1)+(1-s_1)^2h_k)) + 2\frac{n-k}{n}\frac{k}{n-1}(s_0s_1 + s_0(1-s_1)h_k + (1-s_0)s_1\frac{n-k-1}{n-k}h_k + (1-s_0)(1-s_1)h_k^2\frac{n-k-1}{n-k}) + \frac{k}{n}\frac{k-1}{n-1}(s_0^2 + 2s_0(1-s_0)h_k + (1-s_0)^2h_k^2\frac{n-k-1}{n-k})\Big] + s_0^2$$

Solving for $\bar{r}_\bullet = \frac{1}{n-k} + \frac{n-k-1}{n-k}\bar{r}$ and(B.2.1), we obtain

$$\bar{r} = \frac{\alpha}{1-\beta}. \tag{A.2}$$

This CC is used in R_s and R_f expressions that can be found in Table 3.1.

B.3 Prediction in the limit as p goes to 1^-

In this subsection, we characterize the behavior of the critical cost-benefit ratio R_f when the brood failure probability p goes to 1^-. Note that the cost-benefits ratio associated with survival benefits goes to 0 as p goes to 1^-.

We first observe that as $p \to 1^-$, $P_{k,n}$ goes to 0, except when $k = n$. Thus, CC $\bar{r} \to 0$, as $\alpha \to 0$ and $\beta = s_0^2$ for $m = n$. This limit implies that the denominator of R_f (see Table 3.1) goes to 1 as p goes to 1^-.

We then observe that the second term in the numerator goes to 0 as $p \to 1^-$

since the $P_{k,n}$ terms are equal to 0 and ψ_k remain between 0 and 1 for all $k \in [0, n-1]$. We thus look at the first part of the numerator of R_f, which corresponds to the fitness gains for offspring deciding to disperse. Recall that its expression is: $\bar{r}d(1-c)\sum_{k=0}^{n} P_{k,n}\psi_k$

At first glance, this formula appears to be undetermined as $p \to 1^-$: the CC $\bar{r}$ is going to 0, and the reproductive value of the offspring ψ_k goes to $+\infty$ for $k = n$. To compute the limit of R_f as $p \to 1^-$, we use approximations for ψ_n and $\bar{r}$ as $p \to 1^-$. Given the expression of ψ_n, we see that as $p \to 1^-$, $\psi_n = \frac{C}{1-p} + O((1-p)^2)$ in the neighborhood of $p = 1$, where C is a positive constant. Looking at $\bar{r}$, in the neighborhood of $p = 1$, we have

$$\bar{r} = \bar{r}|_{p\to 1^-} + (p-1)\partial_p \bar{r}|_{p\to 1^-} + O((1-p)^2) = 0 + (p-1)\partial_p \bar{r}|_{p\to 1^-} + O((1-p)^2)$$

The derivative of $\bar{r}$ is $\partial_p \bar{r} = \frac{(1-\beta)\partial_p \alpha + \alpha \partial_p \beta}{(1-\beta)^2}$, where α and β are given in the previous appendix. The limit of the denominator as $p \to 1^-$ is $(1-s_0^2)^2 > 0$. As $\alpha \to 0$ when $p \to 1^-$, we also have $\alpha \partial_p \beta \to 0$. We thus only need to compute the derivative of α to determine the behavior of $\bar{r}$ as $p \to 1^-$. We have

$$\partial\alpha = \sum_{k=0}^{n-1} \partial_p P_{k,n}\alpha_k + P_{k,n}\partial_p \alpha_k,$$

where α_k equals

$$\frac{h_k}{n-k}\Big[\frac{n-k}{n}\frac{n-k-1}{n-1}(2s_1(1-s_1) + (1-s_1)^2 h_k) + 2\frac{n-k}{n}\frac{k}{n-1}(s_1(1-s_0) + (1-s_1)(1-s_0)h_k) + \frac{k}{n}\frac{k-1}{n-1}(1-s_0)^2 h_k\Big]$$

Since we have $P_{k,n} \to 0$ for $k < n$, $\sum_{k=0}^{n-1} P_{k,n}\partial_p \alpha_k \to 0$. Now, we have

$$\partial_p \alpha = \partial_p \sum_{k=0}^{n-1} P_{k,n}\alpha_k = \sum_{k=0}^{n-1} nP_{k-1,n-1}\alpha_k - \frac{n!}{(n-k-1)!\,k!} p^k (1-p)^{n-k-1}\alpha_k \to -n\alpha_{n-1} = -C,$$

as $p \to 1^-$ and where C is a positive constant. In the neighborhood of $p = 1$ to 1^-, $\bar{r} = -(p-1)C + O((1-p)^2)$.

Finally, we have:

$$\psi_n \bar{r} = \frac{-C(p-1)}{(1-p)} + o((1-p)^2) = C + O((1-p)^2) \tag{B.1}$$

This result shows that as $p \to 1^-$, $R_f \to C$ where C is a constant in $\mathbb{R}$.

Appendix C

Appendix Chapter 4

Here, I present the numerical methods used to compute the neighbor-modulated fitness and fitness changes of redirected help in a viscous population in a stepping-stone model. These numerical methods compute the neighbor-modulated fitness change for survival and fecundity benefits, which leads to the results presented in Chapter 4.

Libraries

```
using LinearAlgebra
```

To compute the neighbor modulated fitness of the focal individual, we compute this fitness in all possible neighborhoods which depends on the brood successes and failures of the neighbors of the focal individual. In the case of survival benefits, the neighborhood is composed of the neighbors up to 3 steps away which give 2^7 possible neighborhood. In the case of fecundity benefits, we need to look at the neighbors up to 4 steps away which give 2^9 possible neighborhood.

Survival benefits

Parameters

```
Cs = 1 # Cost of helping
Bs = 1 ## Benefits of helping (survival)
d = 0.2 # Dispersal rate
c = 0.5 # Cost of dispersal
u = 1e-6 # Dispersal rate (long distance)
```

We start by looking at the survival benefits. The 2^7 cases can be reduced to 17 thanks to the symmetry of the neighborhood. The probability of the 17 identified cases is given by the following vector:

```
P(p)=[p^3, p*(1-p^2), p^4*(1 - p), 2*p^3*(1 - p)^2, 2*p^2*(1 - p)^3,
    p^2*(1 - p)^3, 2*p*(1 - p)^4, (1 - p)^5, p^4*(1 - p)^3, 2*p^3*(1 - p)^4,
    p^2*(1 - p)^5, 2*p^4*(1 - p)^2, 2*p^3*(1 - p)^3, 2*p^3*(1 - p)^3,
2*p^2*(1 - p)^4, 2*p^2*(1 - p)^4, 2*p*(1 - p)^5]

P (generic function with 1 method)
```

where p is the brood failure probability.

We then define the functions giving the survival of an individual who succeeds its brood, s_1(z1) and of an individual who fails its brood s_0(z)

```
s0(z0,z1) = s_0-Cs*s_0*(z0+z1); # when the focal individual fail, it will
survive with rate s0(z0,0) as it helps. When a 1-step neighbor fails, it will
give help so will survive with rate s0(0,z1).
s1(z0,z1,z2) = s_1+Bs*s_1*(z0+z1+z2) # Here this survival concerns only the
focal individual and its 1-step neighbors.

s1 (generic function with 1 method)
```

We can now compute the probability that an individual will keep its breeding spot, that its offspring will compete successfully on its breeding spot (local) or will successfully compete on the left or right side breeding spot. We store these four probabilities in a 17x4 matrix as we have 17 cases.

```
W(z0,z1,z2) = [
    s0(0)                0  0                                          0;
    s0(z0,0)             0  0                                          0;
    s1(0,2*z1,0)    1 - s1(0,2*z1,0)  1 - s0(0,z1)            1 - s0(0,z1);
    s1(0,2*z1,0)/2 + s1(0,z1,0)/2  1 - (s1(0,2*z1,0)/2 + s1(0,z1,0)/2)  1 -
s0(0,z1)  (1 - s0(0,z1))/2;
    s1(0,z1,0)     (1 - s1(0,z1,0))*(1 - d - u)/(1 - u*c - d/2*(1 + c))  1 -
s0(0,z1)  (1 - s1(0,0,0))*d*(1 - c)/2/(1 - u*c - c*d);
    s1(0,2*z1,0)/4 + s1(0,z1,0)/2 + s1(0,0,0)/4  1 - s1(0,2*z1,0)/4 -
s1(0,z1,0)/2 - s1(0,0,0)/4  (1 - s0(0,z1))/2  (1 - s0(0,z1))/2;
    s1(0,z1,0)/2 + s1(0,0,0)/2  (1 - s1(0,z1,0)/2 - s1(0,0,0)/2)*(1 - d -
u)/(1 - u*c - d/2*(1 + c))  (1 - s0(0,z1))/2  (1 - s1(0,0,0))*d*(1 - c)/2/(1
- u*c - c*d);
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1 - u*c - c*d)  ((1 -
s1(0,0,0))*d*(1 - c))/2/(1 - u*c- c*d)  (1 - s1(0,0,0))*d*(1 - c)/2/(1 - u*c
- c*d);
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1 - u*c - c*d)  ((1 -
s1(0,0,z2))*d*(1 - c))/2/(1 - u*c - d/2*(1 + c))  (1 - s1(0,0,z2))*d*(1 -
c)/2/(1 - u*c - d/2*(1 + c));
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1 - u*c - c*d)  ((1 -
s1(0,0,z2))*d*(1 - c))/2/(1 - u*c - d/2*(1 + c))  (1 - s1(0,0,z2)/2 -
s1(0,0,0)/2)*d*(1 - c)/2/(1 - u*c - d/2*(1 + c));
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1 - u*c - c*d)  ((1 -
s1(0,0,z2)/2 - s1(0,0,0)/2)*d*(1 - c))/2/(1 - u*c - d/2*(1 + c))  (1 -
s1(0,0,z2)/2 - s1(0,0,0)/2)*d*(1 - c)/2/(1 - u*c - d/2*(1 + c));
    s1(0,z1,0)     (1 - s1(0,z1,0))*(1 - d - u)/(1 - u*c - d/2*(1 + c))  (1 -
s1(0,0,z2))*d*(1 - c)/2/(1 - u*c - d/2*(1 + c))  1 - s0(0,z1);
    s1(0,z1,0)     (1 - s1(0,z1,0))*(1 - d - u)/(1 - u*c - d/2*(1 + c))  (1 -
s1(0,0,z2)/2 - s1(0,0,0)/2)*d*(1 - c)/2/(1 - u*c - d/2*(1 + c))  1 -
s0(0,z1);
    s1(0,z1,0)/2 + s1(0,0,0)/2  (1 - s1(0,z1,0)/2 - s1(0,0,0)/2)*(1 - d -
u)/(1 - u*c - d/2*(1 + c))  (1 - s1(0,0,z2))*d*(1 - c)/2/(1 - u*c - d/2*(1 +
c))  (1 - s0(0,z1))/2;
    s1(0,z1,0)/2 + s1(0,0,0)/2  (1 - s1(0,z1,0)/2 - s1(0,0,0)/2)*(1 - d -
u)/(1 - u*c - d/2*(1 + c))  (1 - s1(0,0,z2)/2 - s1(0,0,0)/2)*d*(1 - c)/2/(1 -
u*c - d/2*(1 + c))  (1 - s0(0,z1))/2;
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1- u*c - c*d)  (1 -
s1(0,0,z2))*d*(1 - c)/2/(1 - u*c - d/2*(1 + c))  (1 - s1(0,0,0))*d*(1 -
c)/2/(1 - u*c - c*d);
    s1(0,0,0)        (1 - s1(0,0,0))*(1 - d - u)/(1 - u*c - c*d)  (1 -
s1(0,0,z2)/2 - s1(0,0,0)/2)*d*(1 - c)/2/(1 - u*c - d/2*(1 + c))  (1 -
s1(0,0,0))*d*(1 - c)/2/(1 - u*c - c*d);
]

W (generic function with 1 method)
```

The first column gives the probability that the individual will survive and will keep its breeding spot, the second gives the probabilty its offspring will get the breeding spot given it dies. The third and fourth column gives the probability that the offspring will get the

breeding spot on the left and on the right side respectively. Note that these two last columns use the symmetry of the space to reduce the number of cases.

By summing the two first columns together and weighting each of the 17 terms by the probability that the neighborhood will happen, we obtain the probability that an individual or its offspring will keep the breeding spot, which we denote by p0. Similarly, we can compute the probability that an individual will get a breeding spot one step away, denoted p1. Note that we divide the expression by 2 to compute for only one breeding spot.

```
p0(z0,z1,z2,p) = sum(P(p).*(W(z0,z1,z2)[:,1]+W(z0,z1,z2)[:,2]))
p1(z0,z1,z2,p) = sum(P(p).*(W(z0,z1,z2)[:,3]+W(z0,z1,z2)[:,4]))/2
```

We can now compute the coefficients of consanguinity r_i using the matrix shown in the main text.

```
A(z0,z1,z2,p) = p1(z0,z1,z2,p)^2
B(z0,z1,z2,p) = 2*p0(z0,z1,z2,p)*p1(z0,z1,z2,p)
D(z0,z1,z2,p) = p0(z0,z1,z2,p)^2+2*p1(z0,z1,z2,p)^2

L(z0,z1,z2,p) = 1/A(z0,z1,z2,p)*[-B(z0,z1,z2,p) (1-D(z0,z1,z2,p)) -
B(z0,z1,z2,p) -A(z0,z1,z2,p); A(z0,z1,z2,p) 0 0 0; 0 A(z0,z1,z2,p) 0 0; 0 0
A(z0,z1,z2,p) 0]
```

Following the solution shown in the text, we have the coefficients of consanguinity:

```
function r(p)
    lambda = eigvals(L(0,0,0,p))
    l1, l2 = lambda[abs.(real.((lambda))) .<1]

    cc = (l2-l2*l1^2)/(l2^2*l1-l2*l1^2+l2-l1)
    dd = 1-cc
    r1 = cc*l2+dd*l1 # CC between focal and 1-step neighbor
    r2 = cc*l2^2+dd*l1^2 # CC between focal and 2-step neighbor
    r3 = cc*l2^3+dd*l1^3 # CC between focal and 3-step neighbor
    return r1,r2,r3
end
```

I can now compute the neighbor modulated fitness change associated with survival benefits. I use a finite difference scheme to compute the change of fitness for the focal individual (dWz0), its direct neighbors (dWz1) and its 2 step neighbors (dWz2) for all possible neighborhood. I then sum the three fitness change weighted by the accurate coefficient of consanguinity and the probability that the neighborhood happens. The sum of all the fitness change in all the possible neighborhood gives the neighbor-modulated fitness change associated with survival benefits (dW).

```
function dw(p)
    dWz0 = zeros(17)
    dWz1 = zeros(17)
```

```
    dWz2 = zeros(17)
    h = 1e-4
    for i in range(1,17)
        dWz0[i] = sum(W(h,0,0)[i,:]-W(0,0,0)[i,:])/h
        dWz1[i] = sum(W(0,h,0)[i,:]-W(0,0,0)[i,:])/h
        dWz2[i] = sum(W(0,0,h)[i,:]-W(0,0,0)[i,:])/h
    end
    dW = sum(P(p).*(dWz0.+r(p)[1].*dWz1 .+r(p)[2].*dWz2))
    return dW
end
```

Fecundity benefits

The fecundity benefits case is more complex than the survival ones and require to compute numericall all the possible neighborhoods. To do so, I use a binary representation of all the 2^9 possible cases. The function bin(n) gives the binary representation of an integer. By scanning all the integers between 1 and 2^9=512, I model all possible neighborhood by a vector of 0 and 1 which are written in the tab_fail matrix. By summing the vector associated with every neighborhood in tab_fail, I can compute how many success happened in the neighborhood and give the probability that every neighborhood happens given the brood failure probability p in the function Proba(p).

```
function bin(n)
    binary_str = zeros(9)
    for i=1:9
        if n==0
            break
        end
        if (n-2^(i-1))%2^i==0

            n -= 2^(i-1)
            binary_str[10-i] = 1
        else
            binary_str[10-i] = 0
        end

    end
    return binary_str
end
tab_fail=zeros(512,9)
for j in range(1,512)
    tab_fail[j,:] = bin(j)
end

function Proba(p)
    proba = zeros(512)
    for j=1:512
        proba[j] = p^(9-sum(tab_fail[j,:]))*(1-p)^(sum(tab_fail[j,:]))
```

```
    end
    return proba
end
```

I now define the survival and fecundity (fledging success) function s0f(z) and f(z0,z1,z2,z3) respectively. Recall that helping decreases the survival of the helper and increases the fledging success of the offspring of the helped individual.

```
o = 5; # focal individual position on the vector
nb_class = 512;
# Setting parameters values, these values may change during computations
s_1 = 0.7
s_0 = 0.5
C = 1
B = 1
s0f(z) = s0-C*s0*z

z0 = 0.01
z1 = 0.01
z2 = 0.01
z2 = 0.01
h = 1e-3
F = 2
F0 = 1
f(z0,z1,z2,z3) = F0+F0*F*(z0+z1+z2+z3)
```

```
f (generic function with 1 method)
```

I now compute the survival of the focal individual located at the center of each neighborhood (So(z0)), the survival of the individual located to the right of the focal individual (Sp1(z1)), and the survival of the individual located to the left of the focal individual (Sm1(z1)). If the individual succeeds, they survive at rate s1, if they fail and one of their direct neighbor didn't fail, they help and their survival is s0f(z0). Otherwise, they don't help and survive at rate s0f(0). The survival in each neighborhood is then computed inside a vector.

```
function So(z0)
    so = zeros(nb_class)
    for i=1:nb_class
        if tab_fail[i,o] == 1
            so[i] = s1
        elseif tab_fail[i,o-1]==1 || tab_fail[i,o+1] == 1
                so[i] = s0f(z0)
            else
                so[i] = s0f(0)
        end

    end
    return so
end
```

```
function Sp1(z1)
    sp1 = zeros(nb_class)

    for i=1:nb_class
        if tab_fail[i,o+1] == 1
            sp1[i] = s1
        elseif tab_fail[i,o]==1 || tab_fail[i,o+2] == 1
                sp1[i] = s0f(z1)
            else
                sp1[i] = s0f(0)
        end
    end
    return sp1
end

function Sm1(z1)
    sm1 = zeros(nb_class)

    for i=1:nb_class
        if tab_fail[i,o-1] == 1
            sm1[i] = s1
        elseif tab_fail[i,o-2]==1 || tab_fail[i,o] == 1
                sm1[i] = s0f(z1)
            else
                sm1[i] = s0f(0)
        end
    end
    return sm1
end
```

Similarly, I compute the fledging success of the focal individual (Feco(z1)), its direct neighbors left (Fecom1(z0,z2)) and right (Fecop1(z0,z2), and its 2-step neighbors left (Fecom2(z1,z3)) and right (Fecop2(z1,z3)). Fledging success depends on the help provided by the 2 neighbors of an individual.

```
function Feco(z1)
    feco = zeros(nb_class)
    for i=1:nb_class

        if tab_fail[i,o] == 0
            feco[i] = 0
        elseif tab_fail[i,o-1] == 1 && tab_fail[i,o+1] == 1
            feco[i] = f(0,0,0,0)
        elseif tab_fail[i,o-2] == 0 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o+2] == 0
            feco[i] = f(0,2*z1,0,0)
        elseif tab_fail[i,o-2] == 0 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o+2] == 1
```

```
            feco[i] = f(0,2*z1,0,0)/2+f(0,z1,0,0)/2
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o+2] == 1
            feco[i] = f(0,2*z1,0,0)/4+f(0,z1,0,0)/2+f(0,0,0,0)/4
        elseif tab_fail[i,o-2] == 0 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 1
            feco[i] = f(0,z1,0,0)
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 1
            feco[i] = f(0,z1,0,0)/2+f(0,0,0,0)/2
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o-1] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o+2] == 0
            feco[i] = f(0,2*z1,0,0)/2+f(0,z1,0,0)/2
        elseif tab_fail[i,o+2] == 0 && tab_fail[i,o+1] == 0 && tab_fail[i,o-
1] == 1
            feco[i] = f(0,z1,0,0)
        elseif tab_fail[i,o+2] == 1 && tab_fail[i,o+1] == 0 && tab_fail[i,o-
1] == 1
            feco[i] = f(0,z1,0,0)/2+f(0,0,0,0)/2
        else
          feco[i] = 1000
        end
    end
    return feco
end

function Fecom1(z0,z2)
    feco = zeros(nb_class)
    for i=1:nb_class
        if tab_fail[i,o-1] == 0
            feco[i] = 0
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o] == 1
            feco[i] = f(0,0,0,0)
        elseif tab_fail[i,o-3] == 0 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o+1] == 0
            feco[i] = f(z0,0,z2,0)
        elseif tab_fail[i,o-3] == 0 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o+1] == 1
            feco[i] = f(z0,0,z2,0)/2+f(0,0,z2,0)/2
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o+1] == 1
            feco[i] = f(z0,0,z2,0)/4+f(z0,0,0,0)/4+f(0,0,z2,0)/4+f(0,0,0,0)/4
        elseif tab_fail[i,o-3] == 0 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,0,z2,0)
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,0,z2,0)/2+f(0,0,0,0)/2
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o+1] == 0
```

```
            feco[i] = f(z0,0,z2,0)/2+f(z0,0,0,0)/2
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o] == 0 && tab_fail[i,o+1]
== 0
            feco[i] = f(z0,0,0,0)
        elseif tab_fail[i,o-2] == 1 && tab_fail[i,o] == 0 && tab_fail[i,o+1]
== 1
            feco[i] = f(z0,0,0,0)/2+f(0,0,0,0)/2
        else
          feco[i] = 1000
        end
    end
    return feco
end

function Fecop1(z0,z2)
    feco = zeros(nb_class)
    for i=1:nb_class
        if tab_fail[i,o+1] == 0
            feco[i] = 0
        elseif tab_fail[i,o+2] == 1 && tab_fail[i,o] == 1
            feco[i] = f(0,0,0,0)
        elseif tab_fail[i,o+3] == 0 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o-1] == 0
            feco[i] = f(z0,0,z2,0)
        elseif tab_fail[i,o+3] == 0 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o-1] == 1
            feco[i] = f(z0,0,z2,0)/2+f(0,0,z2,0)/2
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o-1] == 1
            feco[i] = f(z0,0,z2,0)/4+f(z0,0,0,0)/4+f(0,0,z2,0)/4+f(0,0,0,0)/4
        elseif tab_fail[i,o+3] == 0 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,0,z2,0)
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,0,z2,0)/2+f(0,0,0,0)/2
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+2] == 0 && tab_fail[i,o]
== 0 && tab_fail[i,o-1] == 0
            feco[i] = f(z0,0,z2,0)/2+f(z0,0,0,0)/2
        elseif tab_fail[i,o+2] == 1 && tab_fail[i,o] == 0 && tab_fail[i,o-1]
== 0
            feco[i] = f(z0,0,0,0)
        elseif tab_fail[i,o+2] == 1 && tab_fail[i,o] == 0 && tab_fail[i,o-1]
== 1
            feco[i] = f(z0,0,0,0)/2+f(0,0,0,0)/2
        else
          feco[i] = 1000
        end
    end
    return feco
```

```
end


function Fecop2(z1,z3)
    feco = zeros(nb_class)
    for i=1:nb_class
        if tab_fail[i,o+2] == 0
            feco[i] = 0
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+1] == 1
            feco[i] = f(0,0,0,0)
        elseif tab_fail[i,o+4] == 0 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o] == 0
            feco[i] = f(0,z1,0,z3)
        elseif tab_fail[i,o+4] == 0 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o] == 1
            feco[i] = f(0,z1,0,z3)/2+f(0,0,0,z3)/2
        elseif tab_fail[i,o+4] == 1 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o] == 1
            feco[i] = f(0,z1,0,z3)/4+f(0,z1,0,0)/4+f(0,0,0,z3)/4+f(0,0,0,0)/4
        elseif tab_fail[i,o+4] == 0 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 1
            feco[i] = f(0,0,0,z3)
        elseif tab_fail[i,o+4] == 1 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 1
            feco[i] = f(0,0,0,z3)/2+f(0,0,0,0)/2
        elseif tab_fail[i,o+4] == 1 && tab_fail[i,o+3] == 0 &&
tab_fail[i,o+1] == 0 && tab_fail[i,o] == 0
            feco[i] = f(0,z1,0,z3)/2+f(0,z1,0,0)/2
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+1] == 0 && tab_fail[i,o]
== 0
            feco[i] = f(0,z1,0,0)
        elseif tab_fail[i,o+3] == 1 && tab_fail[i,o+1] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,z1,0,0)/2+f(0,0,0,0)/2
        else
          feco[i] = 1000
        end
    end
    return feco
end

function Fecom2(z1,z3)
    feco = zeros(nb_class)
    for i=1:nb_class
        if tab_fail[i,o-2] == 0
            feco[i] = 0
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-1] == 1
            feco[i] = f(0,0,0,0)
        elseif tab_fail[i,o-4] == 0 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 0 && tab_fail[i,o] == 0
```

```
            feco[i] = f(0,z1,0,z3)
        elseif tab_fail[i,o-4] == 0 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 0 && tab_fail[i,o] == 1
            feco[i] = f(0,z1,0,z3)/2+f(0,0,0,z3)/2
        elseif tab_fail[i,o-4] == 1 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 0 && tab_fail[i,o] == 1
            feco[i] = f(0,z1,0,z3)/4+f(0,z1,0,0)/4+f(0,0,0,z3)/4+f(0,0,0,0)/4
        elseif tab_fail[i,o-4] == 0 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 1
            feco[i] = f(0,0,0,z3)
        elseif tab_fail[i,o-4] == 1 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 1
            feco[i] = f(0,0,0,z3)/2+f(0,0,0,0)/2
        elseif tab_fail[i,o-4] == 1 && tab_fail[i,o-3] == 0 && tab_fail[i,o-
1] == 0 && tab_fail[i,o] == 0
            feco[i] = f(0,z1,0,z3)/2+f(0,z1,0,0)/2
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-1] == 0 && tab_fail[i,o]
== 0
            feco[i] = f(0,z1,0,0)
        elseif tab_fail[i,o-3] == 1 && tab_fail[i,o-1] == 0 && tab_fail[i,o]
== 1
            feco[i] = f(0,z1,0,0)/2+f(0,0,0,0)/2
        else
          feco[i] = 1000
        end
    end
    return feco
end
```

I now compute the probability for the focal individual to keep its breeding spot or that one of its offspring compete successfully for it (P0_f(z0,z1,z2,z3,p)). I also compute the probability for the offspring of the focal individual to compete successfully on the breeding spot on the left (P1l_f(z0,z1,z2,z3,p) and on the right (P1r_f(z0,z1,z2,z3,p)). Again, I scan all possible neighborhood and compute the probability to get the breeding spot and weight these probabilities by the probability the neighborhood happens, and finally sum all these probabilities.

```
function P0_f(z0,z1,z2,z3,p)
    P0 = 0
    for i in range(1,nb_class)
        P0 += Proba(p)[i]*(So(z0)[i]+(1-So(z0)[i])*(1-d-u)*Feco(z1)[i]/((1-d-
u)*Feco(z1)[i]+ d/2*(1-c)*(Fecom1(z0,z2)[i]+Fecop1(z0,z2)[i])+u*(1-
c)*f(0,0,0,0)))
    end
   return P0
end

function P1l_f(z0,z1,z2,z3,p)
    P1 = 0
```

```
    for i in range(1,nb_class)
        P1 += Proba(p)[i]*((1-Sm1(z1)[i])*d/2*(1-c)*Feco(z1)[i]/(d/2*(1-
c)*Feco(z1)[i]+(1-d-u)*Fecom1(z0,z2)[i]+d/2*(1-c)*Fecom2(z1,z3)[i]+u*(1-
c)*f(0,0,0,0)))
    end
   return P1
end

function P1r_f(z0,z1,z2,z3,p)
    P1 = 0
    for i in range(1,nb_class)
        P1 += Proba(p)[i]*((1-Sp1(z1)[i])*d/2*(1-c)*Feco(z1)[i]/(d/2*(1-
c)*Feco(z1)[i]+(1-d-u)*Fecop1(z0,z2)[i]+d/2*(1-c)*Fecop2(z1,z3)[i]+u*(1-
c)*f(0,0,0,0)))
    end
   return P1
end

function P1_f(z0,z1,z2,z3,p)
    P1 = 0
    for i in range(1,nb_class)
        P1 += Proba(p)[i]*((1-Sp1(z1)[i])*d/2*(1-c)*Feco(z1)[i]/(d/2*(1-
c)*Feco(z1)[i]+(1-d-u)*Fecop1(z0,z2)[i]+d/2*(1-c)*Fecop2(z1,z3)[i]+u*(1-
c)*f(0,0,0,0))+(1-Sm1(z1)[i])*d/2*(1-c)*Feco(z1)[i]/(d/2*(1-
c)*Feco(z1)[i]+(1-d-u)*Fecom1(z0,z2)[i]+d/2*(1-c)*Fecom2(z1,z3)[i]+u*(1-
c)*f(0,0,0,0)))/2
    end
   return P1
end
function Pinf_f(z1,p)
    Pinf = 0
    for i in range(1,nb_class)
        Pinf += (1-p)*u*(1-c)*Feco(z1)[i]*(1-s1)/(u*(1-c)*Feco(z1)[i]+(1-d-
u)*f(0,0,0,0)+d*(1-c)*f(0,0,0,0)*(1-p)+u*(1-c)*f(0,0,0,0))+p*u*(1-
c)*Feco(z1)[i]*(1-s0)/(u*(1-c)*Feco(z1)[i]+d*(1-c)*f(0,0,0,0)*(1-p)+u*(1-
c)*f(0,0,0,0))
    end
    return Pinf
end
```

As in the survival case, I can compute the matrix used to compute the coefficients of consanguinity r_i by using the expression of the main text.

```
A_t(z0,z1,z2,z3,p) = ((P1l_f(z0,z1,z2,z3,p)+P1r_f(z0,z1,z2,z3,p))/2)^2
B(z0,z1,z2,z3,p) =
2*P0_f(z0,z1,z2,z3,p)*(P1l_f(z0,z1,z2,z3,p)+P1r_f(z0,z1,z2,z3,p))/2
Cm(z0,z1,z2,z3,p) =
P0_f(z0,z1,z2,z3,p)^2+2*((P1l_f(z0,z1,z2,z3,p)+P1r_f(z0,z1,z2,z3,p))/2)^2

L(z0,z1,z2,z3,p) = 1/A_t(z0,z1,z2,z3,p)*[-B(z0,z1,z2,z3,p) (1-
```

```
Cm(z0,z1,z2,z3,p)) -B(z0,z1,z2,z3,p) -A_t(z0,z1,z2,z3,p); A_t(z0,z1,z2,z3,p)
0 0 0; 0 A_t(z0,z1,z2,z3,p) 0 0; 0 0 A_t(z0,z1,z2,z3,p) 0]
```

Now I compute the coefficients of consanguinity using the same method as in the survival case

```
function r(p)
    lambda = eigvals(L(0,0,0,0,p))
    l1, l2 = lambda[abs.(real.((lambda))) .<1]

    cc = (l2-l2*l1^2)/(l2^2*l1-l2*l1^2+l2-l1)
    dd = 1-cc
    r1 = cc*l2+dd*l1
    r2 = cc*l2^2+dd*l1^2
    r3 = cc*l2^3+dd*l1^3
    return r1,r2,r3
end
```

Now I compute the neighbor-modulated fitness change using the same method as in the survival case.

```
function dw(p)
    h = 1e-3
    dW0 = (P0_f(h,0,0,0,p)+2*P1_f(h,0,0,0,p)-P0_f(0,0,0,0,p)-
2*P1_f(0,0,0,0,p))/h
    dW1 = (P0_f(0,h,0,0,p)+2*P1_f(0,h,0,0,p)+Pinf_f(h,p)-P0_f(0,0,0,0,p)-
2*P1_f(0,0,0,0,p)-Pinf_f(0,p))/h
    dW2 = (P0_f(0,0,h,0,p)+2*P1_f(0,0,h,0,p)-P0_f(0,0,0,0,p)-
2*P1_f(0,0,0,0,p))/h
    dW3 = (P0_f(0,0,0,h,p)+2*P1_f(0,0,0,h,p)-P0_f(0,0,0,0,p)-
2*P1_f(0,0,0,0,p))/h
    r1,r2,r3 = r(p)

    dW = dW0+r1*dW1+r2*dW2+r3*dW3
return dW
end
```