
Refugees from Lost Habitat and Reorganization of Genetic Population Structure

ADAM H. PORTER

Department of Entomology and Graduate Program in Organismic and Evolutionary Biology,
University of Massachusetts, Amherst, MA 01003-2410, U.S.A., email aporter@ent.umass.edu

Abstract: *When habitat is lost, individuals of many species are able to flee and reestablish themselves in other sites. Such cases may be studied with refugee models of genetic population structure. The fleeing refugees carry their genes, which can result in a reorganization of genetic structure. Differentiation among demes (F_{ST}) is reduced, and additive genetic variance (V_A) within remaining demes is increased. These effects accumulate for as many generations as habitat continues to be lost. After habitat loss and stabilization of a new habitat structure, genetic differentiation among demes and fixation of rare or deleterious alleles will again increase by genetic drift, a slow process, and will ultimately equilibrate at new levels of F_{ST} and V_A . The temporary increase of V_A in surviving demes increases their mean fitness and can forestall problems with inbreeding depression and the ability of demes to adapt to environmental changes. It is likely that a large proportion of species of conservation concern are able to flee during habitat loss. Many of these have probably recently experienced significant genetic restructuring and have not returned to equilibrium levels of genetic differentiation. This introduces a temporary upward bias when interdemic gene flow rates (Nm) are inferred from F_{ST} , and it inflates the measures of the long-term standing genetic variance, V_A , that these populations are able to maintain. In principle, these biases can be estimated and some gross aspects of pre-fragmentation genetic structure can be recovered, but much more empirical data are needed on the dynamics of refugee establishment.*

Refugiados de la Pérdida de Hábitat y Reorganización de la Estructura Genética Poblacional

Resumen: *Cuando se pierde hábitat, los individuos de muchas especies tienen la capacidad de abandonar el sitio y reestablecerse en otros lugares. Estos casos pueden ser estudiados mediante modelos de refugiados de la estructura genética poblacional. Los refugiados cargan sus genes y esto puede resultar en una reorganización de la estructura genética. La diferenciación entre demes (F_{ST}) se reduce y la varianza genética aditiva (V_A) dentro de los demes remanentes se incrementa. Estos efectos se acumulan por generaciones siempre y cuando el hábitat continúe perdiéndose. Después de la pérdida del hábitat y la estabilización de una nueva estructura de hábitat, la diferenciación genética entre demes y la fijación de alelos raros y dañinos se incrementarían una vez más debido a deriva génica, un proceso lento que finalmente habrá de equilibrar niveles nuevos de F_{ST} y V_A . El incremento temporal de V_A en los demes sobrevivientes incrementa la forma promedio y puede prevenir problemas con depresión por intracruza y la habilidad de los demes para adaptarse a los cambios ambientales. Es probable que una proporción grande de especies de interés conservacionista sean capaces de abandonar sitios durante la pérdida de hábitat. Muchas de estas especies posiblemente han experimentado reciente reestructuración genética y no han retornado a niveles de equilibrio de diferenciación genética. Esto introduce un sesgo temporal cuando se hacen inferencias sobre tasas de flujo interdémico de genes (Nm) a partir de F_{ST} , e infla las mediciones de la varianza genética mantenida a largo plazo, V_A , que estas poblaciones pueden mantener. En principio, estos sesgos pueden ser estimados y ciertos aspectos generales de la estructura genética prefragmentación pueden ser recuperados, sin embargo, aún se necesitan muchos más datos empíricos sobre las dinámicas del establecimiento de refugiados.*

Paper submitted June 17, 1998; revised manuscript accepted November 26, 1998.

Introduction

Genetic population structure is the pattern in which alleles are distributed within and among populations of a species. It has important implications in evolutionary biology because it defines the rate (Fisher 1930) and spatial scale (Wright 1951, 1969) at which populations can evolve in response to environmental perturbations. Patterns of genetic population structure are influenced by basic evolutionary processes including gene flow, genetic drift (a consequence of population size), mutation, natural selection, and assortative mating; as any text will show (e.g., Hartl & Clark 1997), evolutionary geneticists have developed a large body of theory explaining how these processes affect genetic patterns. In many cases it is possible to apply these models in reverse to estimate these processes quantitatively from the genetic patterns, provided that certain assumptions are met (Slatkin 1987). These applications have important roles in conservation biology because managers can make better decisions with knowledge of population parameters measured from genetic population structure (Loeschke et al. 1994).

Much of the theory relating genetic patterns to evolutionary processes has been developed for large and widespread species and concerns long time scales, because these conditions were considered the most relevant for general explanations of evolutionary change (Wright 1969). Presently, conservation biologists are more concerned with the consequences of relatively short-term evolutionary processes, on the scale of tens to hundreds of generations, in populations small enough to undergo relatively rapid stochastic evolution (Loeschke et al. 1994). Understanding these consequences requires that we revisit the basic explanatory models of genetic population structure and modify their conditions to investigate evolutionary changes in populations with structures relevant to conservation biology. Where this has been done, for example in the interactions of mutation and genetic drift in small populations (Franklin 1980; Lande & Barrowclough 1987; Lynch & Gabriel 1990; Lande 1995), the results have led to fundamental conservation policies.

Refugee Models of Population Genetic Dynamics

One common and often valid modeling assumption in conservation genetics is that when a habitat site is destroyed, all residents are exterminated. In these models, the array of demes remaining after habitat loss is studied (e.g., Lacy 1987) or the extinction of demes is modeled in combination with the establishment of new demes as habitat is recolonized (e.g., Slatkin 1977; Wade & McCauley 1988; Whitlock & McCauley 1990; Barton & Whitlock 1997; Hedrick & Gilpin 1997). *Refugee models* of population structure relax the assumption that loss of habitat at a site

means certain death of the occupants, instead permitting refugees to flee and become established in remaining sites. Marshall (1995) was the first to suggest that refugee movements might be important, and she invoked them to explain unexpected patterns in the genetic structure of butterfly populations in forest remnants. Refugee models in general may be based on island (Wright 1931), stepping-stone (Kimura & Weiss 1964), or continuous (also known as isolation-by-distance, Wright 1943, 1969) population structures and may include random or spatially autocorrelated patterns of habitat loss. Although I emphasize genetics, refugee models also apply to purely ecological situations. To demonstrate the general influences of refugee flight on genetic structure, I considered the simplest refugee-model case of random habitat loss in an island model of population structure.

Some taxa are more appropriate than others for refugee models. A significant proportion of taxa are likely to be able to flee from habitat during its destruction and reestablish themselves elsewhere. These include species that encounter the habitat deterioration or loss period during a stage of their life cycle that permits dispersal. Most vertebrates are likely to fall into this category, as are most flying insects. Other less mobile species may also be included, depending on the scale of habitat loss relative to the scale of long-distance dispersal ability. For example, refugee models may be appropriate for forest millipedes when habitat is clearcut on the scale of single-hectare blocks, but not when habitat is cleared over several square kilometers. Many taxa, particularly plants, disperse during gametic or zygotic stages of limited duration, so refugee models are probably inappropriate for them.

I show that when individuals are capable of fleeing to remaining sites as refugees when habitat is lost, a reorganization of genetic structure occurs that has two important consequences for evolutionary and conservation biology. First, genetic reorganization by movements of refugees can throw genetic structure out of the equilibrium state for a long time. Equilibrium is the fundamental assumption that must be met to measure evolutionary processes such as gene flow based on patterns of genetic structure. Second, additive genetic variance within populations increases. This can increase average fitness and ameliorate the inbreeding effects that can accompany habitat fragmentation, with benefits lasting until the genetic structure equilibrates again. Before developing arguments on these points, I briefly review the relevant effects of gene flow on genetic structure in the absence of habitat loss as a baseline for comparison.

Genetic Structure without Habitat Loss

Consider a population of individuals distributed among n habitat patches spread across a landscape, such that there is one deme per habitat patch and all patches are

occupied. Patterns of genetic variation within and among demes can be described with a great variety of population genetic parameters, including heterozygosities, numbers of alleles, genetic distances, and so forth. It is useful to choose those that permit the measurement of the evolutionary processes of interest, and I employed two widely used parameters, F_{ST} and V_A . Differentiation among demes relative to the total variation available is described by F_{ST} . It is widely used in inferences of gene flow among demes and is proportional to the probability of fixation of deleterious alleles in individual demes. It is defined for a single locus as

$$F_{ST} = \frac{E\{(p_i - \bar{p})^2\}}{\bar{p}(1 - \bar{p})}, \quad (1)$$

where p_i is the frequency of allele A in deme i , \bar{p} is the average frequency, and the operator E denotes expectation (i.e., finding the mean value with an infinitely large sample size). The numerator is thus the variance of allelic frequencies among demes, and the denominator is the genetic variance available in the total population when A alleles are scored with values of 1 and non- A alleles are given values of 0 (Falconer 1989). V_A describes the additive genetic variance within a deme and can be derived as a function of allele frequencies (Lynch 1988). Its formula for a single locus in deme i is

$$V_{A(i)} = 2\alpha^2 p_i(1 - p_i),$$

or as an average over demes,

$$\bar{V}_A = E\{V_{A(i)}\} = 2\alpha^2 E\{p_i(1 - p_i)\} \quad (2)$$

(Falconer 1989), where α is the average extent that the phenotype is affected by an A allele, expressed in the units by which the trait is measured. When $\alpha = 1$, the single locus V_A is equal to the expected heterozygosity. The additive genetic variance of a polygenic trait is the sum over loci of this single-locus formula (Falconer 1989). Thus, the effect of evolutionary processes on the single-locus V_A is representative of their effect on the V_A of polygenic traits. Fisher (1930) showed that V_A describes the potential for a population to evolve in response to environmental change, and long-term maintenance of a suitably high value of V_A is a management goal in conservation biology (Lynch & Gabriel 1990; Lande 1995). V_A decreases as populations become fixed for alleles by local inbreeding, so V_A estimated from genetic markers can be used by empirical conservation biologists to loosely assess the relative chances that a particular deme has become fixed for deleterious alleles.

Gene flow homogenizes allele frequencies among demes and increases local genetic variation within demes; genetic drift has the opposite effect (Wright 1969). These effects are captured parametrically in values of F_{ST} and V_A . I further restricted the problem to cases of only neutral loci, random mating within demes, and Wright's (1931, 1969) island model of migration

(immigrant individuals may come with equal probability from any other deme, and generations are discrete). The life cycle is assumed to comprise the movement of pre-reproductive stages (zygotes or juveniles), which then mature and produce gametes that unite randomly. After a generation, the new expected frequency of allele A in deme i is

$$p_{i(t+1)} = p_{i(t)} - mp_{i(t)} + m\bar{p}_{(t)}, \quad (3)$$

where m is the proportion of migrants among demes, $mp_{i(t)}$ is the allele frequency of the emigrants, $m\bar{p}_{(t)}$ is that of the immigrants, and the subscript t denotes generation (Wright 1931). The new average frequency is equal to that in the previous generation,

$$\bar{p}_{(t+1)} = E\{p_{i(t)} - mp_{i(t)} + m\bar{p}_{(t)}\} = \bar{p}_{(t)}.$$

In the dispersal stage of the life cycle, prior to genetic drift, the new values of $F_{ST(t+1)}$ and $\bar{V}_{A(t+1)}$ can be found by substituting $p_{i(t+1)}$ into equations 1 and 2. This yields

$$F_{ST(t+1)} = (1 - m)^2 F_{ST(t)} \quad (4)$$

and

$$\bar{V}_{A(t+1)} = \bar{V}_{A(t)} + m(2 - m)V_{A(\text{total})}F_{ST(t)}, \quad (5)$$

where $V_{A(\text{total})}$ is the additive genetic variance of the total population, pooling all the demes. Thus, gene flow transfers genetic differentiation among demes (F_{ST}) into variation within demes (V_A) in proportion to the gene flow rate (m).

Genetic homogenization is offset by genetic drift, and adding genetic drift to equation 4 yields

$$F_{ST(t+1)} = (1 - m)^2 \left[\frac{1}{2N} + \frac{2N - 1}{2N} F_{ST(t)} \right] \quad (6)$$

(Wright 1931, 1969), where the bracketed term describes the component of differentiation due to genetic drift, and N is the effective population size. The opposing rates of gene flow and drift eventually result in an equilibrium level of differentiation, such that $F_{ST(t+1)} = F_{ST(t)} = F_{ST(\text{eq})}$, giving

$$F_{ST(\text{eq})} = \frac{(1 - m)^2}{2N - (2N - 1)(1 - m)^2}, \quad (7a)$$

$$F_{ST(\text{eq})} \approx \frac{1}{4Nm + 1} \quad (7b)$$

Wright (1931, 1969), where the latter, more familiar approximation (Slatkin 1987) holds when m is small.

Genetic drift reduces the average additive genetic variance in populations because alleles become fixed or lost stochastically. The average fixation rate is $1/(2N)$ per deme per generation (Wright 1969: 346), such that an average of $1/(4N)$ demes become fixed for A alleles ($p = 1$) and $1/(4N)$ lose their A alleles ($p = 0$). The average variance after a generation includes the proportion of demes acquiring zero genetic variance because of loss or

fixation. In the absence of gene flow or other systematic changes in gene frequency, $E\{p_{i(t+1)}\} = p_{i(t)}$, and the new additive genetic variance after gene flow and drift is therefore

$$\bar{V}_{A(t+1)} = \frac{2N-1}{2N} \bar{V}_{A(t)} + m(2-m)V_{A(\text{total})}F_{ST(t)}. \quad (8)$$

The equilibrium level of additive genetic variance can be found from equation 8 by setting $V_{A(t+1)} = \bar{V}_{A(t)} = \bar{V}_{A(\text{eq})}$ and substituting equation 6. This yields

$$\bar{V}_{A(\text{eq})} = (1-m)^2(1-F_{ST(\text{eq})})V_{A(\text{total})}. \quad (9)$$

The equilibrium level of additive genetic variance within demes is therefore slightly less than the additive genetic variance expected from the total population. Equations 8 and 9 are averages because the loss of variance is a stochastic process. Lynch (1988) found similar results for a pair of populations exchanging limited gene flow. In his models of finite deme numbers, the entire system of demes drifts to fixation unless offset by input from new mutations, and the equilibrium $V_{A(\text{total})}$ is determined by the mutation rate. The rate of equilibration depends on the gene flow rate (Lynch 1988). When gene flow is strong, the demes behave as a large, interconnected population and equilibration proceeds at a drift rate close to that of the entire population,

$$\bar{V}_{A(t+1)} \approx \frac{2Nn-1}{2Nn} \bar{V}_{A(t)}, \quad (10a)$$

where n is the number of demes. This process can be quite slow. When gene flow is weak, the demes drift independently and equilibration proceeds at rates close to that of individual demes,

$$\bar{V}_{A(t+1)} \approx \frac{2N-1}{2N} \bar{V}_{A(t)}, \quad (10b)$$

and potentially much faster, but still possibly rather slow on the time scales interesting to many evolutionary and conservation biologists. This represents a sort of null model of additive genetic variance, and $\bar{V}_{A(\text{eq})}$ for particular traits or for fitness will be further influenced by natural selection (Fisher 1930) and by epistasis (Whitlock et al. 1993).

A Refugee Model of Genetic Structure

What happens to these measures of genetic differentiation when a proportion b of the n original habitat patches is lost? In developing new estimates of genetic variation, we must consider the fates of individuals in the surviving demes as well as the refugees from destroyed demes. The life cycle is as above, with the added condition that habitat loss occurs during the dispersal

stage of the life cycle. After habitat loss, the remaining $(1-b)n$ demes will continue to produce emigrants normally, and a total of $Nm(1-b)n$ individuals, carrying

$$2Nm \sum_i^{(1-b)n} p_i$$

A genes, will join the migrant pool. The residents of the bn destroyed habitat patches may either flee as refugees to other patches and establish themselves in extant demes with probability r or die without reproducing with probability $(1-r)$. A total of $Nrhn$ individuals, carrying

$$2Nr \sum_j^{hn} p_j$$

A genes, will join the refugee pool. Thus, the emigration rate from extant patches stays constant at m , but immigration is supplemented by refugees. The refugees distribute themselves among the $(1-b)n$ extant patches, so that the number of individuals arriving in each deme is

$$\frac{2N(1-b)nm}{(1-b)n} + \frac{2Nhn r}{(1-b)n} = 2Nm + \frac{2Nhr}{(1-b)}.$$

If the demes destroyed are a random sample of the total, then $E\{p_i\} = E\{p_j\} = \bar{p}$ (where i and j are indices for normal-migrant and refugee subgroups within the same deme) and the number of A alleles arriving in each deme is

$$2Nm\bar{p} + \frac{2Nhr}{(1-b)}\bar{p}.$$

The new allelic frequency after a generation of migration and habitat fragmentation is therefore

$$\begin{aligned} p_{i(t+1)}^* &= \frac{2Np_{i(t)} - 2Nmp_{i(t)} + 2Nm\bar{p}_{(t)} + \frac{2Nhr}{(1-b)}\bar{p}_{(t)}}{2N - 2Nm + 2Nm + \frac{2Nhr}{(1-b)}}, \\ p_{i(t+1)}^* &= \frac{(1-b)p_{i(t)} - m(1-b)(p_{i(t)} - \bar{p}_{(t)}) + hr\bar{p}_{(t)}}{1-b+hr}. \end{aligned} \quad (11)$$

The average allele frequency after habitat loss is unchanged, $\bar{p}_{(t+1)}^* = E\{\bar{p}_{i(t+1)}^*\} = \bar{p}_{(t)}$.

The influx of refugees causes the actual population size (N_A) to increase by a factor

$$N_{A(t+1)} = \left(1 + \frac{hr}{1-b}\right)N_A. \quad (12)$$

This seems unlikely, however, to have a significant influence on the effective population size because the populations will revert to their average sustainable sizes relatively quickly. I therefore use N throughout and comment where appropriate on the influence of using

equation 12, which can be taken as a maximum effect, in its place.

Differentiation among Demes

After habitat loss, the new level of differentiation among demes can be found by substituting equation 11 into the definition of F_{ST} in equation 1:

$$F_{ST(t+1)}^* = \left[\frac{1-h}{1-h(1-r)} \right]^2 (1-m)^2 F_{ST(t)}. \quad (13)$$

When genetic drift within the extant sites is included following equation 6, this becomes

$$F_{ST(t+1)}^* = \left[\frac{1-h}{1-h(1-r)} \right]^2 (1-m)^2 \left[\frac{1}{2N} + \frac{2N-1}{2N} F_{ST(t)} \right]. \quad (14)$$

Equation 12 may be substituted for N to account for the increase in population size caused by adding refugees, whereupon equation 14 remains a close approximation unless N is very small or the refugee pool is very large. If the population had previously been at equilibrium levels of differentiation, then equation 6 may be substituted, which simplifies to

$$F_{ST(t+1)}^* = \left[\frac{1-h}{1-h(1-r)} \right]^2 F_{ST(eq)}.$$

Relative to unfragmented habitat at equilibrium (equation 7a), habitat loss reduces differentiation in one generation by the ratio of $F_{ST(t+1)}^*/F_{ST(eq)}$ or

$$\frac{F_{ST(t+1)}^*}{F_{ST(eq)}} = \left[\frac{1-h}{1-h(1-r)} \right]^2. \quad (15)$$

This ratio is always <1 when refugees are able to escape destroyed habitat, so genetic homogenization among demes is promoted. If habitat loss is severe ($h > 0.2$) in one generation, and the proportion of refugees at least moderate ($r > 0.2$), then F_{ST} can be reduced by 10% or more (Fig. 1).

Variation within Demes

The lost interdemetic variation must go somewhere. Via refugees, it is translated into increased additive genetic variance within the remaining demes. This can be seen by substituting $p_{i(t+1)}^*$ (equation 11) into the definition of additive genetic variance, leading to

$$E\{V_{A(t+1)}^*\} = \left[\frac{1-h}{1-h+hr} \right]^2 \left[\bar{V}_{A(t)} + m(2-m)V_{A(total)}F_{ST(t)} \right] + \frac{hr(2-2h+hr)}{(1-h+hr)^2} V_{A(total)}. \quad (16)$$

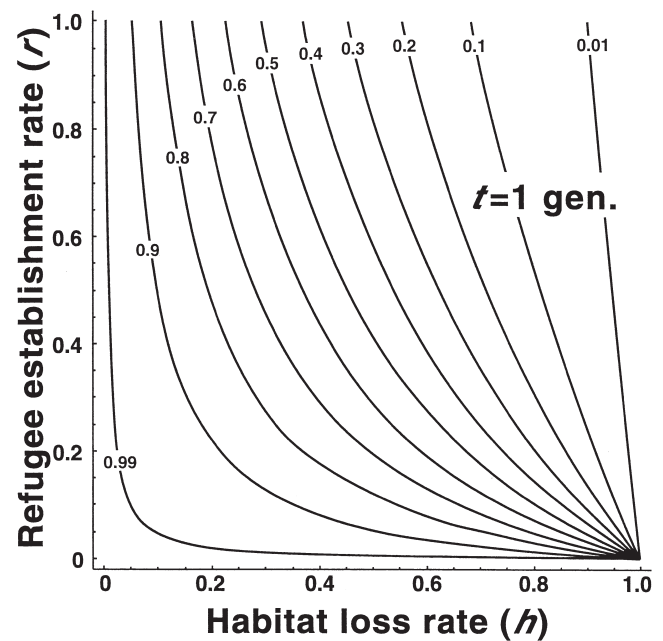


Figure 1. Isoclines of $F_{ST(t+1)}^*/F_{ST(eq)}$ (equation 15), a ratio describing the extent of genetic homogenization among demes produced in a single generation ($t = 1$) by the interaction of the rates of habitat loss (h) and refugee establishment (r). The effect is independent of population size and migration rate.

When genetic drift is added to this model, following equation 8, this becomes

$$E\{V_{A(t+1)}^*\} = \left[\frac{\left[\frac{1-h}{1-h+hr} \right]^2 \left[\frac{2N-1}{2N} \bar{V}_{A(t)} + m(2-m)V_{A(total)}F_{ST(t)} \right] + \frac{hr(2-2h+hr)}{(1-h+hr)^2} V_{A(total)}}{1} \right]. \quad (17)$$

As above, equation 12 may be substituted for N to account for the increase in population size caused by refugees, but the effect is negligible unless N is very small or the refugee pool very large. If the population had previously been at equilibrium with respect to gene flow and genetic drift, then equation 9 may be substituted, which simplifies to

$$E\{V_{A(t+1)}^*\} = \left[\left(\frac{1-h}{1-h+hr} \right)^2 (1-m)^2 (1-F_{ST(eq)}) + \frac{hr(2-2h+hr)}{(1-h+hr)^2} \right] V_{A(total)}. \quad (18)$$

Relative to undamaged habitat (equation 9), a single generation of habitat loss therefore changes intrademic genetic variance by a factor of

$$\frac{E\{V_{A(t+1)}^*\}}{\bar{V}_{A(eq)}} = \left(\frac{1-h}{1-h+hr}\right)^2 + \frac{hr(2-2h+hr)}{(1-h+hr)^2(1-m)^2(1-F_{ST(eq)})} \quad (19)$$

This ratio is always >1 when refugees can flee destroyed habitat (Fig. 2), indicating that additive genetic variance within remaining demes increases.

Multiple Generations of Habitat Loss

Genetic reorganization continues for every generation during which habitat is lost. The extent of reorganization may be found by iterating equation 14 and equation 17 for the required number of generations. The analytical solutions are difficult to obtain, and it is more convenient to use numerical iteration. The reduction in F_{ST} after 10 generations of constant habitat loss is much more than that seen in a single generation (Fig. 3; compare with Fig. 1). Biologically relevant values of h for multiple generations must be rather low to ensure that a reasonable proportion of habitat remains after t generations, and the top axis in Fig. 3 represents the proportion of habitat remaining after 10 generations of loss at rate h . There is a corresponding increase in V_A that is considerably more than that seen in a single generation (Fig. 4;

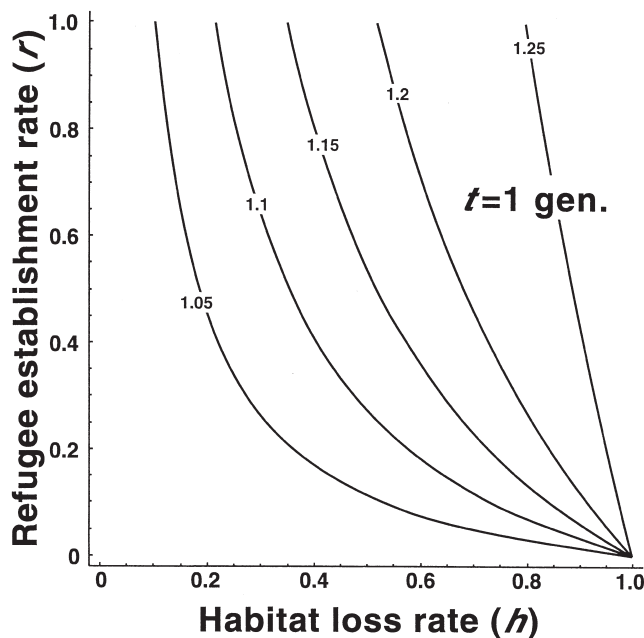


Figure 2. Isoclines of $\bar{V}_{A(t+1)}^*/\bar{V}_{A(eq)}$ (equation 19), describing the increase of additive genetic variance produced in a single generation ($t = 1$) by the interaction of the rates of habitat loss (h) and refugee establishment (r). Shown are contours for population size $N = 100$, migration rate $m = 0.1$, and allele frequency $p = 0.5$.

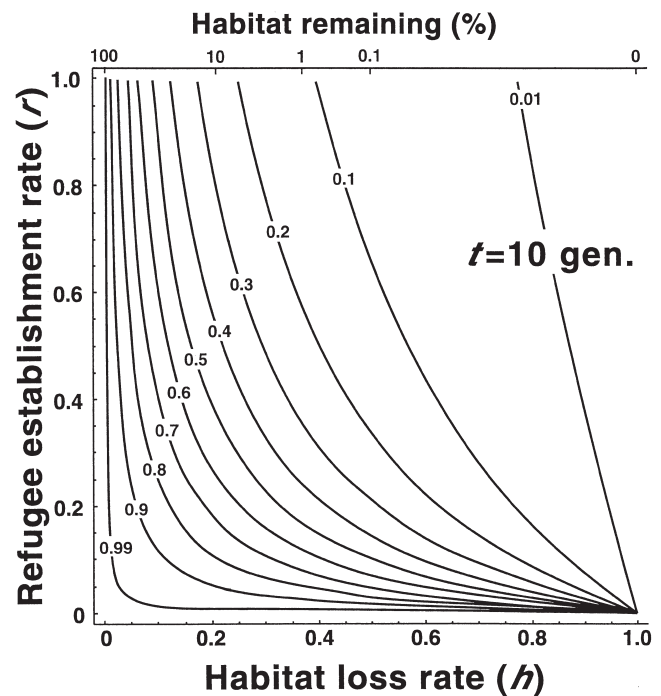


Figure 3. Isoclines of $F_{ST(t+1)}^*/F_{ST(eq)}$ (equation 15; see Fig. 1) iterated over $t = 10$ generations, describing the extent of genetic homogenization among demes produced by the compounded interactions of the rates per generation of habitat loss (h) and refugee establishment (r). The effect is independent of population size and migration rate. The top axis shows the habitat remaining after continued habitat loss $(1-h)^t$; rates of $h > 0.5$ for 10 generations are unrealistic for most species.

compare with Fig. 2). After about 10 generations, continued loss of habitat has little effect on the shape of the contours relative to the habitat-remaining axis. This is because refugees over the period of extended habitat loss can be seen as having an effect similar to that of an increase in normal migration patterns (higher m) in a constant habitat.

The reduction of F_{ST} and increase in V_A occur for all loci simultaneously, so the means of these parameters across loci will be similarly affected. This reorganization is in contrast to stochastic variation about the equilibrium, in which increases in one locus are offset by decreases in another.

After Habitat Loss

The genetic reorganization is a temporary effect, lasting as long as habitat fragmentation continues. Starting at equilibrium (Fig. 5), habitat loss rapidly decreases F_{ST} and increases V_A throughout the episode, following equations 14 and 17. At that point, the populations begin to drift back toward their equilibrium state (equa-

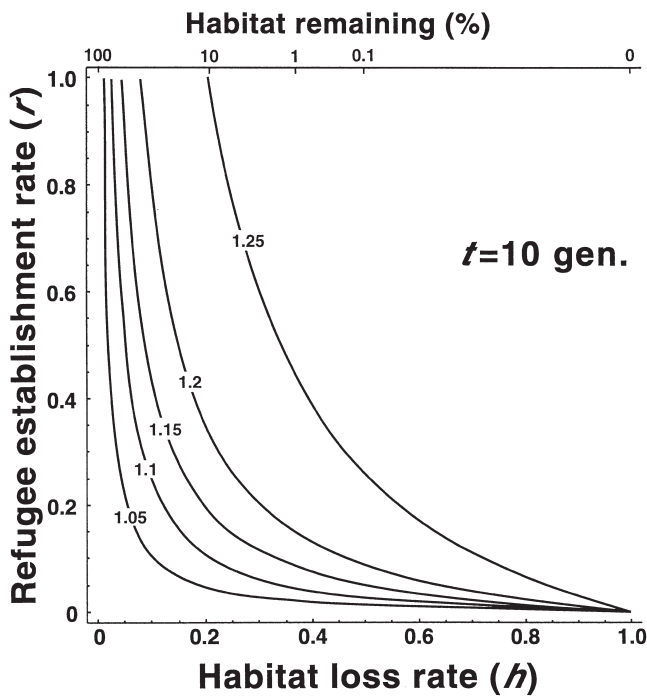


Figure 4. Isoclines of $\bar{V}_{A(t+1)}^*/\bar{V}_{A(eq)}$ (equation 19; see Fig. 2) iterated over $t = 10$ generations, describing the increase of additive genetic variance produced by the compounded interaction of the rates per generation of habitat loss (h) and refugee establishment (r). The top axis shows the habitat remaining after continued habitat loss $(1 - h)^t$; rates of $h > 0.5$ are unrealistic for most species. Shown are contours for population size $N = 100$, migration rate $m = 0.1$, and allele frequency $p = 0.5$.

tions 7a and 9) at the rates given in equations 10a and 10b. The approach to the post-fragmentation equilibrium can be lengthy. If gene flow rates depend on the distances among remaining demes, a new, post-fragmentation effective gene flow rate, m^* , may be established. The remaining demes would then approach a different equilibrium level of genetic differentiation of $F_{ST} \approx 1/(4Nm^* + 1)$. It may also be that a metapopulation structure is established, whereupon the new equilibrium structure would also be influenced by the extinction and colonization rates of demes (Slatkin 1977; Wade & McCauley 1988; Whitlock & McCauley 1990). Unless both m^* and the deme sizes are very small, or the extinction and colonization rates high, the re-equilibration period will be several orders of magnitude longer than the duration of the genetic reorganization experienced during habitat loss. Provided that extinction is avoided, however, the genetic structures of spatially highly fragmented populations will eventually reach high levels of F_{ST} , as exemplified by the naturally fragmented populations of pupfish (*Cyprinodon* spp., Cyprinidae) of the desert southwest of North America (Meffe & Vrijenhoek 1988).

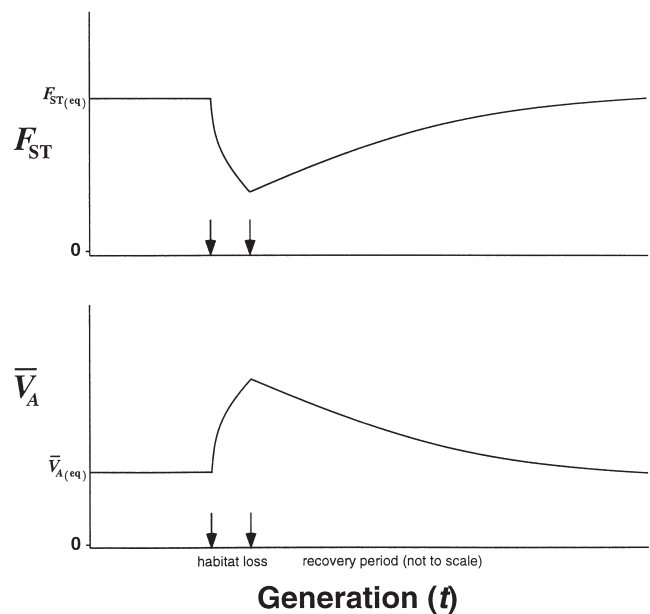


Figure 5. The pattern of genetic reorganization in the generations (t) before, during, and after an episode of habitat loss, beginning and ending at the arrows.

Finite Deme Numbers

The development of this argument is based upon an infinite (or very large) number of demes, and this implies that average allelic frequency (\bar{p}) remains (nearly) constant in the global population. For low-density, spatially restricted species with low values of r , loss of habitat may produce significant changes in \bar{p} , including global loss and fixation of alleles. This in turn produces stochastic changes in the additive genetic variance in the global population $V_{A(\text{total})}$, and in F_{ST} . Although the average effect of refugee flight is to reduce F_{ST} and increase V_A , the stochasticity becomes increasingly important in determining their realized values. In especially restricted species, these fluctuations tend to obscure the refugee effects that would be seen more easily in larger arrays of demes.

Discussion

Refugees fleeing destroyed habitat carry their genes, increasing genetic variation within remaining demes and decreasing differentiation among them. This reorganization of population genetic structure is immediate, it affects all loci, it compounds over generations of habitat loss, and it can be quite strong if the refugee establishment rate is moderate to high (Figs. 2 & 4). Once reorganization is achieved and habitat loss ceases, re-equilibration is a slow process that takes place by genetic drift. Habitat change has been widespread, and it seems reasonable to expect that most animal populations of con-

servation concern and many others studied empirically are likely to be near the peaks of the graphs in Fig. 5, far from equilibrium patterns of genetic structure. There are several consequences important for evolutionary and conservation biologists.

Inferences of Population Processes

It is common for empirical population geneticists to estimate the gene flow rate (Nm) from genetic differentiation (F_{ST}) using equation 7b (Slatkin 1987). Because of the reciprocal relationship of Nm and F_{ST} , a downward bias in F_{ST} due to refugee effects will lead to a proportionally larger upward bias in the Nm estimate. Over a range of realistic natural values of F_{ST} , a 20% decrease of F_{ST} causes a 25–35% jump in the gene flow estimate. I used F_{ST} to describe the among-population effect of genetic reorganization, but other measures of genetic similarity and distance will likewise be affected by refugee movements, and most inferences based on these measures will also be biased. For example, conservation geneticists advise reserve designers to protect demes having divergent frequencies of marker alleles (e.g., Crozier 1992), an indicator that the demes are likely to be more valuable genetically than an array of more similar demes. An influx of refugees will reduce genetic distances, decreasing the apparent conservation value of divergent demes, even though the original causes for their initial divergence may still exist, such that their actual value to conservation efforts may be higher than supposed.

Genetic reorganization resulting from refugee movements during habitat loss can delay the “mutational meltdown” effect, accelerated extinction due to accumulated deleterious mutations. It is well known that isolated populations are more prone to inbreeding depression, the accumulation of unfavorable mutations through genetic drift (Simberloff 1988). This gradual erosion of population mean fitness can result in a mutational meltdown if the population size is too small to generate new mutants at a threshold rate (Lynch & Gabriel 1990; Lynch et al. 1993). If a population has become isolated following habitat loss under the refugee model, then, depending on b , r , and t during habitat loss, its starting level of $V_{A(max)}$ can be considerably higher than that expected from equilibrium conditions. From equation 10b and following Lynch (1988), the increase in the time to mutational meltdown of a newly isolated deme may be roughly estimated from the relationship

$$t \approx -2N \ln \left(\frac{\bar{V}_{A(eq)}}{\bar{V}_{A(max)}} \right). \quad (20)$$

The increased time amounts to roughly $0 \leq t < 0.6N$ additional generations, based on the range of parameter values in Figs. 3 and 4. For many species, this increase buys significant time for management intervention before the effects of inbreeding greatly increase the proba-

bility of extinction. In addition, the temporary increase in additive genetic variance within surviving demes permits a more rapid evolutionary response (Fisher 1930) to local environmental changes associated with habitat modification. Such modifications are likely to occur in demes near areas affected by habitat loss (e.g., Lovejoy et al. 1986; Olesen & Jain 1994).

If values of b , r , and t could be estimated empirically for a population experiencing habitat loss, and if estimates were available for current values of F_{ST} and V_A , it would then be possible to estimate $F_{ST(eq)}$ and $V_{A(eq)}$ by rearranging equations 15 and 19, numerically iterating if necessary over the t generations. If these rates were not constant, then their appropriate time series could as readily be used. This would permit the estimation from equation 7b of “normal” or pre-fragmentation rates of gene flow in the absence of refugees. For many systems, reasonable estimates (within an order of magnitude) of b and t are likely to be available from historical records, but r is rarely studied. If the post-fragmentation constellation of habitats has an influence on gene flow rates, however, the system will reach a new equilibrium genetic structure; the original equilibrium structure will still be of interest to historians of recent evolutionary change.

Other Population Structures

The general effects of refugees seen here in the island model are also expected to occur in other population structures, but the magnitudes of the effects may differ. In stepping-stone (Kimura & Weiss 1964) and isolation-by-distance (Wright 1951, 1969) structures, refugees will carry their genes only to nearby extant demes. We would therefore expect, in the initial stages of habitat loss, that F_{ST} will decrease and V_A increase only near the edges of fragmentation. In the later stages with only a small proportion of demes remaining, the outcome will likely be close to the island model result. This is the scenario proposed by Marshall (1995). She compared the genetic structures of satyrine butterfly (*Megisto cymela*) populations in forest remnants in northwestern Ohio to populations in continuous habitat in southern Ohio. She found high heterozygosities in all sites and F_{ST} to be lower by a factor of two in the fragmented population, in agreement with a refugee model and opposite to the pattern expected from equilibrium genetic structure. Her scenario is consistent with the available historical information on deforestation in northwestern Ohio (Marshall 1995).

As habitat is lost, it may also become difficult for both refugees and normal immigrants to find extant demes, and immigration from both sources may be reduced. This would tend to lessen the extent of reorganization in later stages of habitat loss. If habitat is not lost too rapidly, however, much of the reorganization may be by that

time have already been achieved. Another possibility is that migrants might travel different distances than refugees do. If refugees travel further, the extent of reorganization of genetic population structure could be greater. But refugees from greater distances might import alleles that lead to outbreeding depression, canceling some of the positive effects of increased additive genetic variance.

Other factors influence population structure in addition to the distances among demes, and these may interact with refugee movements in other ways. When demes exist as a metapopulation with extinction and recolonization (Wade & McCauley 1988; Whitlock & McCauley 1990; Whitlock 1992; Barton & Whitlock 1997; Hedrick & Gilpin 1997), the reorganizing effect of refugee flight will depend in its magnitude on the patterns in which refugees join extant demes or found new sites. In systems that include social structure within demes, the magnitude of reorganization will depend on the extent to which refugees can insinuate themselves into extant social units or establish new ones. In some species, only one sex normally migrates, whereupon the refugee establishment process may lead to even greater genetic reorganization because both sexes will be represented in the refugee pool. Each of these scenarios deserves exploration in greater detail.

Refugee Establishment Rates

There are few quantitative data available to address refugee establishment rates (r) empirically. Lovejoy et al. (1986) report crowding effects in understory birds fleeing from Amazonian clearcuts. They found capture rates increasing in proportion to remnant habitat size, up to four times greater in 1-ha reserves. These capture rates decreased with time, probably as birds fled again to nearby larger tracts of forest. Lovejoy et al. (1986) also found evidence that a significant proportion of resident birds in the remnants were displaced by refugees, an effect that would amplify the genetic reorganization modeled here. Ash (1997) followed the flight of plethodontid salamanders (*Plethodon jordani*) from clearcuts and their eventual recolonization from the surrounding forest as habitat returned. He could provide robust population-level data only on the recovery, and he pointed out the logistic difficulties researchers are likely to encounter in determining the rate of refugeeism. Given the magnitude of its potential effect in reorganizing genetic structure, more empirical data are needed on the dynamics of refugee establishment.

Acknowledgments

This work was supported by grant DEB-9532079 from the National Science Foundation. I thank M. Gromko, E. Ja-

kob, N. Johnson, K. Marshall, D. McCauley, K. Scribner, and two anonymous referees for valuable comments.

Literature Cited

- Ash, A. N. 1997. Disappearance and return of plethodontid salamanders to clearcut plots in the southern Blue Ridge mountains. *Conservation Biology* 11:983-989.
- Barton, N. H., and M. C. Whitlock. 1997. The evolution of metapopulations. Pages 183-210 in I. Hanski and M. E. Gilpin, editors. *Metapopulation biology: ecology, genetics, and evolution*. Academic Press, New York.
- Crozier, R. H. 1992. Genetic diversity and the agony of choice. *Biological Conservation* 61:11-15.
- Falconer, D. S. 1989. *Introduction to quantitative genetics*. Longman, London.
- Fisher, R. A. 1930. *The genetical theory of natural selection*. Clarendon Press, Oxford.
- Franklin, I. R. 1980. Evolutionary changes in small populations. Pages 135-149 in M. E. Soulé and B. A. Wilcox, editors. *Conservation biology: an evolutionary-ecological approach*. Sinauer Associates, Sunderland, Massachusetts.
- Hartl, D. L., and A. G. Clark. 1997. *Principles of population genetics*. Sinauer Associates, Sunderland, Massachusetts.
- Hedrick, P. W., and M. E. Gilpin. 1997. Genetic effective size of a metapopulation. Pages 165-181 in I. Hanski and M. E. Gilpin, editors. *Metapopulation biology: ecology, genetics, and evolution*. Academic Press, New York.
- Kimura, M., and G. H. Weiss. 1964. The stepping stone model of population structure and decrease of genetic correlation with distance. *Genetics* 49:561-576.
- Lacy, R. C. 1987. Loss of genetic diversity from managed populations: interacting effects of drift, mutation, immigration, selection, and population subdivision. *Conservation Biology* 1:143-158.
- Lande, R. 1995. Breeding plans for small populations based on the dynamics of quantitative genetic variation. Pages 318-340 in J. D. Ballou et al., editors. *Population management for survival and recovery: analytical methods and strategies for small population conservation*. Columbia University Press, New York.
- Lande, R., and G. F. Barrowclough. 1987. Effective population size, genetic variation, and their use in population management. Pages 87-123 in M. Soulé, editor. *Viable populations for conservation*. Cambridge University Press, New York.
- Loeschke, V., J. Tomiuk, and S. K. Jain. 1994. Introductory remarks: genetics and conservation biology. Pages 3-8 in V. Loeschke et al., editors. *Conservation genetics*. Birkhäuser Verlag, Basel, Switzerland.
- Lovejoy, T. E., R. O. Bierregaard, A. B. Rylands, J. R. Malcolm, C. E. Quintela, L. H. Harper, K. S. Brown, A. H. Powell, G. V. N. Powell, H. O. R. Schubart, and M. B. Hayes. 1986. Edge and other effects of isolation on Amazon forest fragments. Pages 257-285 in M. E. Soulé, editor. *Conservation biology: the science of scarcity and diversity*. Sinauer Associates, Sunderland, Massachusetts.
- Lynch, M. 1988. The divergence of neutral quantitative characters among partially isolated populations. *Evolution* 42:455-466.
- Lynch, M., and W. Gabriel. 1990. Mutation load and the survival of small populations. *Evolution* 44:1725-1737.
- Lynch, M., R. Bürger, D. Butcher, and W. Gabriel. 1993. The mutational meltdown in asexual populations. *Journal of Heredity* 84:339-344.
- Marshall, K. A. 1995. The effect of forest fragmentation on the genetic diversity of the little wood satyr butterfly (*Megisto cymela*): implications for conservation biology. M. S. thesis. Bowling Green State University, Bowling Green, Ohio.
- Meffe, G. K., and R. C. Vrijenhoek. 1988. Conservation genetics in the management of desert fishes. *Conservation Biology* 2:157-169, 244.

- Olesen, J. M., and S. K. Jain. 1994. Fragmented plant populations and their lost interactions. Pages 417-426 in V. Loeschke et al., editors. *Conservation genetics*. Birkhäuser Verlag, Basel, Switzerland.
- Simberloff, D. 1988. The contribution of population and community biology to conservation science. *Annual Review of Ecology and Systematics* 5:473-511.
- Slatkin, M. 1977. Gene flow and genetic drift in a species subject to frequent local extinctions. *Theoretical Population Biology* 236:787-792.
- Slatkin, M. 1987. Gene flow and the genetic structure of natural populations. *Science* 236:787-792.
- Wade, M. J., and D. E. McCauley. 1988. Extinction and recolonization: their effects on the genetic differentiation of local populations. *Evolution* 42:995-1005.
- Whitlock, M. C. 1992. Nonequilibrium population structure in forked fungus beetles: extinction, colonization, and the genetic variance among populations. *American Naturalist* 139:952-970.
- Whitlock, M. C., and D. E. McCauley. 1990. Some population genetic consequences of colony formation and extinction: genetic correlations within founding groups. *Evolution* 44:1717-1724.
- Whitlock, M. C., P. C. Phillips, and M. J. Wade. 1993. Gene interaction affects the additive genetic variance in subdivided populations with migration and extinction. *Evolution* 47:1758-1769.
- Wright, S. 1931. Evolution in Mendelian populations. *Genetics* 16:97-159.
- Wright, S. 1943. Isolation by distance. *Genetics* 28:114-128.
- Wright, S. 1951. The genetical structure of populations. *Annals of Eugenics* 15:323-354.
- Wright, S. 1969. Evolution and the genetics of populations. Volume 2. The theory of gene frequencies. University of Chicago Press, Chicago.

