2 Incorporating Stochasticity in Population Viability Analysis

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ABSTRACT

This chapter reviews recent advances in understanding how stochastic demographic and genetic factors affect population viability, defined by the probability of extinction during a given time interval. Stochastic fluctuations in population growth rate not only cause chance extinctions; they also produce a cumulative deterministic component that tends to decrease populations and drive them toward extinction. Environmental stochasticity decreases the long-run growth rate of a population when it is below carrying capacity, and demographic stochasticity can create a type of Allee effect or unstable equilibrium at small population size below which most population trajectories decline toward extinction. In fragmented habitats, the regional dynamics of a metapopulation interacts with the stochastic dynamics of local populations. For territorial species, there exists an extinction threshold or minimum proportion of suitable habitat necessary for metapopulation persistence. For nonterritorial species, habitat occupancy and metapopulation persistence depend strongly on the "rescue effect" and the "establishment effect," whereby immigrants to local populations decrease the rate of local extinction and increase the probability of successful colonization. Positive temporal and spatial autocorrelations of population fluctuations increase the risk of extinction. These autocorrelations depend on the temporal and spatial scales of environmental stochasticity and on the species' life history and dispersal pattern. Genetic stochasticity due to finite population size also produces deterministic or average reduction of genetic variance and adaptive potential, and loss of fitness through inbreeding depression and accumulation of new mildly deleterious mutations. The probability distribution of extinction times has an initial lag before a characteristic rate of extinction is achieved, which suggests that population viability analyses should consider time frames longer than the typical 100-year limit dictated by political and legal considerations, especially for species with long generations.

I thank I. Hanski for discussion, and S. Engen and B.-E. Sæther for comments on the manuscript. This work was supported by NSF grant DEB 9806363.

INTRODUCTION

All populations fluctuate stochastically, with coefficients of variation in annual census sizes usually in the range of about 20 to 80% (Pimm 1991). Stochastic fluctuations can drive a population or species to extinction even when its expected growth rate is positive at population sizes below carrying capacity. Following Shaffer (1981), population viability is generally defined in terms of the probability of extinction within a specified time interval. Shaffer described stochastic factors of demography and genetics that contribute to extinction risk. "Demographic stochasticity" is caused by random variation in individual fitness that is independent among individuals. This produces random fluctuations in mean fitness or population growth rate that are inversely proportional to population size. "Environmental stochasticity" caused by changes in physical or biological factors affects the fitness of all individuals in a population in a similar fashion. This produces random fluctuations in population growth rate regardless of population size. Catastrophes are sudden collapses in population size, caused by extreme environmental events such as droughts, floods, fires, and epidemics, often with a substantial random component in time occurrence (Young 1994), as well as possible periodic components (Beissinger 1995). Shaffer (1987) later included random catastrophes as the upper tail of a distribution of environmental stochasticity (cf. Erb and Boyce 1999).

Genetic stochasticity in finite populations, also known as random genetic drift, entails random changes in gene frequencies caused both by variance in family sizes and by Mendelian segregation of alleles (Wright 1969; Crow and Kimura 1970). Particularly in small populations, inbreeding due to mating between related individuals produces a random loss of alleles and a reduction of genetic variance required for adaptive evolution. Inbreeding on average increases the homozygosity of preexisting, partially recessive deleterious mutations, but by chance some can achieve appreciable frequencies in small populations. This causes "inbreeding depression," which is manifested as decreased individual fitness and population growth rate (Ralls and Ballou 1983; Falconer and Mackay 1996; Husband and Schemske 1996).

Here I review recent developments in understanding and modeling stochastic factors affecting the risk of population extinction, including (1) the relationship between stochastic demography and Allee effect (i.e., the reduction of expected growth rate in small populations), (2) the role of stochastic local dynamics in metapopulation persistence for territorial and nonterritorial species, (3) the temporal and spatial scales of environmental stochasticity and the synchrony of population fluctuations, and (4) genetic stochasticity, loss of adaptive potential and re-

duced fitness in small populations from fixation of both old and new mutations. I show that stochastic demographic and genetic factors have deterministic components or average effects with significant impacts on population viability. Finally, I discuss the probability distribution of extinction times and appropriate time spans for population viability analysis (PVA).

Despite the importance of stochastic factors, especially in small populations, it is important to realize that most populations initially become threatened or endangered because of deterministic human activities caused by human population growth and economic development, primarily habitat destruction and fragmentation, overexploitation, introduced species, and pollution (Groombridge 1992; Caughley 1994).

STOCHASTIC DEMOGRAPHY AND ALLEE EFFECTS

Stochastic fluctuations in population growth rate contribute to extinction risk for two reasons. Stochasticity not only causes random encounters with the "absorbing boundary" of extinction from which species cannot return; it also has a cumulative deterministic tendency to decrease populations and drive them toward extinction. This occurs because population growth is fundamentally a multiplicative process, and the long-run dynamics of population size are governed by the geometric mean growth rate (or expected rate of increase of the natural logarithm of population size), which is always less than the arithmetic mean growth rate (or expected per capita population growth rate). For example, under density-independent growth, when the population is well below carrying capacity but large enough to neglect demographic stochasticity, deterministic population dynamics in a constant environment are described by a continuous time model in which the rate of change of population size, N, with time, t, is given by $dN/dt = rN$, where r is the per capita growth rate or mean Malthusian fitness in the population. Environmental stochasticity causes r to fluctuate randomly in time with a mean $\bar{r}$ and environmental variance σ_e^2. The expected rate of increase of $\ln N$ (or "long-run growth rate") is $\bar{r} - \sigma_e^2/2$ (Tuljapurkar 1982; Lande and Orzack 1988; Lande 1993). Thus, environmental stochasticity creates a deterministic (or average) decrement in the long-run growth rate of a population. This is not an artifact of using the log scale, since simulation of stochastic discrete-time models demonstrates that surviving populations tend to grow more slowly than the deterministic rate that would occur in a constant, average environment $\bar{r}$ (fig. 2.1).

Demographic stochasticity produces similar effects in small populations. Denoting the demographic variance in individual fitness as σ_d^2, the

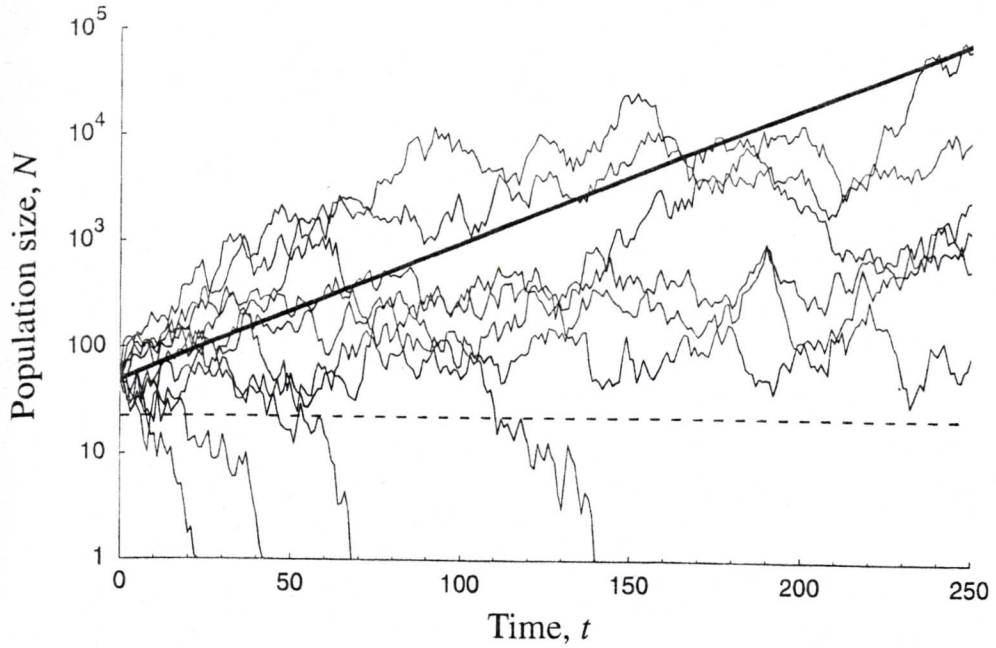

Fig. 2.1 Ten simulated trajectories of a population with initial size of 50 individuals subject to demographic and environmental stochasticity. Dynamics obey the simple discrete-time model $N_{t+1} = \lambda_t N_t$, where N_t is the population size in year t. At a given population size the finite rate of increase, λ_t, is (approximately) lognormally distributed with mean $\bar{\lambda} = 1.03$ and variance $\sigma_e^2 + \sigma_d^2/N_t$, where $\sigma_e^2 = 0.04$ is the environmental variance and $\sigma_d^2 = 1.0$ is the demographic variance. The *heavy line* gives the deterministic dynamics of geometric growth at the mean rate $\bar{\lambda}$. The *dashed line* marks the unstable equilibrium size N^* below which population trajectories tend to decrease rapidly toward the extinction boundary at $N = 1$ (see text).

variance in mean fitness or population growth rate, r, caused by demographic stochasticity is σ_d^2/N. Under both demographic and environmental stochasticity at a given population size well below carrying capacity, r in the above model fluctuates with a mean $\bar{r}$ and variance $\sigma_e^2 + \sigma_d^2/N$ (Leigh 1981; Lande 1993; Sæther et al. 2000). The expected rate of increase of ln N is $\bar{r} - \sigma_e^2/2 - \sigma_d^2/(2N)$ (cf. Lande 1998a). Thus, in addition to causing random fluctuations in population size, particularly in small populations, demographic stochasticity also creates a deterministic decrement in the long-run growth rate that is inversely proportional to population size. With sufficient demographic stochasticity, the long-run growth rate can become negative in small populations.

With both demographic and environmental stochasticity, a generalized scale transformation that resembles ln N for large populations and $\sqrt{N}$ for small populations is necessary to analyze the probabilistic tendencies of population trajectories (Lande 1998a). On this transformed

scale, demographic stochasticity creates an unstable equilibrium at a value corresponding to the population size

$$N^* = \frac{\sigma_d^2/4}{\bar{r} - \sigma_e^2/2}.$$

Below a population size of N^*, most population trajectories tend to decrease toward extinction. Again, this is not an artifact of the scale transformation, as simulations of stochastic discrete-time models demonstrate that populations below N^* tend to decrease and become rapidly extinct (fig. 2.1).

The existence of an unstable equilibrium on this transformed scale bears a close resemblance to the classical Allee effect (Allee et al. 1949). Allee effects usually are defined as a deterministic decrease in individual fitness (and hence a decrease in mean fitness or population growth rate) due to a failure of cooperative interactions among individuals in small or sparsely distributed populations. Some common mechanisms for Allee effects include group foraging, group defense against predators, cooperative breeding, chemical or physical conditioning of the environment (e.g., huddling for warmth during winter), and the difficulty of finding mates (Courchamp et al. 1999). Genetic stochasticity in small populations also produces similar effects through inbreeding depression and through random fluctuations in sex ratio in species with genetic sex determination. Both classical Allee effects and the deterministic components of demographic and genetic stochasticity can cause populations below a certain size to decline rapidly to extinction.

Thus, classical Allee effects, demographic stochasticity, and genetic stochasticity may be indistinguishable in terms of their effects on the dynamics of small populations. Distinguishing them generally will require detailed studies of the behavioral, ecological, and genetic factors affecting fitness in small populations. Statistical methods for joint estimation of demographic and environmental stochasticity and uncertainty in population parameters are described in Engen et al. (1998), Kendall (1998), and Sæther et al. (1998, 2000).

METAPOPULATIONS WITH STOCHASTIC LOCAL DYNAMICS

Metapopulation concepts have become popular for analyzing the effects of habitat fragmentation on populations in which regional persistence is maintained by a balance between local extinction and colonization (Levins 1969, 1970; Hanski and Gilpin 1997). Assuming that equivalent patches of suitable territory are either occupied or unoccupied by a spe-

cies, and that the local extinction rate (e) and the colonization rate (c) are constant, Levins showed that the proportion of suitable habitat patches occupied at equilibrium is $\hat{p} = 1 - e/c$. Thus, regional persistence of a metapopulation is possible ($\hat{p} > 0$) only when the colonization rate exceeds the local extinction rate ($c > e$). This and subsequent metapopulation models reveal that a metapopulation may become extinct in the presence of suitable habitat and that currently unoccupied suitable habitat may be critical for long-term persistence of a species.

However, most metapopulation models still make several of the same simplifying assumptions as in the original model of Levins (1969, 1970). Ignoring the internal dynamics within local populations fails to consider any coupling between local and global dynamics, which is known to be important through the "rescue effect" of immigrants reducing the local extinction rate (Brown and Kodric-Brown 1977). Most metapopulation models contain no description of the amount of suitable versus unsuitable habitat, which precludes their use in predicting effects of either continued habitat destruction and fragmentation or habitat improvement. This section reviews two metapopulation models that relax these simplifying assumptions for territorial species and nonterritorial species.

For territorial species in which individual females or mated pairs have exclusive or largely nonoverlapping territories or home ranges, Lande (1987) developed a metapopulation model that incorporated life history, individual dispersal behavior, and an explicit description of the amount of suitable habitat in a region. Patches of habitat the size of individual territories are assumed to be either suitable or unsuitable for survival and reproduction, and suitable habitat patches are randomly or evenly distributed over a large region such that suitable habitat is not clumped on a scale larger than the typical individual dispersal distance. The proportion of suitable habitat in the region is h. Because individual territories are identified as the unit of suitable habitat, local extinction corresponds to the death of an individual female, and colonization corresponds to settlement of a dispersing juvenile on an unoccupied suitable territory. The most basic model incorporates classical female-biased demography with age structure, assuming that all females are successfully mated. Juveniles disperse prior to reproduction, and their survival is density-dependent, based on the probability of finding a suitable unoccupied territory among a maximum number of potential territories they can search before dying from starvation or predation. The proportion of suitable habitat occupied at equilibrium takes the simple form

$$\hat{p} = 1 - (1 - k)/h.$$

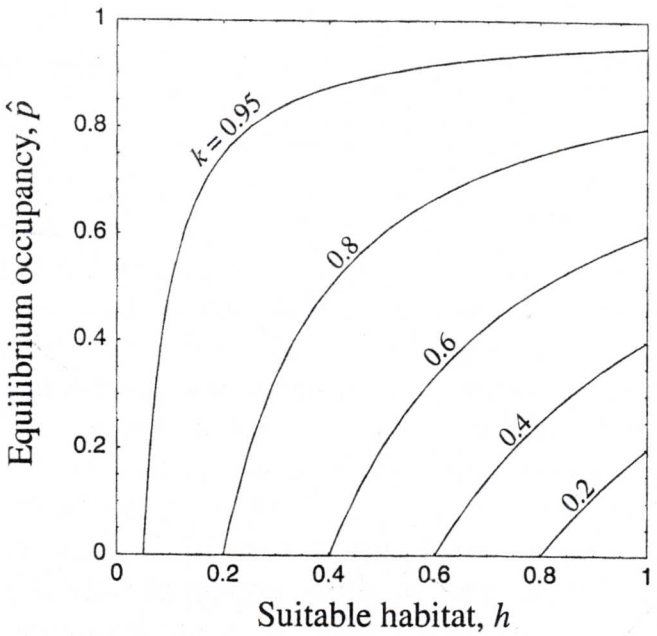

Fig. 2.2 Equilibrium occupancy of suitable habitat, $\hat{p}$, in a metapopulation model for a territorial species as a function of the proportion of suitable habitat, h, randomly or evenly distributed in a region, for different values of the demographic potential, k (modified from Lande 1987).

All information on the life history and dispersal behavior is incorporated in the composite parameter k, termed the "demographic potential" because it gives the maximum proportion of habitat occupied at equilibrium in a completely suitable region ($\hat{p} = k$ when $h = 1$). Even in a completely suitable region, not all habitat is occupied, because some time elapses before a territory vacated by the death of a resident female is settled by a dispersing juvenile. As the amount of suitable habitat decreases in the region, the equilibrium occupancy declines, eventually reaching an "extinction threshold" or minimum proportion of suitable habitat necessary to sustain the population ($h = 1 - k$). For species with high demographic potential, the equilibrium occupancy declines precipitously as the amount of suitable habitat decreases toward the extinction threshold. The equilibrium population size is proportional to the product of the amount of suitable habitat (h) and the equilibrium occupancy of suitable habitat ($\hat{p}$). Hence, the equilibrium population size declines faster than the rate of habitat destruction until the extinction threshold is reached (fig. 2.2). When habitat destruction and fragmentation occur on the same time scale as the generation time of a species, there may be little warning that the extinction threshold is being approached if the decline in population size lags behind the demographic equilibrium.

This metapopulation model for a territorial species was originally applied to data on the demography and habitat of the northern spotted owl (*Strix occidentalis caurina*) in the Pacific Northwest of the United States. It suggested that plans by the U.S. government to preserve this subspecies were seriously inadequate due to excessive habitat destruction and fragmentation. This became critical scientific evidence in litigation that eventually led to greatly increased protection of old-growth forests on which this subspecies depends (Lande 1988). Subsequent analyses using more detailed spatial information confirmed the generality of extinction thresholds in models of habitat fragmentation (Doak 1989; Nee and May 1992; Lamberson and Carroll 1993; McKelvey et al. 1993; Bascompte and Sole 1996; Hill and Caswell 1999; With and King 1999).

Incorporating stochastic dynamics of local populations into metapopulation models of nonterritorial species has proven much more difficult. Some initial results were derived by Lande et al. (1998), who modeled demographic and environmental stochasticity, and stochastic dispersal among a finite number of local populations. This approach allows local extinction and colonization rates to be derived from local population dynamics and permits analysis of the coupling between local population dynamics and metapopulation dynamics. Coupling of local and global dynamics occurs because increasing occupancy of suitable habitat in the metapopulation increases the rate of immigration into local populations. This produces the well-known "rescue effect" (Brown and Kodric-Brown 1977), whereby immigration decreases the rate of local extinction. It also produces an "establishment effect" (Lande et al. 1998), whereby continued immigration can greatly increase the probability of successful colonization during the critical initial phase, when a few individuals struggle to overcome demographic and environmental stochasticity (fig. 2.3). These effects can combine to create multiple equilibria for habitat occupancy. This includes a kind of metapopulation Allee effect or unstable equilibrium at low habitat occupancy below which the metapopulation cannot persist, as suggested by Hanski and Gyllenberg (1993), based on simple phenomenological models.

Infinite metapopulation models with an unlimited number of local populations produce deterministic dynamics of habitat occupancy (Levins 1969, 1970; Hanski and Gyllenberg 1993). In contrast, finite metapopulation models allow estimation of the risk of metapopulation extinction by stochastic local extinction and colonization. Accounting for stochastic dynamics within local populations and the coupling of local and global dynamics by the rescue and establishment effects can greatly increase metapopulation viability compared to classical metapopulation

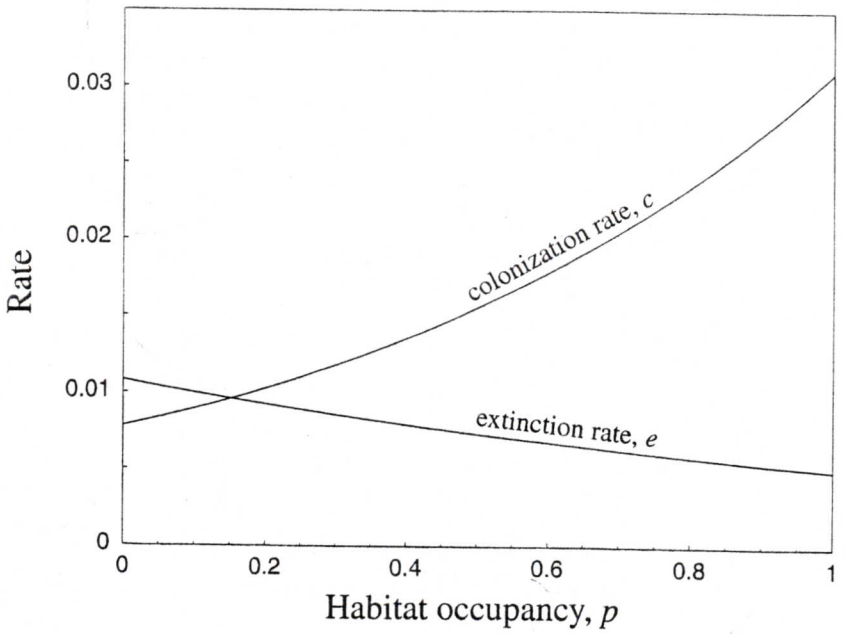

Fig. 2.3 Local extinction and colonization rates, e and c, as a function of the occupancy of suitable habitat, p, in a metapopulation model for a nonterritorial species. Decrease in the local extinction rate and increase in the colonization rate with increasing p (and higher immigration into local populations) are, respectively, the rescue effect and the establishment effect. Expected local dynamics are logistic with carrying capacity $K = 50$, mean intrinsic rate of increase $\bar{r} = 0.01$, environmental and demographic variances $\sigma_e^2 = 0.01$ and $\sigma_d^2 = 1.0$, and a low individual migration rate $m = 0.005$ (modified from Lande et al. 1998).

models that assume constant rates of local extinction and colonization (Lande et al. 1998).

TEMPORAL AND SPATIAL SCALES OF ENVIRONMENTAL STOCHASTICITY

Temporal and spatial autocorrelations in environmental stochasticity can have major impacts on population viability but often are ignored both because of the difficulty of estimating them from limited data usually available on endangered species and because these topics have only recently received attention by ecologists. Autocorrelations begin at unity for populations separated by zero time and distance, generally decline with increasing time or distance, and may be negative at some times or distances. The temporal and spatial scales of autocorrelation in environmental stochasticity determine appropriate methods for modeling and incorporating them in PVA.

Positive temporal autocorrelation in environmental stochasticity increases extinction risk, as demonstrated by several theoretical studies

(Turelli 1977; Foley 1994, 1997; Lande et al. 1995; Ripa and Lundberg 1996; Palmqvist and Lundberg 1998). This occurs because positive autocorrelation produces runs of years with consistently high or low population growth rates. Runs of years with negative growth rates can drive a population extinct, whereas runs of years with positive growth rates are damped by density dependence. The simplest method for modeling temporal autocorrelation is to multiply the environmental variance by the sum or integral of the autocorrelation function (Turelli 1977; Foley 1994, 1997; Lande et al. 1995). For positive autocorrelation, this effectively increases environmental stochasticity and the corresponding extinction risk. This approach is accurate only if the temporal scale of environmental stochasticity is not very long; otherwise, it is necessary to explicitly model the temporal autocorrelation in environmental stochasticity.

Empirical studies of autocorrelation usually deal directly with population size rather than growth rate (Pimm 1991; Halley 1996). Temporal autocorrelation in population size can also result from lags caused by age structure of populations (Lande and Orzack 1988). Studies of temporal autocorrelation in adult or total population size typically do not separate the contributions from environmental autocorrelation and life history (for an exception see Ratner et al. 1997).

Positive spatial autocorrelation in environmental stochasticity also increases extinction risk, as demonstrated in simulations of classical metapopulations with spatially correlated local extinctions (Harrison and Quinn 1989; Gilpin 1990), and simulations of metapopulations with spatially correlated environmental stochasticity in local dynamics (Heino et al. 1997; Heino 1998). This occurs because synchronized fluctuations in the sizes of local populations cause them to become extinct simultaneously, which increases the risk of regional or global extinction.

Spatial autocorrelation in population size, or "population synchrony," results from a combination of spatial autocorrelation in environmental stochasticity and localized individual dispersal. Several studies have attempted to clarify the relative contributions of environmental stochasticity and individual dispersal to population synchrony (Ranta et al. 1995, 1997a,b). Comparisons of related species have shown that population synchrony is higher over short distances for species with greater powers of dispersal. There often is a long-distance component of population synchrony on a scale much greater than the individual dispersal distance, which usually is attributed to environmental autocorrelation (Hanski and Woiwod 1993; Sutcliffe et al. 1996; Lindstrom et al. 1996).

Lande et al. (1999) analyzed a population continuously distributed in space, with environmental stochasticity caused by temporal fluctua-

tions in the intrinsic rate of increase or carrying capacity of local populations, assuming the environmental stochasticity was spatially (but not temporally) autocorrelated. Employing the standard deviation of a function in a given direction as a measure of scale, for small or moderate fluctuations in local population size the spatial scale of population synchrony (l_p) is related to the spatial scales of environmental correlation (l_e) and individual dispersal (l) by the simple general formula $l_p^2 = l_e^2 + ml^2/\gamma$, where m is the individual dispersal rate and γ is the strength of population density regulation (or rate of return to equilibrium, $\bar{r}$, in the logistic model). Relative to environmental autocorrelation, the contribution of individual dispersal to the spatial scale of population synchrony is magnified by the ratio of the individual dispersal rate to the strength of density regulation. Even when the scale of individual dispersal is less than that of environmental autocorrelation, dispersal can substantially increase the spatial scale of population synchrony for weakly regulated populations with low $\bar{r}$. This happens because weaker density regulation allows fluctuations in local population size to build up and spread farther by individual dispersal before they are damped by density dependence.

Many threatened and endangered species are characterized by diminished values of $\bar{r}$ due to overexploitation, habitat degradation, introduced species, and pollution (Groombridge 1992; Caughley and Gunn 1996). This not only contributes to extinction risk by making local populations more susceptible to stochastic declines, but also increases population synchrony and the risk of regional or global extinction.

GENETIC STOCHASTICITY

Incorporating genetic stochasticity into PVA requires that its effects be expressed in terms of population dynamics and extinction risk. Population geneticists have developed models of stochastic evolution in finite populations of constant size, but work on the interactions between stochastic genetics and demography has barely begun. The great complexity of the genetics of finite populations makes this a daunting task because thousands of genes affect fitness. Realistic genetic models are, therefore, far more complex than most demographic models and difficult to accurately parameterize for particular species. This section reviews some recent progress in understanding the impacts of genetic stochasticity on population viability.

Genetic Variance, Adaptive Evolution, and Population Persistence in Changing Environments

Two major mechanisms of population persistence in response to major environmental change are evolution and change of geographic range.

In unfragmented habitats, theory suggests that change of geographic range is the primary mechanism of population persistence in a changing environment. Even though local populations at a fixed spatial location may evolve rapidly, the areas of highest population density move through space and time to track the environmental conditions to which a species is already adapted, so that the population as a whole maintains a nearly constant phenotype (Pease et al. 1989). This occurs through (1) active habitat selection, by individual movement along environmental gradients toward the optimal microenvironments for a species, and (2) passive habitat selection, in which local populations grow in areas to which they are well adapted and decline in areas where they are poorly adapted. Paleontological observations confirm that species often changed their geographic range in the past in response to glacial cycles while maintaining a relatively constant phenotype except for body-size evolution (Coope 1979; Smith et al. 1995).

Habitat destruction and fragmentation restrict dispersal and reduce or eliminate the ability of species to alter their geographic range (Peters and Lovejoy 1992). Species restricted to isolated habitat fragments and reserves can rely only on their limited physiological tolerances, or on evolutionary adaptation in situ, to survive rapid global warming and other environmental challenges in the coming centuries. The persistence of many species will depend increasingly on maintaining sufficient genetic variance for adaptive evolution.

Finite population size produces stochastic changes in gene frequencies known as random genetic drift, due to Mendelian segregation and variance in family size, which tends to reduce genetic variation. In the absence of natural selection, a fraction of $1/(2N_e)$ of the genetic variance (either heterozygosity or the heritable component of variance in quantitative characters) is expected to be lost from a population per generation, where N_e is the effective population size. The effective size of wild populations generally is substantially less than the actual size because of variance in family size, unequal sex ratio, and temporal fluctuations in population size (Wright 1969; Waples, chap. 8 in this volume). The ratio of effective to actual size of wild populations is often on the order of 0.1 to 0.2 (Frankham 1995; Waples, chap. 8 in this volume). To be expected to lose a large fraction of its genetic variance, a population reduced to a small N_e must remain small for at least $2N_e$ generations (Wright 1969). The genetic effects of such a population bottleneck are similar to those produced by frequent local extinction and colonization, which can reduce N_e of a metapopulation orders of magnitude below its actual size (Wright 1940; Maruyama and Kimura 1980; Hedrick 1996).

Based on estimates of mutability in quantitative characters (Lande 1976; Lynch 1988), Franklin (1980) and Soulé (1980) recommended a minimum N_e of 500 to maintain typical levels of heritable variance. Recent experiments indicate that a large fraction of the mutational variance in quantitative characters is associated with recessive lethal and semilethal side effects such that the quasi-neutral, potentially adaptive fraction of mutational variance is about one-tenth as large as previously thought (Mackay et al. 1992; Lopez and Lopez-Fanjul 1993a,b). Lande (1995) suggested that the Franklin-Soulé number should be increased by a factor of ten (but see Franklin and Frankham 1998). Much larger populations may be required to maintain rare alleles with major effects on disease resistance (Lande 1983; Roush and McKenzie 1987). In a relatively constant environment, however, there may be little need for adaptive evolution. Several examples exist of populations or species that recovered after reduction to small numbers, such as the northern elephant seal (*Mirounga angustirostris*; Hoelzel et al. 1993) and American bison (*Bison bison*; Miller 1990, 38–39). Following a population bottleneck and recovery to large population size, genetic variance can be replenished by spontaneous mutation, which occurs more rapidly for quantitative characters than for single-locus molecular polymorphisms (Lande 1976, 1995).

Much of adaptive evolution is based on quantitative (continuously varying) polygenic characters of morphology, behavior, and physiology. Quantitative characters usually are subject to stabilizing natural selection toward an intermediate optimum phenotype that may fluctuate with time, with phenotypes that deviate from the optimum having reduced fitness. Heritable variance in quantitative characters, therefore, imposes a fitness decrement or "genetic load" on a population, which like deleterious mutation is an inevitable cost of maintaining adaptive potential (Crow and Kimura 1970; Lande and Shannon 1996). Thus, there is an optimal level of genetic variance for maintaining both current fitness and future adaptability. When environmental change is partially predictable (i.e., when the optimal phenotype undergoes prolonged directional change, long-period high-amplitude cycles, or substantial temporal autocorrelation), then genetic variance in quantitative characters increases mean fitness and promotes population persistence (Lande and Shannon 1996). Even for very large populations, however, there is a maximum rate of directional or random environmental change to which a population can adapt without becoming extinct, depending on the amount of genetic variability maintained (Lynch and Lande 1993; Bürger and Lynch 1995; Gomulkiewicz and Holt 1995; Lande and Shannon 1996).

Inbreeding Depression and Fixation of New Mildly Deleterious Mutations

Matings between related individuals tend to reduce offspring viability and fertility due to the homozygous expression of (partially) recessive deleterious mutations, which is known as inbreeding depression in fitness. In historically large, outcrossing populations, a 10% increase in the inbreeding coefficient typically reduces fitness by a few to several percent. This applies for domesticated species as well as experimental populations of fruit flies (*Drosophila melanogaster*) and other species recently isolated from the wild (Falconer and Mackay 1996). Data on captive populations of many wild animal species suggest similar values (Ralls and Ballou 1983). Continued brother-sister mating in domesticated animals generally results in extinction of a high proportion of lines within five or ten generations (Soulé 1980). Substantial heterogeneity exists among species and populations in the magnitude of inbreeding depression (Soulé 1980; Lacy et al. 1993; Husband and Schemske 1996).

The genetic basis of inbreeding depression is best understood in *Drosophila* species, with roughly equal contributions from nearly recessive lethal mutations and from more nearly additive mildly deleterious mutations (Simmons and Crow 1977). Both types of mutations arise at thousands of loci throughout the genome in eukaryotic species (Simmons and Crow 1977). Gradual inbreeding allows natural selection to purge recessive lethal mutations from a population as they become expressed in homozygotes, but inbreeding has little or no effect on the efficiency of selection against nearly additive or additive, mildly deleterious mutations (Lande and Schemske 1984; Charlesworth and Charlesworth 1987). For populations with extremely high inbreeding depression, such as some tree species and gynodioecious plants, it may be difficult for close inbreeding to purge recessive lethals because, if nearly all the inbred offspring die before reproduction, the population is effectively outcrossed (Lande et al. 1994).

Sudden reduction to very small population size generally produces substantial inbreeding depression, unless the population quickly grows to a large size that allows natural selection to reverse the short-term effects of inbreeding and random genetic drift (Keller et al. 1994). The more gradual the reduction in population size, the greater the opportunity for purging recessive lethal mutations and avoiding a large part of the inbreeding depression. Therefore, inbreeding depression is not simply proportional to the standard inbreeding coefficient for selectively neutral genes, as was assumed in recent models of the interaction of stochastic demography with inbreeding (e.g., Mills and Smouse 1994). The rule suggested by Franklin (1980) and Soulé (1980), based

on extensive data from animal and plant breeding, is that most inbreeding depression can be avoided in populations with $N_e > 50$. However, inbreeding depression may be more severe in natural than in artificial environments (Jiménez et al. 1994), and more severe in stressful than in optimal environments (Keller et al. 1994; Bijlsma et al. 1997).

Saccheri et al. (1998) showed that in a butterfly metapopulation the rate of extinction of local populations consisting of only a few families was more closely correlated with local heterozygosity than with local population size. This, combined with previous experiments demonstrating a very high inbreeding depression in the species (Saccheri et al. 1996), was used to suggest that genetics was of greater importance than demography in contributing to local extinctions. Such analyses should be viewed with caution, because heterozygosity may be a better indicator of effective population size and the tendency for local population fluctuation over time scales of $2N_e$ generations than direct observations of recent population sizes. However, in this particular case the conclusion is likely to be valid, as the mean persistence time of local populations is only a few years (I. Hanski, personal communication).

Inbreeding depression due to fixation of deleterious partially recessive mutations can be reversed, at least temporarily, by introducing genes from unrelated individuals into an inbred population, which allows natural selection to eliminate the deleterious mutations. It can be permanently prevented by continued immigration every one or two generations of a single unrelated individual into each local breeding population regardless of its size (Lande and Barrowclough 1987). Such a plan was recently implemented for the endangered Florida panther, motivated by strong circumstantial evidence of inbreeding depression and its low genetic divergence from other conspecific populations. Such genetic augmentation may be sufficient to reverse inbreeding effects and not too high to swamp possible local adaptations (Hedrick 1995). Nevertheless, the population still faces demographic threats from small size caused by past habitat destruction.

In contrast to recessive lethal mutations that generally are restricted to low frequencies by natural selection, random genetic drift can fix mildly deleterious mutations in a small population. Weakly selected genes become effectively neutral if the magnitude of selection on them is much less than $1/(2N_e)$ (Wright 1969). In the long run, nearly neutral mutations, with selection coefficients close to $1/(2N_e)$, do the most damage to a population, because strongly selected mutations rarely become fixed and selectively neutral mutations are easily fixed by random genetic drift but have no impact on fitness (Lande 1994; Lynch et al. 1995a,b). The total genomic rate of mildly deleterious mutations is on the order

of one per generation in a variety of organisms. Such mutations reduce fitness on average by a few to several percent and are only partially dominant (nearly additive). After fixation of enough mildly deleterious mutations, the population becomes genetically inviable ($\bar{r} \leq 0$), and extinction rapidly ensues. For a population at carrying capacity in a constant environment with no demographic stochasticity, the mean time to reach genetic inviability from fixation of new deleterious mutations is (asymptotically) proportional to K^{1+1/c^2}, where K is the carrying capacity and c is the coefficient of variation of selection against new mutations (Lande 1994, 1995). Realistic distributions of selection on mildly deleterious mutations have a value for c on the order of one, as for an exponential distribution (Keightley 1994).

For populations with initially high mean fitness, even after reduction to very small numbers, hundreds of generations must elapse before fixation of new mildly deleterious mutations causes extinction (Lande 1994). In large populations, advantageous, compensatory, and reverse mutations can completely prevent the erosion of fitness from fixation of deleterious mutations (Lande 1994, 1998b; Schultz and Lynch 1997). Extinction from fixation of new deleterious mutations is, therefore, a serious concern within the typical 100-year time scale of conservation planning only for small populations with initially low mean fitness. For populations of moderate size, however, with N_e up to a few hundred or a few thousand, fixation of new mutations could substantially erode their mean fitness and decrease their long-term viability (Lande 1995, 1998b).

TIME FRAMES FOR PVAs

The acceptable level of extinction risk in terms of the time frame and the corresponding probability of extinction is ultimately a social (Shaffer 1981) or practical (Goodman, chap. 21 in this volume; Ludwig and Walters, chap. 24 in this volume), rather than scientific, decision. For legal and political reasons, the smallest extinction risk usually considered in classifying endangered species is a 10% chance of extinction within 100 years corresponding to the "vulnerable" category of the Red Lists of the World Conservation Union (IUCN 1994). If all species of conservation concern were managed to this minimum viability level, then within the next millennium a fraction of $1 - 0.9^{10}$, or about 65% of them, would likely go extinct. This exceeds by a factor of thousands the normal background rate of extinction for abundant species that appear in the fossil record, which typically persist for millions of years (Van Valen 1973; Jablonski 1986).

Stochastic fluctuation in population size creates a probability distribution of extinction times. Theory indicates that for populations with a

positive long-run growth rate below carrying capacity, the distribution of extinction times has a lag period until a quasi-stationary distribution of population sizes is established, after which there is a relatively long phase with a nearly constant rate of extinction. Thus, after some lag, the distribution of extinction times is nearly exponential (Nobile et al. 1985; Goodman 1987; Ludwig 1996). If the initial population size is near carrying capacity, the lag period has a relatively low extinction rate. If the initial population size is far below carrying capacity, however, the lag period may begin with a short interval of low extinction rate followed by a burst of high extinction rate. Even populations with a negative long-run growth rate generally take some time to become extinct. Such lags are especially problematic for species with long generations if the standard population viability criteria of a 10% chance of extinction within 100 years is applied blindly. For example, Sukumar and Santiapillai (1993) suggested that a population of 30 Asian elephants (*Elephas maximus*) with demographic parameters producing a negative long-run growth rate should be considered viable because it satisfied these standard criteria. However, 100 years is only a few elephant generations. Using the same demographic parameters, extending the time frame to 200 years or longer beyond the lag period, it was shown that the cumulative probability of extinction is rather high (Armbruster et al. 1999). Thus, PVAs should always consider a range of time frames, including some much longer than those dictated by political and legal considerations. Although serious statistical difficulties exist in making long-term projections (Ludwig 1996, 1999; Fieberg and Ellner 2000), uncertainties are likely to accumulate more slowly with time for species with long generations than for species with short generations.

If we are to have any lasting effect in reducing the ongoing mass extinction that is expected to rival the effects of the major asteroid impact 65 million years ago, or a full-scale nuclear war, conservation plans must encompass longer time frames and lower probabilities of extinction. Conservationists should increasingly be concerned not only with threatened and endangered species and establishment of reserves, but also with maintaining and restoring abundant, healthy populations and functional ecosystems in the matrix between reserves. The proliferation of threatened and endangered species makes it increasingly obvious that effective conservation and restoration plans must be done on a landscape and ecosystem level (Scott et al. 1993; Kiester et al. 1996). In addition to monitoring ecosystem function and species diversity, large-scale conservation and restoration plans should always incorporate PVA of ecologically important, sensitive, or indicator species to confirm and

monitor the efficacy of large-scale plans. PVA should therefore remain an important endeavor as long as conservation biologists exist.

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