Ecology, 87(7), 2006, pp. 1844–1855

ON METAPOPULATION RESISTANCE TO DRIFT AND EXTINCTION

LAURENT LEHMANN[1] AND NICOLAS PERRIN[2]

Department of Ecology and Evolution, University of Lausanne, CH 1015 Lausanne, Switzerland

Abstract. The spatial configuration of metapopulations (numbers, sizes, and localization of patches) affects their ability to resist demographic extinction and genetic drift, but sometimes with opposite effects. Small and isolated patches, for instance, contribute marginally to demography but may play a large role in genetics by maintaining a sizeable amount of genetic variance among demes. In source–sink systems, similarly, connectivity may be beneficial in terms of effective size, but detrimental in terms of survival, by lowering the reproductive value of source populations. How to reconcile these opposite effects? Here we propose an analytical framework that integrates fixation time (ability to resist genetic drift) and extinction time (ability to resist demographic extinction) into a single index of resistance, measuring the ability of a metapopulation to maintain its demo-genetic integrity. We then illustrate with numerical examples how conflicting demands may be resolved.

Key words: biodiversity; connectivity; eigenvalue effective size; fragmentation; landscape; matrix analysis; stochastic patch occupancy model (SPOM).

INTRODUCTION

Many natural populations are discontinuous, consisting of series of local demes connected by a level of dispersal that depends on geographic distance and habitat conditions between them. This spatial structure has strong consequences for the demography, genetics, and evolution of populations, and important efforts have been recently devoted to understand the functioning of such structured systems, known as metapopulations (Hanski and Gaggiotti 2004).

These recent advances are most welcome, since human impact on landscapes is presently driving habitat fragmentation at an unprecedented scale. Many populations once large and continuous are now reduced to sets of discrete and poorly connected demes, undergoing high local extinction risks. Fragmentation thus raises a series of important issues for conservation biology. Relevant short-term issues deal with the delineation of the conditions for population survival in a fragmented landscape, and of the sensitivity of such conditions to characteristic features of both the landscape (habitat quality, connectivity, etc) and the species under study (mating system, fecundity, dispersal patterns, etc.). Longer-term issues (evolutionary consequences of fragmentation) are obviously also of importance, including the questions of local adaptation, evolution of dispersal rate, or loss of adaptive potential. It is thus crucial that the problem of fragmentation be considered in terms of both demography and genetics. Fragmentation affects both aspects, and in return both interact to affect population viability prospects. Small and isolated populations suffer from higher genetic drift, accumulate deleterious mutations and loose their potential for adaptation. These genetic effects combine with demographic stochasticity to drive populations into an extinction vortex (Gilpin and Soulé 1986).

The viability prospects of a metapopulation depend in part on landscape features, including mainly the size and quality of patches, as well as their localization, which determines connectivity. Thus, some landscape configurations are obviously better than others. Within a given landscape, similarly, some habitat fragments (patches) are better than others, in the sense that, owing to their size and localization, they contribute more to the survival prospects of the species or to the maintenance of genetic diversity (Gaggiotti and Hanski 2004). A first difficulty in this context is that of properly defining and measuring the quality of a habitat or the value of a patch. A second difficulty stems from the fact that the "value" measured is bound to depend on the currency used (i.e., on the conceptual framework applied). The optimal landscape structure, or the value of a given patch, may differ depending on whether one is interested in minimizing the effects of demographic or environmental stochasticity, of inbreeding load, or of genetic drift, to mention only a few possible objectives. Small and isolated patches may have low value in terms of metapopulation dynamics, because extinction risk is high and colonization probability is low. But they might prove important from a genetic point of view, by maintaining a sizeable amount of genetic variance among demes, or by playing a significant role in the evolutionary fate of species (see, e.g., Wright's shifting-balance theory). It is thus important to adopt as far as

Manuscript received 2 September 2005; revised 9 December 2005; accepted 6 January 2006. Corresponding Editor: A. M. de Roos.

[1] Present address: Department of Genetics, University of Cambridge, Cambridge CB2 3EH, UK.

[2] Corresponding author. E-mail: nicolas.perrin@unil.ch

possible a synthetic approach, and try to properly weight the different threats and prospects.

In the present paper, we propose a metric for evaluating metapopulation quality that combines its ability to resist both demographic extinction (measured by expected time to extinction) and genetic drift (measured by expected time to fixation), and we develop analytical tools aimed at evaluating how biological traits (fecundity, dispersal rate, etc.) and environmental parameters (patch size and localization, environmental stochasticity) affects this measure of metapopulation resistance. Our approach should provide operational ways to answer questions of practical importance in conservation biology, in particular concerning the optimal structure of a landscape for threatened populations.

Outline of the Model

Symbols and notation used throughout the paper are collected in Appendix B.

Characteristic time to extinction

Any finite population is bound to become eventually extinct as a result of random fluctuations in fecundities or mortalities, sudden catastrophes, outbreaks of diseases, competitors, or predators, or any other stochastic changes in the environment. Assuming a population entails an expected extinction risk e per unit time, the probability of not being extinct at time t is given by the following recurrence equation:

$$p_{t+1} = (1 - e)p_t. \tag{1}$$

The population's capacity to resist extinction can be measured by its time to extinction T_{ex} (MacArthur and Wilson 1967, Richter-Dyn and Goel 1972, Gabriel and Burger 1992, Lande 1993), obtained as the inverse of the extinction probability per generation:

$$\frac{1}{T_{\text{ex}}} = \frac{p_t - p_{t+1}}{p_t} = e. \tag{2}$$

If the population fluctuates among several states that differ in extinction rate, then extinction time can be calculated from the matrix $\mathbf{P}$ describing the transition probabilities between the demographic states of the population (e.g., MacArthur and Wilson 1967, Gabriel and Bürger 1992; Appendix C):

$$\mathbf{p}_{t+1} = \mathbf{P}\mathbf{p}_t \tag{3}$$

where $\mathbf{p}_t$ is a vector collecting the probabilities of being in the different states at time t. Conditional to non-extinction, this vector converges toward the so-called quasi-stationary distribution (Grimm and Wissel 2004), provided by the right eigenvector associated to the leading non-unit eigenvalue of $\mathbf{P}$. This eigenvalue ($\lambda_{\mathbf{P}_o}$) measures the asymptotic probability of not becoming extinct on the next time step, once the population has reached the quasi-stationary distribution. From this eigenvalue, the characteristic time to extinction, measuring the life expectancy of a population drawn from the quasi-stationary distribution, is calculated as follows (Halley and Iwasa 1998, Ovaskainen and Hanski 2004 [Box 4.2]):

$$\frac{1}{T_{\text{ex}}} = 1 - \lambda_{\mathbf{P}_o}. \tag{4}$$

If $\lambda_{\mathbf{P}_o} = 1$, the population survives indefinitely (the characteristic time to extinction is infinite). The lower $\lambda_{\mathbf{P}_o}$, the faster extinction occurs.

This approach is readily extendable to metapopulations by canonical analysis of the matrix describing the transition probabilities between the possible states of the metapopulation. However, because such analysis is complex, metapopulation dynamics has often been studied under deterministic settings, following the influential inroads of Levins (1969) (e.g., Lande 1987, Hanski and Ovaskainen 2000, 2003). In such settings, metapopulations can avoid deterministic extinction and survive indefinitely, provided connectivity exceeds a threshold set by local extinction rates and landscape features. Analysis in realistic spatial settings (Hanski and Ovaskainen 2000, 2003, Ovaskainen and Hanski 2001, 2004) shows that the size and localization of local patches affect their individual contribution to metapopulation survival. Under source-sink dynamics, however, connectivity might become less advantageous, since sinks behave as ecological traps (Gundersen et al. 2001).

Effective size and characteristic time to fixation

In absence of mutation, any finite population ultimately becomes genetically homogeneous as a result of genetic drift (i.e., random sampling in the contribution of individuals to the gene pool). Assuming a deme of constant size N and ideal settings (including non-overlapping generations and Poisson distribution of fecundities), two genes stem from the same parental gene with probability $1/N$ ($1/2N$ for diploid monoecious organisms). Thus, the expected variance (measured at any generation t as the probability h_t that two randomly sampled alleles differ) disappears at a geometric rate:

$$h_{t+1} = \left(1 - \frac{1}{N}\right)h_t. \tag{5}$$

If settings differ from ideal (e.g., dioecious individuals, population subdivision, age classes), then Eq. 5 allows defining the effective size (N_e) of the population, obtained as the inverse of the proportion of variance lost per generation:

$$\frac{1}{N_e} = \frac{h_t - h_{t+1}}{h_t}. \tag{6}$$

Since the right-hand side of Eq. 6 represents a rate, effective size actually measures a time, which can be thought of either as a coalescence time (expected number of passed generations before the two gene lineages coalesce in a common ancestor) or a fixation time. Indeed, as developed in Appendix C, the rate of maintenance of genetic diversity in an ideal population ($1 - 1/N$ in Eq. 5) is in fact equivalent to the leading

non-unit eigenvalue ($\lambda_{\mathbf{G}}$) of the matrix describing the transitions between all the genetic states of the population (i.e., sets of allele frequencies). Effective size can therefore be interpreted as a characteristic time to fixation, i.e., the expected number of generations until genetic variance is entirely lost, given that the initial state of the population (in terms of allele frequencies) is drawn from its quasi-stationary distribution.

Importantly, the eigenvalue $\lambda_{\mathbf{G}}$ can be directly evaluated from the dynamics of pairs of genes, because asymptotic changes of the genetic states of the population are equivalently given by asymptotic changes of probabilities of identity between pairs of genes (Ewens 1979, Whitlock and Barton 1997, Rousset 2004). Let the matrix $\mathbf{G}$ describe the per generation changes in the probabilities that pairs of genes differ:

$$\mathbf{h}_{t+1} = \mathbf{G}\mathbf{h}_t \tag{7}$$

where $\mathbf{h}_t$ is a vector collecting the probabilities that two genes sampled in various locations (e.g., different patches or age classes) differ at time t. The leading eigenvalue of $\mathbf{G}$ allows calculating the eigenvalue effective size (Hill 1972, Ewens 1979, Rousset 2004):

$$\frac{1}{N_e} = 1 - \lambda_{\mathbf{G}} \tag{8}$$

which establishes the asymptotic rate of convergence to fixation. If $\lambda_{\mathbf{G}} = 1$, variance is kept indefinitely (the characteristic time to fixation is infinite). The lower $\lambda_{\mathbf{G}}$, the more rapidly variance is lost. Note that effective size is defined conditional to non-extinction (the focal population displays effective size N_e as long as the patch is occupied).

Population structure has the potential to impact effective size. Under finite-island model assumptions (n_d demes of equal and constant size and productivity, connected by homogeneous dispersal), population structure is expected to boost effective size by maintaining a substantial amount of variance among demes, out of reach of drift (Wright 1931). At the limit (no dispersal, no local extinction), effective size tends to infinity.

However, island-model assumptions are often untenable. Real demes fluctuate, owing to demographic and environmental stochasticity, they differ in size, productivity, and localization, which affects their extinction and recolonization rates. Several extensions of Wright's pioneering work have been proposed to account for the disequilibrium dynamics of metapopulations (e.g., Whitlock and Barton 1997, Nunney 1999, Pannel and Charlesworth 1999, Iizuka 2001, Rousset 2003; see review in Wang and Caballero 1999). Whitlock and Barton (1997) in particular provided an extensive analysis of the effects of population structure on Ewens' eigenvalue effective size. Their approach allows disentangling the effects due to within-deme variance in individual reproductive success, from those due to among-demes variance in contribution to migrant pool and metapopulation dynamics. It turns out that population structure decreases N_e as soon as the among-demes variance in reproductive output exceeds the value expected from a Poisson distribution with parameter constant among demes. Thus, when patches are allowed to vary in productivity and contribution to the migrant pool (source–sink dynamics or extinction–recolonization events), population structure has the potential to drastically lower fixation time.

Combining extinction and fixation time

The concepts of extinction time and fixation time are both highly relevant for conservation biology, which aims at developing tools to limit simultaneously extinction risks and genetic drift. However, as already noted, genetics and demography may conflict over important issues. Small and isolated patches, for instance, may have positive effect on fixation time, but be of low demographic value. In the case of source–sink systems, similarly, connectivity might be detrimental in terms of demography, but beneficial in terms of genetics. How to properly weight the demographic and genetic benefits of connectivity? As outlined below, the concepts of extinction time and fixation time can be put in a common framework, which allows investigating their joint effect on the ability of a system to maintain its integrity.

Under ideal settings, the two equations, Eq. 1 and Eq. 5 can be combined into a single recurrence equation:

$$d_{t+1} = (1 - e)\left(1 - \frac{1}{N}\right)d_t \tag{9}$$

where $d_t \equiv p_t h_t$ is an index of diversity measuring the genetic variance expected at time t conditional to non-extinction, weighted by the probability of non-extinction.

Under more complex demography, demo-genetic transition probabilities can be collected into a matrix $\mathbf{T}$ (see Appendix A), which drives the dynamic of demo-genetic states:

$$\mathbf{d}_{t+1} = \mathbf{T}\mathbf{d}_t. \tag{10}$$

The leading eigenvalue ($\lambda_{\mathbf{T}}$) of $\mathbf{T}$ provides a global index of resistance to genetic drift and extinction. If $\lambda_{\mathbf{T}} = 1$, then the population will keep indefinitely its integrity (measured as survival probability times genetic variance). The lower $\lambda_{\mathbf{T}}$, the quicker integrity is lost. As shown in Appendix A, this demo-genetic eigenvalue turns out to be the simple product of the demographic and genetic eigenvalues:

$$\lambda_{\mathbf{T}} = \lambda_{\mathbf{P}_o}\lambda_{\mathbf{G}} \tag{11}$$

which thus combine in a simple way. The rate of loss of diversity is given by the complement to unity of $\lambda_{\mathbf{T}}$,

$$\frac{1}{T} = 1 - \lambda_{\mathbf{T}} \tag{12}$$

and can be expressed in terms of extinction- and fixation rates as follows:

$$\frac{1}{T} = \frac{1}{T_{ex}} + \left(1 - \frac{1}{T_{ex}}\right)\frac{1}{N_e}. \quad (13)$$

Eq. 13 receives the intuitive interpretation that, once in the quasi-stationary distribution, a population can loose diversity either by becoming extinct (at rate $1/T_{ex}$), or, if not extinct ($1 - 1/T_{ex}$), by fixing alleles (at rate $1/N_e$). This rate of fixation ($1/N_e$) can be intuitively understood as an average rate over the possible states of the population, weighted by their probabilities of occurrence (derived from the quasi-stationary distribution). Note that N_e is necessarily defined conditional to non-extinction, and that Eq. 11 provides a way to infer it that will prove useful when the dynamics of genes depends on complex demographic processes.

Our Appendix A provides a mathematical formalization of the above results for a structured metapopulation made of n_d demes of variable sizes undergoing local extinctions and recolonizations. Here we propose numerical examples that will help grasping the practical relevance of our approach, before discussing the concepts and results presented here.

Numerical Examples

The examples below are not aimed at investigating realistic situations, but at illustrating our approach: the conceptual relationships among eigenvalues and their interpretation will be better understood under simple situations. Accordingly, we first address a situation with a single deme, which can reside in two non-extinct states, so that corresponding matrices **P** and **T** have dimension 2×2 only. We then consider a system of three demes simplified to a stochastic patch occupancy model (SPOM), where each deme can take either of the two possible values: empty or occupied at a patch-specific density value N_i. Hence, the size of vector **p** reduces to 2^{n_d} (i.e., 8 in our case), and that of vector **d** to $n_d^2(2^n - 1)$ (i.e., 63 in our case). These examples were implemented in Mathematica 5.1 (Wolfram Research, Champaign, Illinois, USA), and the worksheets are available in the Supplement.

One deme, three states

As a first illustration, we assume a single deme that can reach three demographic states (Fig. 1); it can be at high density (N_h), at low density (N_l), or extinct ($N = 0$). The non-empty states become extinct with probabilities e_h and e_l, respectively (including both environmental and demographic extinction), and, conditional to non-extinction, stay in the same state with probabilities β and α, respectively. The dynamics of the transient states is thus governed by the transition matrix

$$\mathbf{P}_o \equiv \begin{bmatrix} \alpha(1-e_l) & (1-\beta)(1-e_h) \\ (1-\alpha)(1-e_l) & \beta(1-e_h) \end{bmatrix} \quad (14)$$

and the demo-genetic matrix becomes the following:

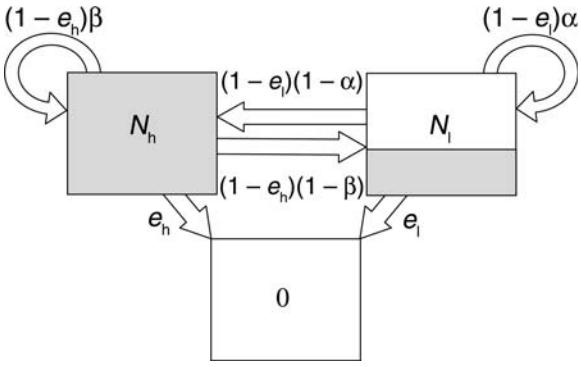

FIG. 1. The focal population can take three states: high density (N_h), low density (N_l), or extinct (0). When at low density, the population goes extinct with probability e_l and, conditional to non-extinction, remains at low density with probability α. When at high density, it goes extinct with probability e_h and, conditional to non-extinction, remains at high density with probability β.

$$\mathbf{T} \equiv \begin{bmatrix} \alpha\,(1-e_l)\left(1-\frac{1}{N_l}\right) & (1-\beta)(1-e_h)\left(1-\frac{1}{N_h}\right) \\ (1-\alpha)(1-e_l)\left(1-\frac{1}{N_l}\right) & \beta(1-e_h)\left(1-\frac{1}{N_h}\right) \end{bmatrix}. \quad (15)$$

The leading eigenvalues of $\mathbf{P}_o$ and **T** measure the ability of the focal population to resist demographic and demo-genetic drift, respectively. Effective size can be calculated from these values using Eqs. 8 and 11. For the purpose of illustration, eigenvalues can be analytically derived from Eqs. 14 and 15 as

$$\lambda_{\mathbf{P}_o} = \frac{1}{2}\left\{x + [x^2 + 4(1-\alpha-\beta)(1-e_l)(1-e_h)]^{\frac{1}{2}}\right\} \quad (16)$$

where $x = \alpha(1 - e_l) + \beta(1 - e_h)$, and

$$\lambda_T = \frac{1}{2N_hN_l}\{y + [y^2 + 4(1-\alpha-\beta)(1-e_h) \times (1-e_l)N_h(N_h-1)N_l(N_l-1)]^{\frac{1}{2}}\} \quad (17)$$

where $y = \alpha(1 - e_l)N_h(N_l - 1) + \beta(1 - e_h)N_l(N_h - 1)$; $\lambda_{\mathbf{G}}$ (and hence effective size) is readily obtained as the ratio of these two quantities.

Genetics and demography are intimately intertwined in the demo-genetic matrix **T**, because effective size depends on quasi-stationary distribution, itself a function of state-specific extinction rates. The two can only be disentangled when extinction risk is made independent of the state of the population. For instance, assuming $e_l = e_h = e$ and $\alpha + \beta = 1$ (so that population fluctuations are uncorrelated; Iizuka 2001), then Eq. 16 simplifies to

$$\lambda_{\mathbf{P}_o} = 1 - e \quad (18)$$

and Eq. 17 to

$$\lambda_{\mathbf{T}} = (1-e)\left(1 - \frac{\alpha}{N_h} - \frac{1-\alpha}{N_l}\right). \quad (19)$$

Hence, $\lambda_{\mathbf{G}}$ is readily extracted as follows:

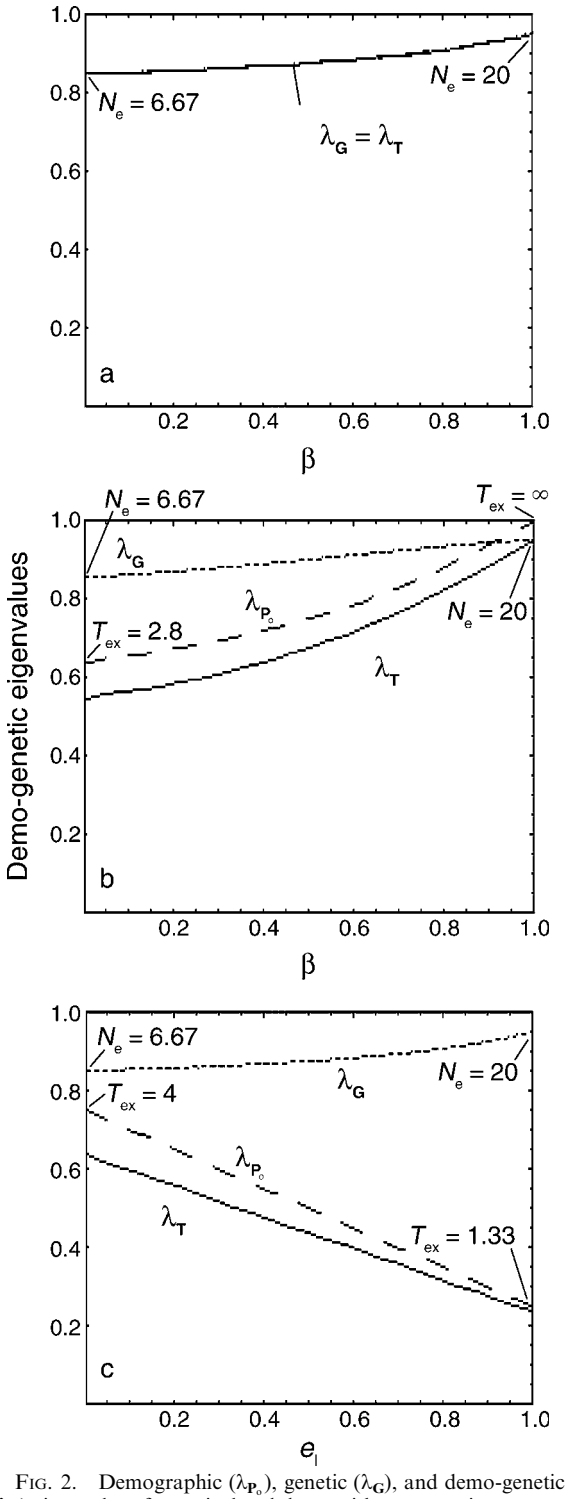

FIG. 2. Demographic ($\lambda_{\mathbf{P}_o}$), genetic ($\lambda_{\mathbf{G}}$), and demo-genetic ($\lambda_{\mathbf{T}}$) eigenvalues for an isolated deme with two transient states. Parameter values are fixed to $N_l = 5$, $N_h = 20$, and $\alpha = 0.5$. (a) In the absence of extinction ($e_l = e_h = 0$), $\lambda_{\mathbf{P}_o}$ reaches unity, so that $\lambda_{\mathbf{T}} = \lambda_{\mathbf{G}}$ (solid line), and $\lambda_{\mathbf{G}}$ increases with β (probability to stay in the high-density state). (b) If extinction occurs only at low density ($e_h = 0$, $e_l = 0.5$), then both $\lambda_{\mathbf{G}}$ (dotted line) and $\lambda_{\mathbf{P}_o}$ (dashed line) increase with β. (c) Note that $\lambda_{\mathbf{G}}$ (dotted line) and $\lambda_{\mathbf{P}_o}$ (dashed line) respond in opposite ways to an increase in the extinction risk at low density (e_l). Parameters β and e_h are fixed at 0.5.

$$\lambda_{\mathbf{G}} = \frac{\lambda_{\mathbf{T}}}{\lambda_{\mathbf{P}_o}} = 1 - \frac{\alpha}{N_h} - \frac{1-\alpha}{N_l}. \tag{20}$$

As expected (Karlin 1968, Iizuka 2001), the effective size correspond to the harmonic mean of population size over time, since α and 1 − α represent the proportion of time spent in state h and l respectively, conditional to non-extinction (i.e., the quasi-stationary distribution).

In absence of extinction ($e_l = e_h = 0$), $\lambda_{\mathbf{P}_o}$ reaches unity (i.e., extinction time is infinite), so that $\lambda_{\mathbf{T}} = \lambda_{\mathbf{G}}$ (Fig. 2a). Effective size is affected by transition rates between states, in accordance with the proportion of time spent at low versus high density (here fixed to $N_l = 5$ and $N_h = 20$). The effect of extinction is illustrated in Fig. 2b, where α is fixed to 0.5, and β varies from 0 to 1. If extinction affects only the small state (e_h is here fixed to 0 and e_l to 0.5), then both demographic and genetic prospects improve as β increases, from $T_{ex} \approx 2.78$ and $N_e \approx 6.67$ when $\beta = 0$ (the population then has then a 2/3 chance of being in the small and extinction-prone state) to $\lambda_{\mathbf{P}_o} = 1$ (i.e., T_{ex} is infinite) and $N_e = 20$ when $\beta = 1$ (the population is then always in the large and extinction-proof state).

By contrast, demographic and genetic prospects show opposite responses to changes in the extinction risk of the low-density state (Fig. 2c). As e_l increases, the population becomes increasingly prone to extinction, but also more likely to reside in the high-density state (conditional to non-extinction), so that effective size increases. This potential conflict is dominated by demography: the overall prospects of the population are maximized by limiting the extinction rate of the low-density state, even though effective size is thereby decreased.

Three demes, two states each

As a second situation, we consider the case of three demes that may differ in size, connectivity, and extinction risk (Fig. 3). Under SPOM simplifications, a non-extinct deme takes only one possible value (which may be thought of as the harmonic mean of its size over time). State transitions are obtained as the product over demes of the relevant transition probabilities, given state $\mathbf{n}$:

$$\Pr_f(\mathbf{n}'|\mathbf{n}) = \prod_{i=1}^{n_d} \Pr_{f_i}(0|\mathbf{n})^{1-o_i'} [1 - \Pr_{f_i}(0|\mathbf{n})]^{o_i'} \tag{21}$$

where o_i' is the indicator function, equal to 1 if patch i is occupied in the descendant generation (when the metapopulation is in state $\mathbf{n}'$), and 0 otherwise. The probability that deme i gets extinct at time $t + 1$, given state $\mathbf{n}$ at time t, is obtained by

$$\Pr_{f_i}(0|\mathbf{n}) = e_i + (1 - e_i)e^{-q_i[\mathbf{n}]} \tag{22}$$

where $q_i[\mathbf{n}] = \sum_{j=1}^{n_d} bN_j m_{ij} o_j$ sums up the expected contributions of all demes to the focal deme i, b is the per capita fecundity, assumed here constant and identical among patches, and m_{ij} the forward dispersal rate from deme j to deme i. In our numerical analysis, all m_{ii} were set to $1 - m$, and all m_{ij} to $m/2$ (under more

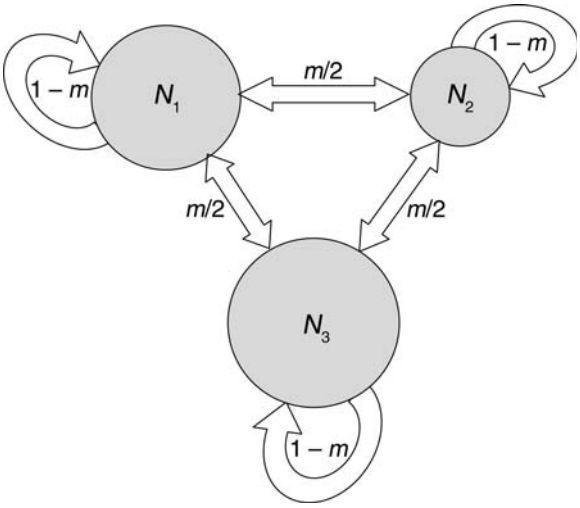

FIG. 3. Model metapopulation made of three demes of sizes N_1, N_2, and N_3 that can be either occupied or empty (stochastic patch occupancy model [SPOM]). The rate of philopatry is $1 - m$, and dispersers reach either of the two other populations randomly.

realistic settings, dispersal rates would be made dependent on the distance between pairs of patches). The parameter e_i represents environmental extinction risk, and $e^{-q_i[\mathbf{n}]}$ demographic extinction risk (probability of sampling zero from a Poisson distribution with parameter $q_i[\mathbf{n}]$).

The effects of demographic stochasticity are illustrated in Fig. 4a–c, where environmental extinction risks are set to 0, fecundity to 1.1, and all three demes have the same size N when non-extinct. In absence of dispersal, N_e and T_{ex} converge to similar values, because all populations behave independently (both demographic extinction and coalescence scale as $1/N$). For the lowest population sizes ($N = 1$, Fig. 4a) these values are close to 3 (though exact numbers are bound to depend on fecundity), so that the global resistance of the metapopulation is quite weak ($\lambda_{\mathbf{T}} < 0.5$). Demographic stochasticity decreases rapidly as demes increase in size, and so does the overall risk for the metapopulation (Fig. 4b and 4c with $N = 2$ and 5, respectively). As can be seen, global extinction risk becomes negligible with three populations of five individuals each, so that the loss of genetic variance becomes the predominant concern.

Demography and genetics show contrasted responses to dispersal in the situations illustrated here (Fig. 4a–c). T_{ex} increases with dispersal owing to rescue effects (once extinct, a population might be recolonized), so that the population survival is maximized by panmixis (corresponding to $m = 2/3$ for $n_d = 3$). But, as expected (Wright 1931), N_e is maximized by philopatry, owing to the maintenance of interdemic genetic variance. With local demes of size $N = 5$ (Fig. 4c), stochastic demography is too low to induce significant extinction risks. Effective size thus tends to infinite at zero dispersal (the among-deme component of genetic variance is kept indefinitely out of reach of drift), but rapidly declines to 15 (sum of the three census sizes) as dispersal increases and the system becomes panmictic. This potential conflict is dominated by genetics in that case, because $\lambda_{\mathbf{G}} < \lambda_{\mathbf{P}_0}$. Hence, in absence of environmental extinction risk, population integrity is best maintained by preventing connectivity.

Dispersal, however, becomes globally favorable as soon as environmental stochasticity is introduced (Fig. 4d): owing to higher local extinction risks, philopatry is then unable to maintain a significant amount of interdemic genetic variance. Dispersal, by contrast, increases through rescue effects the probability that several demes are occupied simultaneously (and hence both N_e and T_{ex}).

Potential conflicts may emerge when populations differ in size and/or extinction rates. In Fig. 4e, we assume three demes of identical sizes ($N = 5$) but among which only one is safe from environmental extinction ($e_1 = e_2 = 0.5$, $e_3 = 0.0$). In absence of dispersal, the two first populations go rapidly extinct, so that $N_e = 5$ (corresponding to the size of the remaining deme). T_{ex} is maximal, however, though not infinite because the safe deme still incurs a slight extinction risk from demographic stochasticity. Increasing dispersal has a negative impact on demography (because offspring emigration from the safe deme increases its risk of demographic extinction) but a positive impact on effective size (because rescue effects increase the probability that several demes are simultaneously occupied). It is thus worth noting that dispersal increases the effective size of metapopulations under the kind of source–sink dynamics modeled here (contrasting with the negative effect documented under stable settings; Fig. 4a–c). As genetics is of greater concern under our parameter values (global extinction is anyway unlikely), this conflict is dominated by genetics, and the dispersal value that best maintains population integrity is close to panmixis (Fig. 4e, arrow).

Let us finally assume that the three demes differ in their susceptibilities to demographic and environmental extinctions (Fig. 4f). Deme 1 is protected against both risks ($e_1 = 0$, $N_1 = 5$), deme 2 incurs a large risk of environmental extinction ($e_2 = 0.5$, $N_2 = 5$), and deme 3 a large risk from demographic stochasticity ($e_3 = 0$, $N_3 = 2$). Global extinction risk is then weak, and increases slightly with connectivity (through increased risks of demographic extinction of the environmentally safe patches). Potential problems stem thus mostly from the low effective size, which displays a non-monotonous response to dispersal. Two local maxima emerge, one corresponding to complete philopatry, and the other to complete dispersal. N_e increases with connectivity over most of its range for the aforementioned reason (low dispersal limits rescue effects and thus lowers metapopulation size). At very low dispersal value, however, effective size is boosted because, though patch 2 goes rapidly extinct from environmental causes, the system maintains some among-deme variance thanks to its two

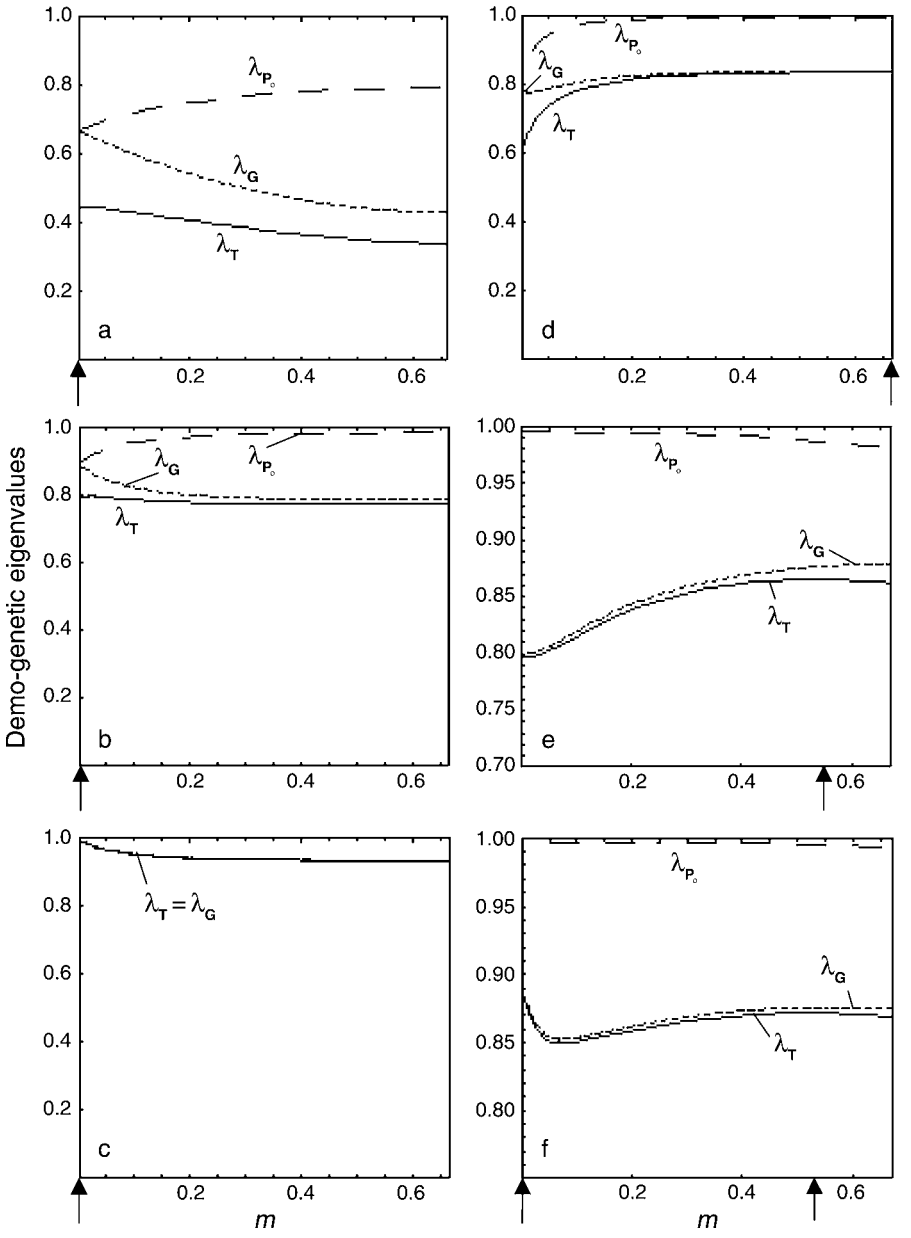

FIG. 4. Demographic ($\lambda_{\mathbf{P}_0}$), genetic ($\lambda_{\mathbf{G}}$), and demo-genetic ($\lambda_{\mathbf{T}}$) eigenvalues for a metapopulation (three demes of varying sizes and extinction rates) as functions of dispersal rate (m). Arrows indicate the dispersal rate maximizing the overall demo-genetic resistance. (a–c) In the absence of environmental stochasticity, demographic and genetic eigenvalues respond differently to connectivity (m): $\lambda_{\mathbf{P}_0}$ (and hence extinction time) increases, but $\lambda_{\mathbf{G}}$ (and hence fixation time) decreases with increasing dispersal. Both values increase with the size of local demes, from (a) $N_i = 1$ to (b) $N_i = 2$ to (c) $N_i = 5$. (d) Both $\lambda_{\mathbf{P}_0}$ and $\lambda_{\mathbf{G}}$ increase with connectivity when the risk of environmental extinction is significant. N_i values are fixed to 5, and e_i values to 0.1. (e–f) . Complex patterns emerge when demes differ in demographic and environmental risks of extinction. Dispersal may then have opposite effects on extinction and fixation time (e: $N_1 = N_2 = N_3 = 5$, $e_1 = e_2 = 0.5$, $e_3 = 0$) or display multiple optima (f: $N_1 = N_2 = 5$, $N_3 = 2$, $e_1 = 0.5$, $e_2 = e_3 = 0$).

environmentally safe demes. Note, however, that this variance is not kept indefinitely, since these demes (and particularly deme 3) incur a significant risk of extinction from demographic stochasticity). Under our parameter values, the global optimum corresponds to complete philopatry (thus reconciling demands from demography and genetics), but this optimum might suddenly shift toward nearly complete connectivity under very slight changes in parameter values.

DISCUSSION

Large efforts have been recently devoted to investigate the demographic and genetic consequences of population structure (Hanski and Gaggiotti 2004). In partic-

ular, the effects of disequilibrium demography (demographic and environmental stochasticity, extinction–colonization or source–sink dynamics, etc.) on the effective size of metapopulations have received extensive treatments (e.g., Whitlock and Barton 1997, Pannell and Charlesworth 1999, Wang and Caballero 1999, Iizuka 2001, Laporte and Charlesworth 2002, Rousset 2003, 2004). As underlined by the present analysis, genetics and demography are indeed intimately intertwined, since eigenvalue effective size depends on quasi-stationary distribution, itself a function of state-specific and patch-specific extinction rates, demographic transition, and dispersal patterns.

Our results relate to previous treatments on several aspects. In line with Karlin (1968) and Iizuka (2001) we find that, when population size varies temporally, the harmonic mean of census size does correctly measure effective size, provided fluctuations are not correlated. Our own formulation actually differs slightly from Karlin (1968) and Iizuka (2001) in that we incorporate the possibility that the focal population becomes extinct. If the several states present different extinction rates, then the quasi-stationary distribution will be affected, and so will in turn be effective size. From our results, enhancing extinction rate might either increase or decrease effective size, depending on whether extinction affects low-density or high-density states, respectively. Note that an increase in effective size with extinction rate (Fig. 2c) presents no paradox, since this quantity is defined conditional to non-extinction.

In the case of several connected populations, we also find, in line with previous treatments (e.g., Whitlock and Barton 1997), that the consequences of structure depend on the among-deme variance in reproductive output. In particular, structure increases effective size as long as stochasticity remains only demographic (Fig. 4a–c), but decreases it as soon as environmental extinctions induce a large variance in demic reproductive outputs (Fig. 4d–e). In the examples chosen, the negative effect of environmental extinction on effective size is further boosted by the autocorrelation in patch-specific extinction rates. As a result, dispersal might have positive or negative impacts on effective size, depending on the source of stochasticity. Interestingly, non-monotonous relations with multiple local maxima may also arise, depending on specific patterns of demographic vs. environmental risk (Fig. 4f).

But the main interest and originality of the present approach lies in integrating demographic and genetic aspects into a unified analytical framework, which allows delineating how these aspects combine or oppose each other. Indeed, as our numerical examples clearly show, demography and genetics may impose conflicting demands on optimal landscape designs. Depending on demographic and environmental stochasticity, for instance, connectivity may increase or decrease genetic resistance (fixation time), and increase or decrease demographic resistance (extinction time). Furthermore, both indices may respond in similar or in opposite ways. Our approach allows exploring analytically these conflicts, and the synthetic index of demo-genetic resistance offers a common currency that permits weighting demands and solving conflicts in a natural way. Indeed, because the two eigenvalues associated to demographic and genetic matrices combine in a multiplicative way ($\lambda_{\mathbf{T}} = \lambda_{\mathbf{P}_o}\ \lambda_{\mathbf{G}}$), the sensitivity of $\lambda_{\mathbf{T}}$ to a change in $\lambda_{\mathbf{G}}$ is proportional to the value of $\lambda_{\mathbf{P}_o}$, and vice versa. Hence, $\lambda_{\mathbf{T}}$ responds more to the lowest of the two components (the one presenting the highest risk).

Genetics will take the leading role as soon as extinction risk is light relative to the risk of loosing genetic diversity. Management options are thus bound to differ from those based on demography only. While large and well-connected demes are of crucial importance when it comes to avoid extinction and maximize metapopulation capacity (Hanski and Ovaskainen 2000, 2003), small and isolated patches are likely to gain importance if genetic aspects are considered (provided global extinction risk is limited), because they potentially constitute reservoirs of interdemic genetic variance preserved for some time from the action of drift (e.g., Margan et al. 1998).

Our approach assumes that genetic variance is beneficial per se, but of course does not address directly the important question of the viability benefits of having a large effective size. Though this is still a controversial and speculative issue, the overall benefits of having a large effective size is supported by empirical data (see, e.g., the meta-analysis of Reed and Frankham [2003]) together with several lines of arguments. First, the ability to maintain diversity in general (i.e., over the whole genome) should increase a population's ability to respond adaptively to environmental changes (e.g., Nunney 1999), which might become a crucial issue with the raise of global changes. Effective size might hence represent a surrogate for adaptiveness. Secondly, a large effective size should efficiently counteract drift load. Indeed, small populations tend to accumulate mildly deleterious mutations, resulting in a progressive loss of fitness, which may eventually result in mutational meltdowns (Lynch and Gabriel 1990, Lynch et al. 1995). It turns out that the effect of population structure on drift load depends on whether structure increases or decreases effective size (Whitlock 2004).

By contrast, structure should consistently favor such accumulation at a local scale ("local drift load"). Were the within-deme component of genetic variance more important for fitness than the among-deme, then effective size would not be the best target, since it does not distinguish among these components. Management should, in such a case, aim at maximizing the observed (rather than expected) heterozygosity, which might be obtained by favoring connectivity in order to fully benefit from heterosis. Further formalization would obviously be required to integrate these alternative genetic aspects to the present approach. However,

analysis tends to show that both within-deme and total-metapopulation genetic diversity are similarly affected by important aspects of metapopulation dynamics (Pannell and Charlesworth 1999). It also turns out that all forms of genetic load are affected by the total amount of variance (together with its apportionment), since the effects of population structure on load can generally be described as functions of N_e and F_{ST} (Whitlock 2004). Hence, effective size is anyway bound to contribute directly to any indicator of metapopulation quality.

We think our approach might prove useful for conservation biology, in addressing questions related to the optimal design of protected habitat patches (SLOSS-type debates), as practitioners begin to realize that the maintenance of genetic diversity in natural populations should also constitute an important goal for conservation (e.g., Garner et al. 2005). Our approach may, in theory, address any realistic situation and incorporate any detailed feature of the landscape and species under scrutiny, provided relevant information (e.g., density-dependent patterns of fecundity and survival, dispersal kernels, etc.) is available to calibrate the model. Actually, the limitations are mostly of practical nature, and set by the huge sizes of the matrices involved. Implementation at a large scale (landscape) will thus require very large computer power, or procedures to simplify and reduce in some way the sizes of transition matrices.

It is also worth noting that our approach might readily be extended to metacommunity issues (Loreau et al. 2003). The neutral theory of the niche (Hubbell 2001) considers the dynamics of competing species within communities, in complete analogy with the neutral theory of evolution, which considers the dynamics of competing alleles within populations. Biodiversity will be best maintained by maximizing, not only the extinction time of local communities, but also their fixation time (i.e., minimizing the rate of loss of constituting species). When designing optimal metacommunity structures, trade-off and conflicts between these goals are bound to emerge, that the approach delineated here might help solving.

Acknowledgments

This work benefited from inputs from J. Goudet, O. Ovaskainen, and F. Rousset, as well as discussions from participants to the "Ecole buissonnière 2005": L. Braendli, T. Broquet, F. Guillaume, A. Hirzel, and J. Jaquiéry. This project was funded by the Swiss National Science Foundation (3100A0-108100).

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APPENDIX A: ANALYTICAL DEVELOPMENTS

We consider a landscape described by the sizes and localizations of a series of n_d local patches affected by demographic events and linked by juvenile dispersal. Probabilities of non-identity of pairs of genes are measured here among adults after dispersal, but similar developments might be made for pre-dispersal measurements. Our model is similar to Rousset (2004) except that we include the possibility of metapopulation extinction.

Fixation time

Let us first assume that the n_d demes comprise stable numbers of individuals N_1, N_2, N_3, etc. Genetic diversity is described by the vector $\mathbf{h}_t$ (dimension n_d^2) of pairwise heterozygosities (h_{ik} is the probability that two alleles randomly sampled from demes i and k are different). The dynamics of diversity depends on both dispersal patterns and coalescence events. The $n_d \times n_d$ backward dispersal matrix $\mathbf{F} = |f_{ij}|$ describes the probabilities that an adult sampled in i originates from j:

$$\mathbf{F} = \begin{pmatrix} f_{11} & f_{12} & \cdots \\ f_{21} & f_2 & \cdots \\ \cdots & \cdots & \cdots \end{pmatrix}. \tag{A.1}$$

As the lines of $\mathbf{F}$ sum to 1, its leading eigenvalue equals unity, and the corresponding left eigenvector measures patch reproductive values v_i. The probabilities that two adults, sampled in i and j, stem for k and l respectively, are obtained from the tensor product of the matrix $\mathbf{F}$ by itself ($\mathbf{A} = \mathbf{F} \otimes \mathbf{F}$):

$$\mathbf{A} = \begin{pmatrix} f_{11}f_{11} & f_{11}f_{12} & \cdots \\ f_{11}f_{21} & f_{11}f_{22} & \cdots \\ \cdots & \cdots & \cdots \end{pmatrix}. \tag{A.2}$$

The rows of matrix $\mathbf{A}$, which describes the backward movement of pairs of genes, sum up to 1, so that its leading eigenvalue also equals unity. The corresponding left eigenvector measures pairwise products of patch reproductive values $v_i v_j$. The matrix of genetic transitions $\mathbf{G} = |g_{ij,kl}|$ is then obtained by multiplying each entry of $\mathbf{A}$ by the corresponding probability of non-coalescence (probability that the two individuals considered are not born to the same parent), equal to $1 - 1/N_k$ if the two individuals originate from the same deme ($k = l$), and 1 otherwise:

$$\mathbf{G} = \begin{bmatrix} f_{11}f_{11}\left(1 - \dfrac{1}{N_1}\right) & f_{11}f_{12} & \cdots \\ f_{11}f_{21}\left(1 - \dfrac{1}{N_1}\right) & f_{11}f_{22} & \cdots \\ \cdots & \cdots & \cdots \end{bmatrix}. \tag{A.3}$$

The vector of genetic diversity $\mathbf{h}_t$, whose dynamics is described by Eq. 7, converges toward a quasi-stationary distribution ($\boldsymbol{\eta}$) given by the right eigenvector associated to the leading eigenvalue $\lambda_{\mathbf{G}}$ of $\mathbf{G}$, and satisfying the relation

$$\mathbf{G}\boldsymbol{\eta} = \lambda_{\mathbf{G}}\boldsymbol{\eta}. \tag{A.4}$$

The effective size of the metapopulation is obtained from Eq. 8 and can be decomposed as follows (Rousset 2004):

$$\frac{1}{N_e} = \sum_i \frac{v_i^2}{N_i}\frac{h_{ii}}{h} \tag{A.5}$$

where h measures the probability of non-identity of two genes randomly sampled in the population. This expression makes explicit that the asymptotic rate of coalescence depends on three distinct factors. First, differences in productivity between patches are taken into account by the probabilities v_i^2 that two randomly-sampled lineages originate from the same deme i. Second, the sizes of local patches determine the intra-patch rate of coalescence ($1/N_i$). Finally, population subdivision is taken into account by the terms h_{ii}/h measuring the ratio of within-deme to total genetic variance.

Under our assumption of stability, N_e increases with structure (i.e., deviation from panmixia owing to low dispersal rate among demes; $h_{ii} < h$). Since coalescent events occur only within demes, a large amount of genetic variance (the interdemic component) is retrieved

from the action of drift. Demographic variability and local extinctions will of course counteract this effect.

Extinction time

Let us now assume that demography is affected by stochasticity (be it intrinsic or environmental), and consider the ensuing metapopulation dynamics. Each deme i can reach any state between 0 and carrying capacity K_i, so that describing the full system requires a vector $\mathbf{p}$ of dimension $\prod_{i=1}^{n_d}(K_i + 1)$ listing the probabilities $\Pr[\mathbf{n}]$ of being in demographic state $\mathbf{n}$:

$$\mathbf{p} \equiv \{\Pr(0\,0\,\cdots)\Pr(1\,0\,\cdots)\Pr(\cdots\ \cdots\ \cdots)\Pr(K_1\,K_2\,\cdots)\}.$$

This vector changes with time as $\mathbf{p}_{t+1} = \mathbf{P}\mathbf{p}_t$ (Eq. 3), where $\mathbf{P} = |\Pr_f[\mathbf{n}'|\mathbf{n}]|$ is the matrix of forward transition probabilities (probability that a metapopulation in state $\mathbf{n}$ changes to state $\mathbf{n}'$ the next time step). Deleting the first line and first column of $\mathbf{P}$, we obtain the matrix $\mathbf{P}_o$ containing only the transition probabilities for the transient states of the Markov chain (e.g., Halley and Iwasa 1998, Ovaskainen and Hanski 2004 [Box 4.2]; Appendix C). These transient states converge toward a quasi-stationary distribution ($\mathbf{u}$) given by the right eigenvector associated to the leading eigenvalue $\lambda_{\mathbf{P}_o}$ of $\mathbf{P}_o$, and satisfying the relation

$$\mathbf{P}_o\mathbf{u} = \lambda_{\mathbf{P}_o}\mathbf{u}. \tag{A.6}$$

The rate of convergence to extinction $1 - \lambda_{\mathbf{P}_o}$ determines the characteristic time to extinction T_{ex} according to Eq. 4, and can be decomposed as

$$\frac{1}{T_{ex}} = \sum_{\mathbf{n}} \Pr_f(0|\mathbf{n})u(\mathbf{n}). \tag{A.7}$$

The asymptotic rate of extinction is thus the probability that a metapopulation in state $\mathbf{n}$ becomes extinct in the next time step, averaged over the quasi-stationary distribution (Halley and Iwasa 1998).

Integrating demography and genetics

The heterozygosity $\mathbf{h}[\mathbf{n}']_{t+1}$ expected at time $t + 1$, conditional to being in state $\mathbf{n}'$, is obtained as the sum of all transition probabilities over all possible states $\mathbf{n}$ at time t:

$$\mathbf{h}[\mathbf{n}\prime]_{t+1} = \sum_{\mathbf{n}} \Pr_b[\mathbf{n}|\mathbf{n}\prime]\mathbf{G}[\mathbf{n}]\mathbf{h}[\mathbf{n}]_t \tag{A.8}$$

(Karlin 1968, Chia and Pollak 1974), where $\Pr_b[\mathbf{n}|\mathbf{n}']$ is a backward transition probability (probability that a population in state $\mathbf{n}'$ stems from a population in state $\mathbf{n}$). Assuming Poisson distributions of reproduction, and, more generally, that life cycle events affect individuals independently, then $\mathbf{G}$ is independent of $\mathbf{n}'$ (Rousset 2004). Noting that

$$\Pr_b[\mathbf{n}|\mathbf{n}\prime] = \Pr_f[\mathbf{n}\prime|\mathbf{n}]\frac{\Pr[\mathbf{n}]_t}{\Pr[\mathbf{n}\prime]_{t+1}}$$

we can write

$$\mathbf{d}[\mathbf{n}\prime]_{t+1} = \sum_{\mathbf{n}} \Pr_f[\mathbf{n}\prime|\mathbf{n}]\mathbf{G}[\mathbf{n}]\mathbf{d}[\mathbf{n}]_t \tag{A.9}$$

where

$$\mathbf{d}[\mathbf{n}]_t = \Pr[\mathbf{n}]_t\mathbf{h}[\mathbf{n}]_t \tag{A.10}$$

represents the vector (dimension n_d^2) of expected pairwise heterozygosities (given state $\mathbf{n}$) weighted by the probability of being in state $\mathbf{n}$. There is one such vector for all possible states $\mathbf{n}$, which can all be collected into one large vector $\mathbf{d}$ (of dimension $n_d^2(\prod_{i=1}^{n_d}(K_i + 1) - 1)$). This vector changes with time as $\mathbf{d}_{t+1} = \mathbf{T}\mathbf{d}_t$ (Eq. 10), where $\mathbf{T}$ is the matrix of transition probabilities obtained by multiplying the matrix $\mathbf{G}[\mathbf{n}]$ with each relevant scalar element $\Pr_f[\mathbf{n}'|\mathbf{n}]$ of the matrix $\mathbf{P}_o$:

$$\mathbf{T} = |\mathbf{t}_{ij\mathbf{n}\prime;kl\mathbf{n}}| = |\Pr_f[\mathbf{n}\prime|\mathbf{n}]g_{ij,kl}[\mathbf{n}]|. \tag{A.11}$$

The dynamics is dominated asymptotically by the first eigenvalue ($\lambda_{\mathbf{T}}$) of $\mathbf{T}$, so that, once quasi-stationarity is reached, each element of $\mathbf{d}_t$ decreases geometrically at rate $\lambda_{\mathbf{T}}$. Since elements of $\mathbf{p}_t$ also decrease asymptotically at rate $\lambda_{\mathbf{P}_o}$ (Eq. 4), it follows from Eq. A.10 that elements of $\mathbf{h}_t$ must decrease at rate $\lambda_{\mathbf{G}} = \lambda_{\mathbf{T}}/\lambda_{\mathbf{P}_o}$, which by definition (Eq. 8) provides the effective size. The fixation rate of the metapopulation, conditional to non-extinction, is thus given by

$$\frac{1}{N_e} = 1 - \lambda_{\mathbf{G}} = 1 - \frac{\lambda_{\mathbf{T}}}{\lambda_{\mathbf{P}_o}} \tag{A.12}$$

and can be decomposed as follows (Rousset 2004):

$$\frac{1}{N_e} = \sum_{\mathbf{n}} u[\mathbf{n}] \sum_i \frac{\kappa_{ii}[\mathbf{n}]}{N_i} \frac{h_{ii}[\mathbf{n}]}{h} \tag{A.13}$$

where both the asymptotic diversity in deme i ($h_{ii}[\mathbf{n}]$) and the probability that two gene lineages descend from deme i ($\kappa_{ii}[\mathbf{n}]$) are defined conditional on the metapopulation residing in state n. The corresponding decomposition of the asymptotic rate of loss of diversity ($1/T$) is thus obtained by introducing Eqs. A.13 and A.7 into Eq. 13.

APPENDIX B: SYMBOLS AND NOTATIONS

$\Pr[0]_t$	Probability that the focal population is extinct at time t
$p_t \equiv 1 - \Pr[0]_t$	Probability that the focal population is occupied at time t
$\mathbf{p}_t \equiv \lvert\Pr[\mathbf{n}]_t\rvert$	Vector of probabilities that the metapopulation is in state $\mathbf{n}$ at time t
$\mathbf{P} \equiv \lvert\Pr_f[\mathbf{n}'\lvert\mathbf{n}]\rvert$	Matrix of forward transition probabilities among states
$\mathbf{P}_o$	Matrix of forward transition probabilities among transient states
$\lambda_{\mathbf{P}_o}$	Leading eigenvalue of $\mathbf{P}_o$ (index of resistance to extinction)
$\mathbf{u}$	Right eigenvector associated to $\lambda_{\mathbf{P}_o}$ (quasi-stationary distribution of $\mathbf{p}$)
$e \equiv \dfrac{\Pr[0]_{t+1} - \Pr[0]_t}{1 - \Pr[0]_t}$	Extinction rate of the population
$T_{ex} \equiv \dfrac{1}{e}$	Extinction time of the population
N_i	Size of deme i
K_i	Carrying capacity of deme i
N_e	Effective size of the population (time to fixation)
n_d	Number of demes
b	Per capita fecundity
m_{ij}	Forward dispersal rate (probability that a juvenile born in j disperses to i)
$q_{ij} \equiv bN_j m_{ij}$	Contribution of patch j to patch i (numbers of individuals per generation)
$q_i = \sum_j^{n_d} q_{ij}$	Number of individuals settling in patch i
$f_{ij} \equiv q_{ij}/q_i$	Backward dispersal rate (probability that an adult settled in i stems from j)
$\mathbf{F} \equiv \lvert f_{ij}\rvert$	Matrix of backward dispersal rates
v_i	Leading right eigenvector of $\mathbf{F}$ (patch reproductive value)
$\mathbf{A} \equiv \mathbf{F} \otimes \mathbf{F} \equiv \lvert f_{ij} f_{kl}\rvert$	Matrix of backward dispersal rate for pairs of genes
h_t	Expected heterozygosity in the population
$\mathbf{h}_t \equiv \lvert h_{ik}\rvert_t$	Vector of pairwise demic heterozygosities
$\mathbf{G} \equiv \lvert g_{ij,kl}\rvert$	Matrix of transition among pairwise demic heterozygosities
$\lambda_{\mathbf{G}}$	Leading eigenvalue of $\mathbf{G}$ (index of resistance to genetic drift)
$\boldsymbol{\eta}$	Right eigenvector associated to $\lambda_{\mathbf{G}}$ (quasi-stationary distribution of $\mathbf{h}$)
$d_t \equiv p_t h_t$	Index of demo-genetic diversity
$\mathbf{d}_t \equiv \lvert\Pr[\mathbf{n}]\mathbf{h}[\mathbf{n}]\rvert_t$	Vector of probabilities of demo-genetic states
$\mathbf{T} \equiv \lvert\Pr_f[\mathbf{n}'\lvert\mathbf{n}] g_{ij,kl}[\mathbf{n}]\rvert$	Matrix of forward transition among demo-genetic states
$\lambda_{\mathbf{T}}$	Leading eigenvalue of $\mathbf{T}$ (index of demo-genetic resistance)

APPENDIX C

Markov chains and absorption times for demography and genetics (*Ecological Archives* E087-108-A1).

SUPPLEMENT

Mathematica worksheets for calculating and plotting demo-genetic eigenvalues (*Ecological Archives* E087-108-S1).