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Conservation corridors affect the fixation of novel alleles

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Abstract

Corridors are a popular tool for conservation of small populations. However, two purported benefits of corridors, increasing gene flow and providing a means for the recolonization of extinct patches of habitat (population rescue), may have unappreciated impacts on the likelihood that a new allele will become incorporated (fixed) within a population. Using a simulation model, I demonstrate that connecting a stable, isolated population with a population that requires periodic rescue (due to extinction via natural or anthropogenic disturbance) can affect fixation of alleles in the stable population, largely by changing the effective population size *Ne* of the two-patch complex. When disturbance is rare, connecting the two patches with corridors can increase fixation of beneficial alleles and increase loss of harmful alleles. However, the opposite occurs when rates of disturbance are high: corridors can promote fixation of harmful alleles and reduce fixation of beneficial alleles. Because the impact of corridors hinges upon disturbance frequency (i.e. rate of population rescue), population growth rate, movement rates, and habitat quality, different species are likely to have different responses to corridor-mediated fixation, even if the species reside within the same ecological community. By changing fixation, corridors could thus either promote adaptation or extinction.

Introduction

Corridors that connect otherwise isolated populations are a popular conservation strategy (Rosenberg et al. 1997). Corridors have been shown to increase gene flow (e.g. Mech and Hallett 2001; Tewksbury et al. 2002; Haddad et al. 2003), potentially reducing the detrimental effects of inbreeding in small populations. Corridors may also promote population 'rescue', i.e. corridors provide a means for recolonization of a patch of habitat when stochastic events (e.g. anthropogenic disturbance, weather fluctuations, disease; Pickett and White 1985) lead to the extinction of a local population (Rosenberg et al. 1997). However, by increasing gene flow and population rescue, corridors may have unappreciated consequences for the likelihood that a new allele will become incorporated (fixed) in a population, a process that can ultimately promote adaptation (Dobzhansky 1970) or extinction (e.g. mutational meltdown; Lynch et al. 1995).

The likelihood that a single new allele (e.g. a new mutation) will become fixed in a large, stable population is approximately $2s$, where s represents the additive selective advantage of the new allele in a heterozygote (Haldane 1927). In large, stable populations where spatial structure does not change population size, this probability remains unchanged (Maruyama 1970). However, when

populations behave in a non-ideal sense, e.g. when populations are growing or shrinking, the likelihood of fixation can be affected (see Otto and Whitlock 1997; Whitlock and Barton 1997; Whitlock 2003). In growing populations, new beneficial alleles are more likely to become fixed relative to stable populations because genetic drift is less likely to remove the new allele while it is rare (Otto and Whitlock 1997). Conversely, harmful (deleterious) alleles are more likely to be lost in growing populations compared to stable populations, because selection removes the new deleterious allele before it drifts to fixation (Otto and Whitlock 1997). Spatial structure can have similar impacts on fixation, largely because spatial population structure can result in changes in effective population size, *Ne* (Whitlock 2003).

Although the consequences of fixation are well-appreciated by population geneticists and the importance of corridors is well-known by conservation biologists, the consequences of corridors on fixation have not been examined. The theory developed by Whitlock (2003) provides a method for generalizing fixation probability among an array of spatial population structures, and its results suggest that corridors could affect fixation dynamics, but its message is not specific to conservation and corridors are not specifically addressed. The goal of this paper is to provide conservation biologists with insight useful for evaluating the potential consequences of corridors on fixation. Additionally, this work evaluates how familiar ecological parameters (e.g. population growth rate, carrying capacity) interact with corridors to change fixation dynamics in small populations of conservation concern.

Methods

Description of basic model

Using a simulation model, I consider two populations that are identical with one exception: periodic disturbance leads to local extinction of one population (the ephemeral population) but does not affect any individuals in the other population (the stable refuge population, Figure 1). As such, the ephemeral population must be rescued by the refuge population. Colonists from the stable refuge population that arrive in the ephemeral population just after disturbance exhibit rapid logistic growth because of greatly reduced density dependence (Figure 1c). Within-patch population growth between timesteps is determined by the discrete logistic model: $N_{t+1} = N_t + rN_t(1 - N_t/k)$; where N_t represents number of individuals present in the patch time t, k represents carrying capacity, and r is the discrete rate of increase of the population. Both populations are identical (i.e. k, r, and s are the same), except the ephemeral population is disturbed every D generations, whereas the stable refuge population never experiences disturbance. Values of D were evaluated from $D = 0$ (the ephemeral population is disturbed every generation) to $D = 275$. Over the parameter space evaluated, additional simulations suggested no change in model output when $D > 275$.

At the start of each simulation, the stable population is at carrying capacity ($N_{\text{stable}} = k$) and contains normal alleles and a single mutant allele, as if a new mutation has just arisen; the ephemeral population is empty (i.e. $N_{\text{ephemeral}} = 0$), as if a disturbance has just occurred. Each generation, N_t pairs of alleles are selected with replacement from the gene pool, and these alleles form the next generation in that population. The likelihood that an allele will be selected is determined by: $p_t \times ((1 + s)/W_{t-1})$, where p_t is the frequency of the allele at timestep t, s represents the additive selective advantage of the new allele in a heterozygote ($S_{\text{normal allele}} = 0$), and W_{t-1} is the mean fitness of the previous generation (Otto and Whitlock 1997). After growth, a fixed proportion of alleles (m), selected randomly and without replacement from each population, moves to the other population, as if moving through a corridor or permeable landscape. On average, 1,779,909 simulations were performed for each value of D and m. A simulation concluded when the new allele either fixed (frequency of new allele $= 1$) or was lost from both populations.

I evaluate how changing the magnitude of k, m, r, and s affect fixation probability. Carrying capacity, k, is of interest because modern conservation efforts typically focus on the minimum size at which a population can be maintained without loss of viability (Frankham 1995). I examine $k = 200$, $k = 500$, and $k = 1250$, to represent population sizes potentially typical of endangered populations where corridors might be employed. Values of m, r, and s were chosen to be representative of

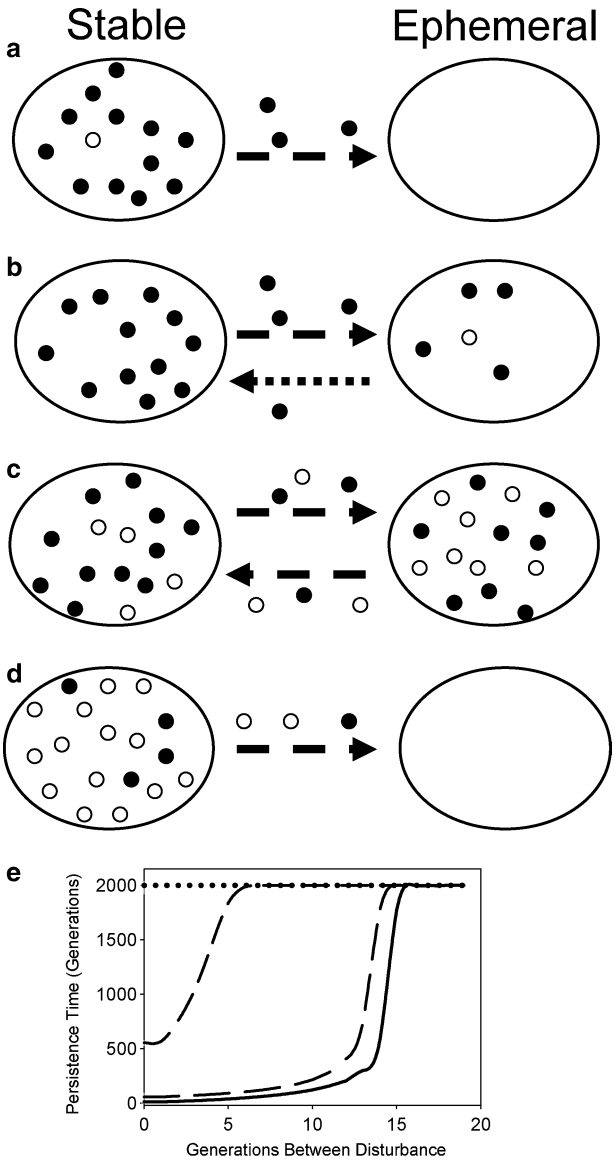

Figure 1. The two-patch model. An individual with a new allele (open circles) moves from the stable refuge population to the ephemeral population that has recently experienced local extinction (a) The ephemeral population experiences rapid logistic growth because of reduced density dependence (b) Migrants from the ephemeral population return to the refuge population and affect the likelihood that the new allele will fix there (c) Local extinction occurs again in the ephemeral population (d), but not before the new allele has become more frequent in the stable refuge population. (e) Local extinction can lead to global extinction of both populations when disturbance is frequent and movement rates are high because individuals leave the stable population more quickly than they are replaced by immigration or reproduction. Dotted, dashed, and solid lines represent four levels of increasing per capita movement rates (m): 0.01, 0.05, 0.1, and 0.3, respectively.

empirical estimates from natural populations (Dobzhansky 1970; Baker 1978; Blueweiss et al. 1978; Bowne and Bowers 2004) and comparable with values used in other studies of fixation (e.g. Otto and Whitlock 1997; Whitlock 2003).

Model output

Model output was compared to the null fixation probability (P^*) that would be observed if a single novel allele were to arise in a single, unconnected

Table 1. Model output was validated by comparison to haploid simulation values presented in Otto & Whitlock (1997) and those generated using the general formula of Kimura (1962). Null models using diploid populations were used to generate P/P^* (Figure 2) are in close agreement ($R^2 > 0.99$) with values obtained using Kimura (1962)

Model	Parameters				Fixation Probability (P)		
	Growth	r	k	S	Orrock	O&W	Kimura
Validation	Constant	—	100	0.01	0.0229	0.0227	0.0229
	Constant	—	100	−0.01	0.00313	0.00303	0.00316
	Logistic	0.1	200	0.01	0.0365	0.0364	0.0369
	Logistic	0.1	200	−0.01	0.000877	0.000853	0.000980
Null	Constant	—	500	0.001	0.002309	—	0.002311
	Constant	—	500	−0.001	0.000283	—	0.000313
	Constant	—	200	0.001	0.003651	—	0.003628
	Constant	—	200	−0.001	0.001604	—	0.001634
	Constant	—	500	0.01	0.019839	—	0.019801
	Constant	—	500	0.002	0.004117	—	0.004067
	Constant	—	500	−0.0015	0.000134	—	0.000157
	Constant	—	1250	0.001	0.002140	—	0.002012
	Constant	—	1250	−0.001	0.000022	—	0.000014

Models were run as a single, stable, closed population, i.e. there was no disturbed patch and no movement ($m = 0$). When population size was constant, $N_0 = N_t = k$. When population growth was simulated for the validation models, initial population size, N_0, was 100 haploid individuals.

refuge patch with no migration (Table 1). This is analogous to a tracking the fixation dynamics of an isolated population of concern that has just been connected with an empty, suitable patch of habitat with a corridor, or following the fate of an allele that enters the stable population just after the ephemeral population requires rescue. Values obtained via simulation (Table 1) were in excellent agreement with values obtained via analytical equation (Kimura 1962) and simulation (Otto and Whitlock 1997).

Results and discussion

The frequency with which the ephemeral population goes extinct has important impacts on whether corridors have positive or negative affects on fixation. When the ephemeral population rarely requires rescue due to infrequent local extinctions, corridor-mediated movement between patches leads to an increase in the effective size of the overall population (Ne), i.e. $Ne_{\text{stable}+\text{ephemeral}} > Ne_{\text{stable}}$ (Figure 1a–d; Whitlock 2003), making it more likely that beneficial alleles will fix and deleterious alleles will be lost (Figure 2). When disturbance is frequent (thus rescue occurs more often), the effective size of the refuge population is decreased (Whitlock 2003), and deleterious alleles are more likely to become fixed, whereas the converse is true for beneficial alleles (Figure 2a–e). When disturbance is very frequent and the ephemeral population requires regular rescue, continued losses of individuals to the ephemeral population lead to negative growth rates in the refuge, further increasing the likelihood that a deleterious mutation will become fixed (Figure 2a–e) and promoting demographic extinction of both populations (Figure 1e; Skellam 1951).

The qualitative effect of corridors on fixation depends upon the rate of growth, r, of the population relative to the rate at which the ephemeral population goes extinct and requires rescue (Figure 2a). Whether corridors have positive or beneficial effects on fixation also depends upon the proportion of alleles moving between patches (m, Figure 2b), and the number of individuals each patch can support (carrying capacity, k; Figure 2c). For example, when populations are large ($k = 1250$) deleterious alleles are very unlikely to fix in an unconnected stable population (Table 1). However, frequent rescue of the ephemeral population reduces the size of the stable population, making deleterious alleles more likely to fix (Figure 2c). As a result, corridor-mediated population rescue and gene flow may lead to qualitatively different effects on fixation within and among

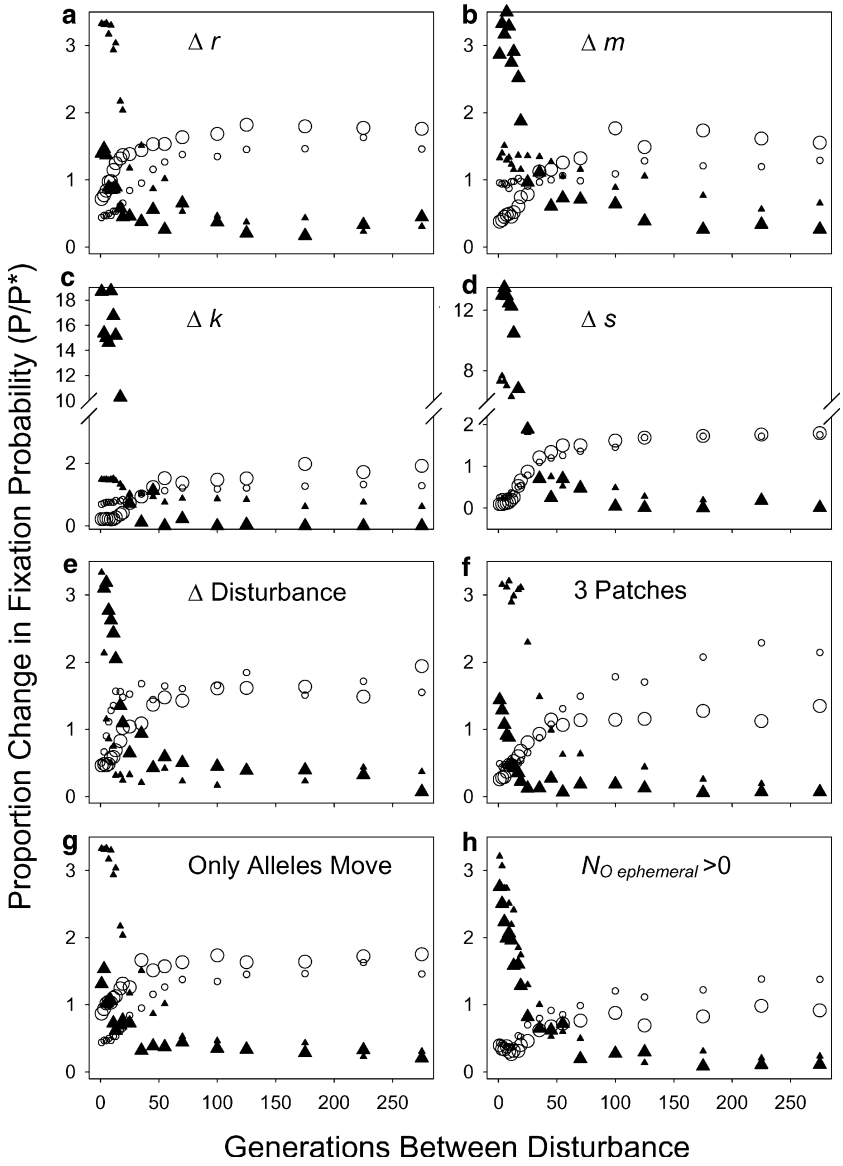

Figure 2. Connectivity-mediated fixation. Unless noted, intrinsic rate of population growth, r, is 0.05, carrying capacity, k, is 500, per capita movement rate, m, is 0.1, and the selective effect of the new allele, s, is ± 0.001. Beneficial alleles are represented by open circles; harmful alleles are represented by black triangles. P/P^* represents how fixation probability in the stable refuge population (P) changes relative to the null value, P^* (Table 1). For example, if P/P* = 2, a new allele is twice as likely to fix relative to the null model. Note that the y-axis may differ among figures. (a) Larger symbols indicate greater rates of population growth ($r = 0.05$ and $r = 0.3$, respectively). (b) Larger symbols indicate greater rates of movement ($m = 0.005$ and $m = 0.5$, respectively). (c) Increased carrying capacity is indicated by increasing symbol size ($k = 200$ and $k = 1250$, respectively). (d) The absolute selective effect of alleles is represented increasing symbol size: larger symbols for $s = -0.002$ and $s = 0.01$, smaller symbols for $s = -0.0015$ and $s = 0.002$. (e) Partial disturbance results loss of either 25% (small symbols) or 75% (large symbols) of the ephemeral population. (f) A stable population connected to two ephemeral populations (small symbols), and a stable population connected to an ephemeral population and a stable population (large symbols). (g) Populations where alleles move but individuals do not (large symbols), compared to the same model where individual and allelic movement are synonymous (small symbols, identical to (a) where $r = 0.05$). (h) Model outcome when there are 125 individuals (small symbols) or 375 individuals (large symbols) in the ephemeral patch when the new allele is introduced and the model begins.

ecological communities, because rate of growth, carrying capacity, and movement ability vary widely among species (Baker 1978; Blueweiss et al. 1978). For example, at high to moderate rates of local extinction in the ephemeral patch, corridors would benefit species with high intrinsic rates of growth (e.g., microbes, invertebrates) because they would be more likely to fix beneficial alleles under scenarios where frequent population rescue occurs (Figure 2). Under the same conditions, corridors could have negative impacts on species with lower rates of growth (e.g., large mammals) because they become more likely to fix deleterious alleles and less likely to fix beneficial alleles.

The effect of corridors on fixation is greater for beneficial alleles of small effect and deleterious alleles of large effect (Figure 2d). Since most new mutations are deleterious and of small effect (Dobzhansky 1970), corridor-mediated fixation dynamics may be most important in affecting the fixation probability of deleterious alleles and their subsequent accumulation in populations (i.e. mutation load (Dobzhansky 1970)). Reduction of deleterious alleles may be especially important for populations of conservation concern because they are often small and thus susceptible to accumulation of mutations (Frankham 1995; Lynch et al. 1995) and inbreeding depression (Frankham 1995). When rates of local extinction are relatively low, corridors could ameliorate these potential hazards to persistence (Figure 2). However, the opposite situation can occur when periodic extinction is frequent enough to greatly reduce overall *Ne*: greater fixation of highly deleterious alleles of large effect could reduce the viability of the connected populations (Figure 2d) and promote mutational meltdown (Lynch et al. 1995).

Additional scenarios

Disturbance may not always be absolute; partial disturbance may lead to a reduction in the size of the ephemeral population without causing local extinction. Partial disturbance dampens the effect of corridors on fixation of both beneficial and deleterious alleles because overall population sizes fluctuate less, exhibiting less population reduction (due to partial disturbance) and less growth (because populations are bounded by carrying capacity) between disturbance events. That is, partial disturbance dampens changes in *Ne*, and fixation is a function of the relative magnitude of change in *Ne* (Otto and Whitlock 1997). Fixation of deleterious alleles is further reduced by partial disturbance (compare Figure 2a and Figure 2e) because the overall *Ne* tends to remain larger, reducing the importance of drift in affecting fixation.

In realistic conservation scenarios, more than two populations may be connected with corridors. When three populations are connected, the subsequent impact on fixation again depends upon how the three-population complex can change *Ne* relative to the original stable population. The potential change in *Ne* depends upon the frequency of disturbance and whether the three-population complex consists of two stable or two ephemeral populations. Larger changes in *Ne* are possible when 66% of the stable–ephemeral complex is subject to disturbance (i.e. when two ephemeral populations exist) than when only 33% is subjected to disturbance (i.e. when two stable populations exist). As such, when a stable refuge population is coupled with an ephemeral population and an additional stable population (Figure 2f), fixation of beneficial and deleterious alleles is reduced relative to the model where a single stable is connected to an ephemeral population (Figure 2a) because the relative magnitude of possible changes in *Ne* is reduced. The opposite occurs when a single stable refuge population is coupled with two ephemeral populations because a wide array of changes in *Ne* is possible: fixation of beneficial alleles is potentially greater, but fixation of deleterious alleles is also potentially greater, with the ultimate outcome contingent upon the frequency of disturbance (Figure 2f). Although the three-population case is presented here, models with greater complexity have been explored by Whitlock (2003).

Gene flow can occur without movement of individuals, as in pollen movement between plants. When gene flow is uncoupled from individual movement, frequent disturbance has a smaller impact on fixation rates of deleterious alleles in the stable–ephemeral complex (Figure 2g). This outcome occurs because the stable refuge population is buffered from demographic losses to the ephemeral population, eliminating the dramatic reductions in *Ne* that occur under frequent disturbance. These results suggest that corridor-mediated fixation may have important implications for plant communities and other organisms where gametes and propagules are mobile but

reproductive individuals are not, including species where non-reproductive juveniles disperse but reproductive adults remain within specified territories.

The fixation dynamics of any particular allele will depend upon when it arises relative to the disturbance regime, because timing of disturbance determines the changes in *Ne* that affect fixation (Figure 1; Otto and Whitlock 1997). For example, Figure 2a–g model fixation of an allele that has just arisen following disturbance (i.e. the ephemeral population is empty at the start of the simulation, $N_{0\ \text{ephemeral}}=0$), a reasonable assumption since at least a few new mutations are likely to arise each generation, even in small populations. However, if the mutation arises sometime between disturbance events, the impact of connectivity and disturbance on fixation is reduced (Figure 2h), because the mutation arises within a population during a time when *Ne* is changing less rapidly than when disturbance has just occurred.

When disturbance occurs with a constant probability, rather than when disturbance is periodic, results are similar: when populations are likely to be disturbed (i.e. probability of disturbance = 0.5), the likelihood of fixation is decreased for beneficial alleles and increased for deleterious alleles is increased (Figure 3). When disturbance is unlikely, fixation of beneficial alleles is increased and fixation of deleterious alleles is decreased. As with the model of periodic disturbance (Figure 2h), the magnitude of this impact is greater when the ephemeral patch starts at zero ($N_{0\ \text{ephemeral}}=0$; Figure 3a). Generally, the models that start with $N_{0\ \text{ephemeral}}=0$ (Figure 2a–g, Figure 3a) and $N_{0\ \text{ephemeral}}=k$ (Figure 3b) serve as endpoints that demonstrate the largest and smallest impacts of disturbance and connectivity on fixation, respectively. Models where $0 < N_{0\ \text{ephemeral}} < k$ (Figure 2h) demonstrate intermediate scenarios. Ultimately, this is simply a reflection of how timing and disturbance influence the context in which a new allele enters the population, i.e. whether a new allele enters while *Ne* is changing rapidly or not at all.

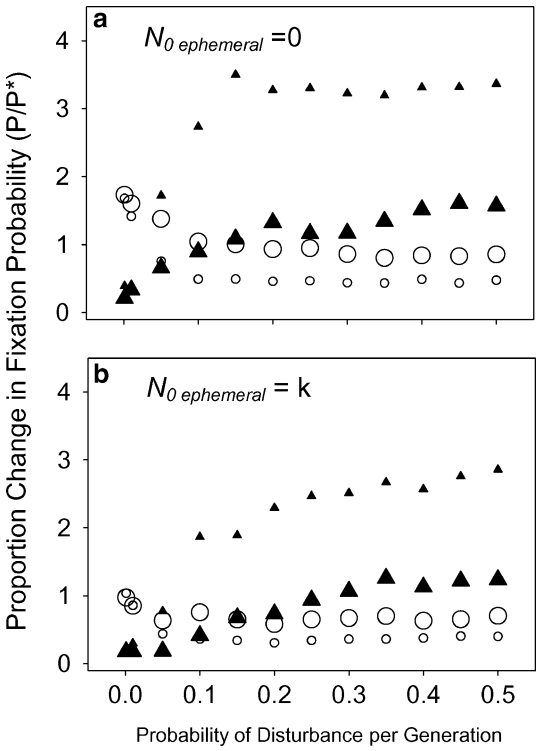

Figure 3. Changes in fixation when disturbance occurs with a constant probability per generation. In both figures, $k=500$ and $m=0.1$. Triangles represent simulations where $s=-0.001$, circles represent simulations where $s=0.001$. Small symbols indicate $r=0.05$, large symbols represent $r=0.3$. Two scenarios are modeled: (a) the ephemeral patch starts empty ($N_{0\ \text{ephemeral}}=0$), as if a mutation had arisen just after a disturbance; (b) the ephemeral patch is full at the start of the simulation ($N_{0\ \text{ephemeral}}=k$), as if a mutation arose when the ephemeral population had not recently experienced disturbance. As in Figure 2, P/P^* represents how fixation probability in the stable refuge population (P) changes relative to the null value, P^* (Table 1).

Conclusions

Corridors are known to increase movement of individuals and alleles among patches (Mech and Hallett 2001; Tewksbury et al. 2002; Haddad et al. 2003), promoting gene flow and population rescue. Corridor impacts on fixation may be either beneficial or harmful: by promoting movement and rescue, corridors can either promote adaptation and persistence, or speed extinction via genetic (Figures 2, 3) and demographic means (Figure 1e). This work suggests that corridors may have evolutionary consequences that are not immediately intuitive, and that the impact of corridors could differ among organisms within the same ecological community. Future work evaluating the role of corridor-mediated fixation in the context of other ecological (e.g. demographic stochasticity) and evolutionary (e.g. genetic hitchhiking, epistasis,

pleiotropy) factors will continue to shed light on the potential impacts of using conservation corridors to connect populations.

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