In "Ecology, Genetics, and Evolution of Metapopulations" I. Hanski + O.E. Gaggiotti eds. Elsevier, 2004

14. MECHANISMS OF POPULATION EXTINCTION

Oscar E. Gaggiotti and Ilkka Hanski

14.1 INTRODUCTION

Population ecologists have traditionally been concerned with questions about population regulation and the mechanisms that increase population stability (Elton, 1949; Nicholson, 1954, 1957; Milne, 1957, 1962; Andrewartha, 1957; den Boer, 1968; Andrewartha and Birch, 1984; Sinclair, 1989; Hanski, 1990b; Price and Cappuccino, 1995; Turchin, 1995, 2003). Population ecologists tended to study large populations, often of recognized "pest" species, which appeared to exhibit great persistence. In fact, until the early 1960s the predominant view in population ecology considered population extinctions unlikely in the presence of effective population regulation, wide dispersal, and generally large population sizes. This view predominated because little attention was paid to the actual spatial structure of populations (Allee et al., 1949). Notable exceptions were three Australian ecologists who recognized the possibility of small populations with high rate of extinction, although they reached this conclusion for entirely different reasons. Nicholson (1957), the principal architect of the population regulation paradigm, envisioned spatially structured populations and extinctions of small local populations, but principally in the case of host–parasitoid dynamics with strong density dependence leading to oscillations with increasing amplitude and, therefore, to local extinction (Nicholson, 1933). In contrast, Andrewartha and Birch (1954), who were not impressed by the

Copyright 2004, Elsevier, Inc.
0-12-323448-4

effectiveness of population regulation in preventing extinctions, developed proto-metapopulation ideas of large-scale persistence of species with ephemeral local populations (see discussion in Hanski, 1999b). One of the more influential studies that gradually changed ecologists' views about the spatial structure and dynamics of populations was Ehrlich's study on the checkerspot butterfly *Euphydryas editha* in California, showing apparently independent dynamics of similar populations over short distances in the absence of obvious density dependence (Ehrlich, 1961, 1965; Singer, 1972). The checkerspot studies are noteworthy for having addressed both ecological and genetic processes in local populations and for having contributed many insights about the processes of population extinction for the last four decades (for a comprehensive review, see Ehrlich and Hanski, 2004).

The perspective in population biology changed greatly in the 1970s in the wake of the emergence of modern conservation biology and its emphasis on questions about reserve design and population viability (Simberloff, 1988; Hanski and Simberloff, 1997). Questions about reserve design stemmed from the dynamic theory of island biogeography (MacArthur and Wilson, 1963, 1967), which, of course, was explicitly concerned with population extinctions. Early analyses of population viability in conservation biology emphasized genetic factors, inbreeding and drift (Foose, 1977; Chesser et al., 1980; Soulé and Wilcox, 1980; Frankel and Soulé, 1981; O'Brien et al., 1983; Schonewald-Cox et al., 1983; Soulé 1986). In the late 1980s, increasing recognition of habitat loss and fragmentation as the main threats to biodiversity (Wilson, 1988, 1989; Reid and Miller, 1989; Groombridge, 1992; Ehrlich and Daily, 1993) contributed to the growth of metapopulation biology (see Fig. 1.2 in Chapter 1), with emphasis on the spatial structure of populations and on the often high rate of extinction of small local populations (Gilpin and Hanski, 1991; Hanski and Gilpin, 1997; Hanski, 1999b).

The relative importance of ecological versus genetic factors in population extinction has been the subject of controversy ever since the birth of modern conservation biology. As already mentioned, conservation biology emerged as a discipline on two foundations, the island theory and the vision of population extinction due to genetic deterioration. Lande's (1988) influential paper reviewed the issue 15 years ago. He concluded that focusing primarily on genetic mechanisms of extinction was misguided and would not provide an adequate basis for understanding the processes underpinning the survival of endangered species. He also stressed the need for a realistic integration of demography and population genetics that would be applicable to species in their natural environments. Following the publication of this paper, a consensus started to form supporting the primary role of ecological factors in extinction. This consensus was later challenged by a series of theoretical studies (see later) of the decrease in fitness due to the accumulation of deleterious mutations ("genetic meltdown"). These analyses suggested that even relatively large populations might go extinct due to genetic deterioration. Undoubtedly, it has been difficult to reach a robust understanding about the mechanisms of population extinction because of the multitude of factors involved and the likely interactions among them, including ecological and genetic factors. Despite these difficulties, there has been substantial progress in this area during the last decade.

Although it is appropriate to emphasize interactions among different kinds of mechanisms influencing population extinction, it is practical to start with a review of particular ecological and genetics factors, which is done in Sections 14.2 and 14.3. One way of integrating the different factors is to relate them to the most important correlate of extinction risk, small population size. A common surrogate of local population size in metapopulation studies is the size of the habitat fragment in which the population occurs. Effects of population size and habitat patch size on extinction risk are reviewed in Section 14.4. The range of significant extinction mechanisms is expanded further when we consider local extinction in the metapopulation context (Section 14.5) and extinction of entire metapopulations (Section 14.6). Some challenges for further research are discussed in Section 14.7.

It is customary in reviews like the present one to make the point that the reasons why populations and species are currently going extinct at a distressingly high rate have primarily to do with loss of habitats and interactions with species that humans have displaced around the globe. This is what Caughley (1994), in an influential paper, called the declining-population paradigm. In contrast, most of the factors reviewed in this chapter belong to Caughley's (1994) small-population paradigm and relate to the ecological and genetic mechanisms that render the persistence of small populations precarious even without any added threats introduced by humans. A major exception is metapopulation theory, which can be employed to elucidate the risk of metapopulation extinction due to habitat loss and fragmentation (examined in Chapter 4; see also Chapter 2 on landscape ecology). It is important to realize that such a distinction can be made, but it is equally important to realize that, to some extent, Caughley's (1994) dichotomy is false (Hedrick et al., 1996; Holsinger, 2000). The dichotomy between small-population and declining-population paradigms is partly false because mechanisms in the two realms interact. This is especially apparent in the context of metapopulation biology, where our interest is focused on species with spatially structured populations, often consisting of many small local populations even if the metapopulation as a whole is large. To properly understand the dynamics and population biology of such species, we need to understand the mechanisms of extinction of the local populations that are often small. The main objectives of this chapter are to provide an update on the status of our understanding of these issues and to outline avenues of future research that could help improve it.

14.2 POPULATION EXTINCTION: ECOLOGICAL FACTORS

Demographic and Environmental Stochasticities

The classic models of population dynamics are deterministic and of little use in the study of population extinction, except in making the trivial but hugely important point that if the population growth rate r is negative, the population will surely, and rather quickly, go extinct. This is important because the human onslaught on the environment introduces changes, such as habitat loss and alteration, and spreading of invasive species, which will make r negative in many

populations. In deterministic models without age structure, the time to extinction from initial population size N_0 (which is assumed to be much below the carrying capacity) is given by $-\ln N_0/r$ (Richter-Dyn and Goel, 1972). In contrast, populations with $r > 0$ will not go extinct in simple deterministic models.

Deterministic models are inadequate for real populations because their dynamics are influenced by stochastic effects. It is useful to distinguish between two forms of stochasticity. *Demographic stochasticity* is due to random independent variation in the births and deaths of individuals. *Environmental stochasticity*, in contrast, is generated by random effects affecting all individuals in the population similarly. The label "environmental" signifies that the effects are caused by the shared environment of the individuals in the same population, such as adverse weather effects increasing mortality. These are the exogenous factors of population ecologists (Turchin, 2003).

In line with the two forms of stochasticity maintaining fluctuations in population size, the variance in the change in population size ΔN conditioned on population size N may be partitioned into two components, which are demographic and environmental variances (Engen et al., 1998). Assuming that these components are constant and denoting them by σ_d^2 and σ_e^2, respectively, $\mathrm{var}(\Delta N|N) = \sigma_d^2 N + \sigma_e^2 N^2$. Engen et al. (1998) presented general definitions of the demographic and environmental variances in terms of the lifetime reproductive contributions of individuals to the next generation, R_i. They showed that the demographic variance σ_d^2 is half of the variance in the difference of the R_i values for pairs of individuals (conditioned on current population size). Thus, if all individuals would make exactly the same contribution to the next generation, the demographic variance would be zero, that is, there would be no "demographic stochasticity." In reality, of course, this will not happen because of the intrinsic uncertainty involved in individual births and deaths. The environmental variance is a covariance of the R_i values (Engen et al., 1998). "Environmental stochasticity" is hence great when R_i values vary in parallel, as will happen if the performance of all individuals is influenced by the same common (environmental) factors. Note that positive covariance of the individual R_i values means that the population growth rate exhibits temporal variation. It is also noteworthy that environmental covariance may be negative, that is, an environmental effect may reduce the variance of the change in population size. Engen et al. (1998) gave the (hypothetical) example of space limitation and territoriality leading to a completely constant population size. In this case, the individual R_i values would be necessarily negatively correlated.

The approach developed by Engen et al. (1998) to characterize population fluctuations can be applied to real populations to estimate the demographic and environmental variances and to predict changes in population size, including the risk of population extinction. The drawback of this approach, however, is that one requires data on individual lifetime reproductive contributions, which data are not often available. Saether et al. (1998a) analyzed long-term data on the great tit population at Wytham Wood near Oxford. The environmental variance turned out to be large in this case, but the population was not expected to go extinct because the growth rate was also large. In contrast, in a brown bear population the environmental variance was very small and smaller than the demographic variance (Saether et al.,

1998b). This is consistent with the general expectation that large-bodied vertebrates (like the brown bear) are less influenced by environmental stochasticity than small-bodied vertebrates (like the great tit) and invertebrates. We have more to say about this in the next section.

As a more detailed example, we outline the analysis by Engen et al. (2001) of the stochastic population dynamics of the barn swallow population studied by A.P. Møller at Kraghede, Denmark, since 1970. At this site, the barn swallow population had declined from 184 pairs in 1984 to 58 pairs in 1999. Reasons for the decline appear to be changes in agricultural practices reducing the reproductive success of the birds.

The model fitted by Engen et al. (2001) to data on barn swallows assumes that the stochasticity in the population size is described by a Markov process and that the year-to-year change in the logarithm of population size $X(= \ln N)$ is normally distributed with the expectation

$$E(\Delta X|X = x) = r - {}^1/_2\sigma_e^2 - {}^1/_2\sigma_d^2/N \tag{14.1}$$

and variance

$$\text{var}(\Delta X|X = x) = \sigma_e^2 + \sigma_d^2/N \tag{14.2}$$

The quantity $r_0 = r - {}^1/_2\sigma_e^2$ is defined as the stochastic growth rate and indicates the extent to which stochastic fluctuations in population size reduce the long-term ("long-run") growth rate (Tuljapurkar, 1982; Lande and Orzack, 1988; Lande, 1993). Demographic stochasticity also reduces the long-term growth rate, and the combined effects of demographic and environmental stochasticity lead to the expectation in Eq. (14.1).

Engen et al. (2001) obtained an estimate of the demographic variance σ_d^2 from data on the individual contributions of breeding females to the next generation, R_i (number of female offspring recorded in the next or following generations plus 1 if the female itself survived), calculated as

$$1/(k-1)\sum(R_i - \overline{R})^2, \tag{14.3}$$

where $\overline{R}$ is the mean contribution of the individuals and k is the number of recorded contributions in 1 yr. If data are available for several years, σ_d^2 is estimated as the weighted average of the yearly estimates (Saether and Engen, 2002). In the case of the barn swallow, there were extensive data on individual reproduction and survival, and hence σ_d^2 was assumed to be accurately known as estimated from data for several years, $\sigma_d^2 = 0.180$. Next the values of r_0 and σ_e^2 were estimated from time series data on yearly population sizes by maximizing a likelihood function numerically (Engen et al., 2001). The maximum likelihood parameter estimates were $r_0^* = -0.076$ and $\sigma_e^{2*} = 0.024$. This barn swallow population has thus shown a mean decline of 7.6% per year.

Figure 14.1 shows the lower bound of the prediction interval, which includes the predicted population size with probability $1 - \alpha$. Comparison between Figs 14.1A and 14.1B demonstrates that ignoring uncertainty in parameter estimates (and using their maximum likelihood estimates) increases the predicted time to extinction. In other words, acknowledging the uncertainty in the

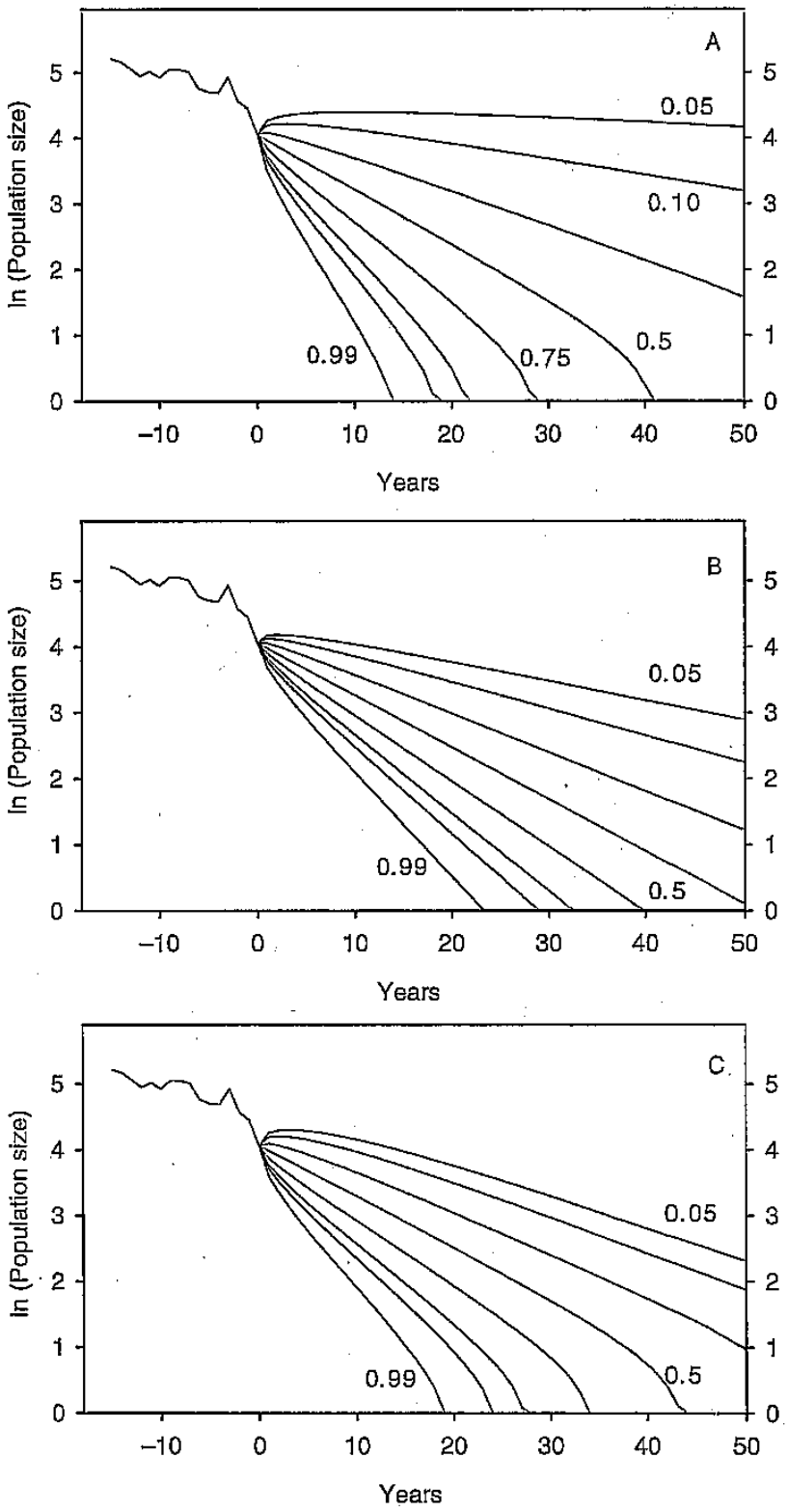

Fig. 14.1 Annual variation in the number of breeding pairs of the barn swallow at a study site in Denmark from 1984 until 1999 (the time period until zero on the *x* axis), followed by the lower bound of different prediction intervals for the future population size for different values of α. Results when (A) all available information is included, (B) uncertainty in parameter estimates is ignored, and (C) demographic variance is set to zero (from Engen et al., 2001).

parameter values leads to more cautious predictions: the population may go extinct sooner than the maximum likelihood estimates would suggest. In Fig. 14.1C, the demographic variance is assumed to equal zero. Ignoring this component increases the predicted time to extinction. Additionally, ignoring environmental variance reduces the range of variation of the prediction interval (Engen et al., 2001). In other words, the fate of the population would be much easier to predict without environmental stochasticity.

Scaling of Extinction Risk with Carrying Capacity

A useful framework for examining many ecological factors in population extinction is provided by the simple "ceiling" model of population dynamics (Lande, 1993; Foley, 1994, 1997; Middleton et al., 1995). Although this model does not incorporate any details of demography and life history of species, it is helpful in encapsulating in general terms the effects on extinction probability of those factors that should always be considered. This theory is also helpful in providing a submodel of local extinction that can be used in metapopulation models (Hanski, 1998a, 1999b; Chapter 4). The ceiling model is described in Box 14.1.

BOX 14.1 The Ceiling Model of Population Extinction

Population dynamics are assumed to obey the following equations:

$$\begin{aligned} n_{t+1} &= n_t + r_t \quad && \text{if } 0 \le n_{t+1} \le k \\ n_{t+1} &= k && \text{if } n_{t+1} > k \\ n_{t+1} &= 0 && \text{if } n_{t+1} < 0, \end{aligned}$$

where n_t is the natural logarithm of population size (N) at time t, k is the logarithm of the population ceiling (K), and r_t is a normally distributed random variable with mean r and variance σ_e^2. The model assumes that the population size performs a random walk between the absorbing lower boundary of population extinction and the reflecting upper boundary of population ceiling. Population fluctuations are driven by environmental stochasticity. Using the diffusion approach to analyze this model (Foley, 1994; Lande, 1993; Middleton et al., 1995), the expected time to extinction of a population with $r > 0$ and starting at the ceiling K is given by

$$T(K) = K^2/sr\,[1 - (1 + sk)/\exp(sk)], \tag{B1}$$

where $s = 2r/\sigma_e^2$. For reasonably large values of sk the term in square brackets is close to 1 and hence the result simplifies to

$$T(K) \approx K^2/sr. \tag{B2}$$

These results were obtained for a model that ignores demographic stochasticity. Hanski (1998a) compared the scaling of time to extinction with population ceiling predicted by (B1) and by the comparable model (from Foley, 1997) with both demographic and environmental stochasticities. For values of σ_d^2/σ_e^2 less than 1, which is likely to be valid for most natural populations, the scaling result (B1) is little affected by the added demographic stochasticity (see Hanski, 1998a).

The most lucid and useful result is obtained by assuming that population fluctuations are driven solely by environmental stochasticity. In other words, we assume, for simplicity, that the demographic variance equals zero. The key parameters are then the population ceiling (absolute carrying capacity) K, which the population size cannot exceed (Box 14.1), and the stochastic population growth rate r_0 discussed in the previous section and given by $r_0 = r - {}^1/_2\sigma_e^2$. Note that if $r < {}^1/_2\sigma_e^2$, the population will go extinct with probability 1 even in the absence of any density dependence. For convenience, we denote the ratio $2r/\sigma_e^2$ by s. Assuming that $r > 0$ and that sk is reasonably large (where k is the logarithm of K), the time to extinction scales asymptotically as

$$T \approx K^s/sr. \tag{14.4}$$

Thus, if population fluctuations are caused solely by environmental stochasticity, the time to extinction scales as a power function of the population ceiling. In the other extreme, when there is no environmental stochasticity and population fluctuations are caused by demographic stochasticity alone, the scaling is nearly exponential (MacArthur and Wilson, 1967; Lande, 1993; Foley, 1994). Exponential scaling means that for reasonably large r, only very small populations have an appreciable risk of extinction. The extreme case of only demographic stochasticity is of academic interest only, as all real populations are more or less influenced by both environmental and demographic stochasticities. Adding demographic stochasticity to the model leading to Eq. (14.4) will shorten the time to extinction (see Fig. 14.1), but the scaling is little affected unless both the ceiling and s are very small (Foley, 1997; Hanski, 1998a; Box 14.1). Hence we focus on the simple result given by Eq. (14.4).

Taking now the interpretation of the power-function scaling further, let us observe that the value of $s = 2r/\sigma_e^2$ is an inverse measure of the strength of environmental stochasticity, scaled by r. The greater the impact of environmental stochasticity on the population growth rate (the smaller the value of s), the shorter the expected lifetime of the population and the smaller the increase in lifetime with increasing population ceiling [Eq. (14.4)]. A high growth rate (r) has the net effect of increasing population lifetime and the opposite effect to that of σ_e^2 on the scaling with population ceiling.

A useful feature of Eq. (14.4) is that the value of the scaling constant s can be estimated with empirical data. Recording actual extinction rates ($1/T$) for particular populations is impractical, but in the context of metapopulations with many local populations in a patch network, one may use the spatially realistic metapopulation theory (Chapter 4) to estimate s from data on the incidence of patch occupancy. Hanski (1998a) applied a mainland-island metapopulation model (Hanski 1993) to data on the occurrence of four species of *Sorex* shrews on small islands. The key assumptions were that island area multiplied by an estimate of population density is an adequate surrogate of the population ceiling and that the occurrence of the species on islands represents a balance between stochastic extinctions and recolonizations [as supported by the results of Hanski (1986) and Peltonen and Hanski (1991)]. Figure 14.2 shows the relationship between the expected lifetime of populations and their carrying capacity for the four species based on the parameter values estimated with the metapopulation model (Hanski, 1993). This result shows wide

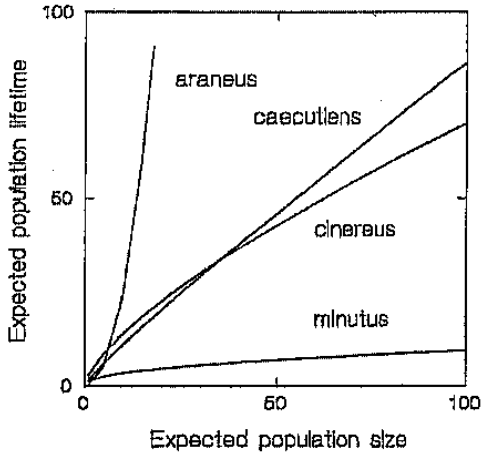

Fig. 14.2 Relationship between the expected population lifetime and the carrying capacity (island area times average density) in four species of *Sorex* shrews on islands. The result was calculated with the parameters of the incidence function metapopulation model fitted to data on island occupancy (from Hanski, 1993).

variation in the value of s, which can be interpreted as variation in the impact of environmental stochasticity among the species, as their r values are comparable. Furthermore, a positive correlation exists between the body size of the species and the value of s, suggesting that environmental stochasticity plays a greater role in the dynamics of small than large species of shrew [Cook and Hanski (1995) reported the same relationship for birds on oceanic islands]. This result makes biological sense because the smallest species of shrew, which weigh less than 3 g and starve in a few hours, are particularly vulnerable to temporal variation in food availability. Hanski (1998a) further estimated the values of r and σ_e^2 for the common shrew (*Sorex araneus*) from the parameter values of the metapopulation model, as $r = 0.75$ and $\sigma_e^2 = 0.42$. These values are consistent with the biology of shrews, which live for 1 yr only and produce one to three litters of seven young on average (Sheftel, 1989). The coefficient of variation calculated from these values of r and σ_e^2 is 0.86, which is consistent with the observed CV calculated from trapping data, 0.67 (average of four independent estimates; Hanski and Pankakoski, 1989). These results are encouraging in highlighting a clear connection between parameters of the extinction model for single populations and parameters of the metapopulation model. One general difficulty, however, is that the estimates of r and σ_e^2 thus obtained are sensitive to the estimate of population density (Hanski, 1998a). Luckily, the scaling constant s is not similarly affected.

Complex Population Dynamics and Extinction

Population dynamics may be called simple if the growth rate is a monotonically decreasing function of population density and if the density-dependent feedback itself does not suffice to generate population oscillations. In this case, exemplified among others by the continuous-time logistic model and the ceiling model in Box 14.1, population density would settle to a stable state with

constant population size in the absence of environmental perturbations and demographic stochasticity. Population extinction is typically caused by a low growth rate (e.g., due to poor habitat quality), a high variance in growth rate (environmental stochasticity), or a small population size due to low carrying capacity or other factors, which increases extinction risk for many reasons (Section 14.4).

Not all populations exhibit such simple dynamics, however, and their extinction risk may be affected by the extra complexities of population dynamics. Most commonly, the population growth rate may be expected to be reduced at very low densities due to difficulty of locating a mate or performing other cooperative behaviors; this is called the Allee effect (Allee, 1938; Allee et al., 1949). If the reduction in growth rate is severe enough, a small population will go deterministically extinct. Demographic stochasticity also substantially increases the risk of extinction of very small populations, especially if their growth rate is low, and there can be a threshold population size below which the most likely population trajectory is a decreasing population size. In this sense, demographic stochasticity creates a sort of stochastic Allee effect (Lande, 1998; Dennis, 2002). In models with both conventional Allee effect and demographic stochasticity, there is an inflection point in the probability of reaching a small population size before reaching a large size. This inflection point, which corresponds to the unstable equilibrium in the underlying deterministic model, represents a threshold in the probabilistic prospects for the population (Dennis, 2002). The incidence and importance of the Allee effect have been reviewed most recently by Saether et al. (1996), Kuussaari et al. (1996), Wells et al. (1998), Courchamp et al. (1999), and Stephens and Sutherland (1999). It should be recognized that small populations have a high risk of extinction for many reasons, including both ecological and genetic factors (Section 14.4), and factors that reduce the expected growth rate as well as factors that increase the variance in growth rate (Stephens et al., 1999; Dennis, 2002). Therefore, it is generally difficult to conclusively isolate the operation of any particular mechanism, including the Allee effect. Many mechanisms are often likely to operate in concert.

A strong Allee effect creates an unstable equilibrium point below which the population goes extinct in a deterministic model. In this case there are two alternative stable equilibria, one corresponding to large population size (set by density dependence at high density) and the other one corresponding to population extinction. If the dynamics exhibit such alternative stable equilibria, a small population below the unstable equilibrium is unlikely to become large, although it may do so and cross the unstable equilibrium thanks to a favorable environmental perturbation. Likewise, a large population above the unstable equilibrium is expected to remain large, but a perturbation may take it below the treshold population size and send it toward extinction. This is a worrying possibility because it implies that currently large populations may have a much greater risk of extinction than one might expect and predict with models that fail to include the mechanism creating alternative equilibria. Unfortunately, it is difficult to assess how likely this scenario is for real populations.

Complex population dynamics in the sense of cyclic or chaotic fluctuations maintained by population dynamic processes (as opposed to environmental effects) have received much attention during the past decades (May, 1974; Schaffer, 1985; Turchin, 2003). Population variability generated by intraspecific

and interspecific interactions is expected to increase the risk of extinction just like variability generated by environmental stochasticity. It has even been argued that extinctions caused by chaotic dynamics would exert a (group) selection pressure that would make chaotic dynamics less likely and that local extinctions due to chaotic dynamics would enhance metapopulation persistence because the extinctions would be asynchronous (Allen et al., 1993; Gonzalez-Andujar and Perry, 1993; Bascompte and Solé, 1994; Ruxton, 1996). Although these issues involve many challenges for further research, it seems unlikely that complex dynamics in this sense would be a major factor in population extinctions.

14.3 POPULATION EXTINCTION: GENETIC FACTORS

Natural populations are also subject to extinction due to genetic factors even in the absence of any human impact and the threat posed by ecological processes. Genetic threats are a function of the effective population size, N_e. Strictly speaking, N_e is defined as the number of individuals in an ideal population that would give the same rate of random genetic drift as observed in the actual population (Wright, 1931, 1938). The ideal population consists of N individuals with nonoverlapping generations that reproduce by a random union of gametes. More intuitively, N_e can be defined as the number of individuals in a population that contribute genes to the following generation. This number can be much lower than the observed population size because of unequal sex ratios, variance in family size, temporal fluctuations in population size, and so forth (for a review, see Frankham, 1995). Thus, apparently large populations may still be quite small in a genetic sense and hence face genetic problems. Small N_e can have multiple effects that include loss of genetic variability, inbreeding depression, and accumulation of deleterious mutations. The time scales at which these factors operate differ and, to a large extent, determine the risk of population extinction that they entail (Table 14.1).

Loss of Genetic Variability

Genetic variation comprises the essential material that allows natural populations to adapt to changes in the environment, to expand their ranges, and even to reestablish following local extinctions (e.g., Hedrick and Miller, 1992). The types of genetic variation considered most often are the heterozygosity of

TABLE 14.1 Time Scales at Which Genetic Factors Operate and Their Importance for Population Extinction[a]

Factor	Time scale	Extinction risk involved	Extinction vortex
Inbreeding depression	Short	High	F
Loss of genetic diversity	Long	Low	A
Mutational meltdown	Medium/long	Unknown	A

[a] The last column indicates the extinction vortex (as defined by Gilpin and Soulé, 1986) under which each genetic factor operates.

neutral markers, H, and the additive genetic variance, V_a, which underlies polygenic characters such as life history traits, morphology, and physiology.

In small populations, random genetic drift leads to stochastic changes in gene frequencies due to Mendelian segregation and variation in family size. In the absence of factors that would replenish genetic variance, such as mutation, migration, and selection favoring heterozygotes, populations lose genetic variance according to

$$V_a(t+1) = V_a(t)\left(1 - \frac{1}{2N_e}\right), \tag{14.5}$$

where $V_a(t)$ is the additive genetic variance in the tth generation. A similar equation is obtained for heterozygosity by replacing V_a with H. When a population is reduced to a small effective size N_e and maintained at that size for more than $2N_e$ generations, its genetic variability is reduced greatly (Wright, 1969). Genetic variability can be restored to its original level through mutation if the population grows back to its original size. The number of generations required to attain the original level is of the order of the reciprocal of the mutation rate, μ. Thus, for a nuclear marker with a mutation rate of 10^{-6}, genetic variation is restored after 10^6 generations, but genetic variation of quantitative characters can be restored after only 1000 generations because the relevant mutation rate is two orders of magnitude higher.

The maximum fraction of genetic variation lost during a bottleneck is a function of the population growth rate (Nei et al., 1975). Populations that recover quickly after the bottleneck lose little genetic variation even if the population was reduced to a few individuals only. For example, a growth rate of $r = 0.5$ ($\lambda = e^r = 1.65$) allows a population that is reduced to only two individuals to retain 50% of its genetic variability (Fig. 14.3). If the population is reduced to 10 individuals, then a growth rate of $r = 0.1$ ($\lambda = 1.10$) would allow it to retain 60% of its variability. Additionally, generation overlap can buffer the effect of environmental fluctuations on population sizes. In general,

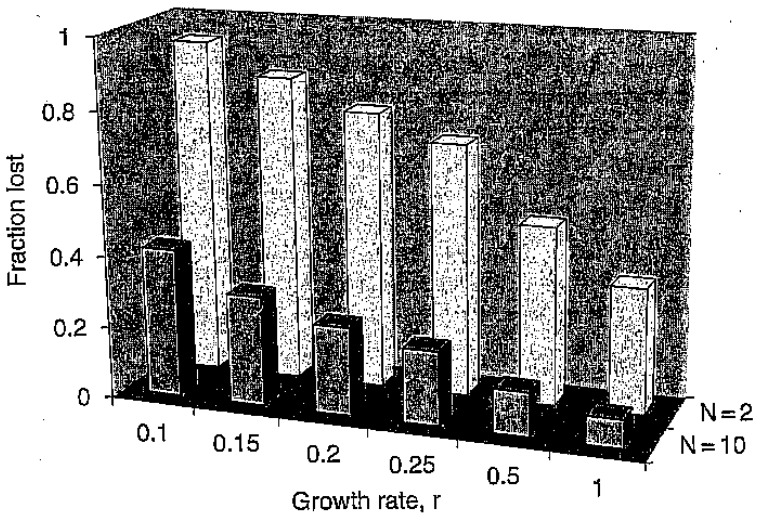

Fig. 14.3 Fraction of the genetic variation lost during a population bottleneck of $N = 2$ or 10 individuals. Calculated using Eq. (8) in Nei et al. (1975).

reductions in population size are brought about by environmental changes that cause fluctuations in vital rate parameters (environmental stochasticity; Section 14.2). The effect of these fluctuations on N_e depends on the life history of the species. The ratio of N_e to census size is directly proportional to the total reproductive value of a population, but the sensitivity of this ratio to environmental fluctuations is proportional to the generation overlap. The larger the generation overlap, the smaller the effect of environmental fluctuations on the level of genetic variability maintained by natural populations (Gaggiotti and Vetter, 1999). Thus, genetic variability is maintained through the "storage" of genotypes in long-lived stages. Adult individuals representing these stages reproduce many times throughout their lives and, therefore, the genetic variability present in a given cohort is more likely to be transferred to future generations than in the case of organisms with discrete generations.

These buffering mechanisms may explain why there are very few clear examples of populations that have lost a very large fraction of their genetic variability due to a bottleneck. One of the few cases is that of the Mauritius kestrel, which was reduced to a single pair in the 1950s. A comparison of microsatellite diversity present in museum specimens collected before the bottleneck and in extant individuals reveals that at least 50% of the heterozygosity was lost due to the bottleneck (Groombridge et al., 2000). Another example is the northern elephant seal, which was exploited heavily during the 19th century and reduced to a bottleneck population size estimated to be 10–30 individuals (Hoelzel et al., 2002). A comparison of genetic diversity in prebottleneck and postbottleneck samples shows a 50% reduction in *mt*DNA-haplotype diversity. The reduction in heterozygosity at microsatellite loci was less pronounced, however.

An important caveat concerning the effect of reductions in population size on genetic diversity is that although such reductions may not have a very large effect on H, they will have a large impact on allelic diversity because random genetic drift will eliminate low-frequency alleles very rapidly (Nei et al., 1975). This is of particular concern because the long-term response of a population to selection is determined by the allelic diversity that remains after the bottleneck or that is gained through mutations (James, 1971). A second caveat is that in the case of quantitative genetic characters, genetic variability may not always be beneficial. Using a model with overlapping generations and assuming weak stabilizing selection, Lande and Shannon (1996) showed that the effects of additive genetic variance on the average deviation of the mean phenotype from the optimum, and the corresponding "evolutionary" load, depend on the pattern of environmental change. In an unpredictable (random) environment, additive genetic variance contributes to the evolutionary load because any response to selection increases the expected deviation between the mean phenotype and the optimum. However, when environmental changes are unidirectional, cyclic, or positively correlated (predictable), additive genetic variance allows the mean phenotype to track the optimum more closely, reducing the evolutionary load.

Most empirical studies on the effects of population bottlenecks on genetic diversity focus on the heterozygosity of neutral markers. Although neutral genetic variation may become adaptive if the environment changes, the ability of a population to respond to novel selection pressures is proportional to the additive genetic variation underlying the traits that are the target of selection (Falconer and Mackay, 1996). Unfortunately, direct quantification of the genetic

variation underlying polygenic traits is difficult to measure, and hence heterozygosity of nuclear markers is used as an indicator of additive genetic variation [see Pfrender et al. (2001) and references therein]. This practice is unwarranted, however, because of the different rates at which genetic variation is replenished in neutral and quantitative markers (Lande 1988; see earlier discussion). Indeed, Pfrender et al. (2001) detected no significant relationship between heritability for reproductive traits and heterozygosity in natural populations of *Daphnia pulex* and *D. pulicaria*. Thus, the absence of genetic diversity in nuclear markers does not necessarily indicate an immediate genetic threat.

In general, the loss of genetic variation is detrimental for the long-term survival of populations. However, as pointed out by Allendorf and Ryman (2002), there is one case where a reduction in genetic variability can represent an imminent extinction threat. This is the case for loci associated with disease resistance, such as the major histocompatibility complex (MHC), which is one of the most important genetic systems for infectious disease resistance in vertebrates (Hill, 1998; Hedrick and Kim, 2000). Allelic diversity at these loci is extremely high; for example, Parham and Otha (1996) documented 179 alleles at the MHC class I locus in humans. However, species that have been through known bottlenecks have very low amounts of MHC variation. A study of the Arabian oryx found only three alleles present at the MHC class II DRB locus in a sample of 57 individuals (Hedrick et al., 2000). Hunting pressure led to the extinction of this species in the wild in 1972. Captive populations have been susceptible to tuberculosis and foot-and-mouth disease, which is consistent with low genetic variability at MHC loci. Low genetic diversity at the MHC complex was also observed in the bison, which went through a bottleneck at the end of the 19th century (Mikko et al., 1997). In the Przewalski's horse, in which the entire species is descended from 13 founders, Hedrick et al. (1999) observed four alleles at one locus and two alleles at a second locus. The northern elephant seal is another example of low MHC diversity, as Hoelzel et al. (1999) found only two alleles at the MHC class II DQB gene in a sample of 69 individuals.

To summarize, we may conclude that loss of genetic variation as measured by heterozygosity and additive genetic variance represents a long-term extinction threat. In the short term, the loss of allelic diversity can have important consequences if it occurs at loci associated with disease resistance.

Inbreeding Depression

The decrease in fitness due to mating between related individuals is known as inbreeding depression and results from the segregation of partially recessive deleterious mutations maintained by the balance between selection and mutation. Deleterious mutations occur continuously in all populations and most mutations are at least partially recessive. In large populations, selection keeps these detrimental mutations at low equilibrium frequencies. Thus, under random mating, most copies of detrimental alleles are present in a heterozygous state and hence their detrimental effects are partially masked. Mating between relatives, however, increases homozygosity and, therefore, the deleterious effects become fully expressed, decreasing the fitness of inbred individuals.

Although it is generally agreed that increased expression of deleterious partially recessive alleles is the main cause of inbreeding depression, there is an additional mechanism that can contribute to inbreeding depression. If the fitness of a heterozygote is superior to that of both homozygotes (heterozygous advantage or overdominance), the reduced frequency of heterozygotes will reduce the opportunities to express heterozygous advantage. This mechanism may be important for certain traits (e.g., sperm precedence in *Drosophila melanogaster*) and may contribute to the very high inbreeding depression for net fitness observed in *Drosophila* and outcrossing plants (Charlesworth and Charlesworth, 1999).

The degree of inbreeding in a population is measured by the inbreeding coefficient F, which can be defined as the probability that the two alleles of a gene in an individual are identical by descent. The effect of inbreeding in a population with inbreeding coefficient F can be measured in terms of the logarithm of the ratio of the mean fitness values for the outbred, W_O, and the inbred, W_I, populations (Charlesworth and Charlesworth, 1999),

$$\ln\left(\frac{W_I}{W_O}\right) = BF. \tag{14.6}$$

The coefficient B can be interpreted as the reduction in log fitness associated with complete inbreeding ($F = 1$).

In small populations, the opportunities for mating are restricted, even under random mating. Thus, mating among relatives is common and the proportion of individuals that are homozygous at many loci increases, which results in inbreeding depression. The amount of inbreeding depression manifested by a population depends not only on F, but also on the opportunity for selection to purge recessive lethal and semilethal mutations. Gradual inbreeding by incremental reductions in population size over many generations allows selection to eliminate the lethal and sublethal mutations when they become homozygous (Falconer, 1989). However, the component of inbreeding depression due to more nearly additive mutations of small effect is difficult to purge by inbreeding (Lande, 1995). As to empirical results, recent reviews indicate that purging is inefficient in reducing inbreeding depression in small inbred populations [see Allendorf and Ryman (2002) and references therein].

Most of the evidence for inbreeding depression comes from domesticated or captive populations. This, together with the theoretical expectation that a large fraction of inbreeding depression can be purged in small populations and the numerous mechanisms of inbreeding avoidance observed in many species, has led many researchers to question the importance of inbreeding depression for the persistence of natural populations (Keller and Waller, 2002). However, in the last decade there has been a rapid accumulation of evidence showing that many populations do exhibit inbreeding depression. For example, the Soay sheep on the island of Hirta (Saint Kilda archipelago, UK) suffer of significant inbreeding depression in survival (Coltman et al., 1999). More homozygous sheep suffered higher rates of parasitism and, in turn, lower overwinter survival than heterozygous sheep. Another example comes from song sparrows living on Mandarte Island (western Canada). In this case, inbred birds died at a much higher rate during a severe storm than outbred birds (Keller et al., 1994). A more recent study (Keller, 1998) was able to quantify

inbreeding depression in this population and estimated that inbreeding depression in progeny from a mating between first-degree relatives was 49%. The negative effect of inbreeding has also been documented in the red-cockaded woodpecker living in the southeastern United States. Inbreeding reduced egg hatching rates, fledgling survival, and recruitment to the breeding population (Daniels and Walters, 2000). Extensive long-term data sets can help uncover inbreeding depression in large populations with a low rate of inbreeding. An 18-yr study of a large population of the collared flycatcher revealed that inbreeding was rare, but when it did occur it caused a significant reduction in egg hatching rate, in fledgling skeletal size, and in postfledging juvenile survival (Kruuk et al., 2002). This study also found that the probability of mating between close relatives ($F = 0.25$) increased throughout the breeding season, possibly reflecting increased costs of inbreeding avoidance. Inbreeding depression is also evident in plants. Byers and Waller (1999) documented many examples of inbreeding depression in natural populations and indicated that purging does not appear to act consistently as a major force in natural plant populations.

Evidence shows that stressful environmental conditions can amplify inbreeding depression. Crnokrak and Roff (1999) gathered and analyzed a data set that included seven bird species, nine mammal species, four species of poikilotherms, and 15 plant species. They were able to show that conditions experienced in the wild increase the cost of inbreeding. A more recent study by Keller et al. (2002) showed that the magnitude of inbreeding depression in juvenile and adult survival of cactus finches living in Isla Daphne Major (Galápagos Archipelago) was strongly modified by two environmental conditions; food availability and number of competitors. In juveniles, inbreeding depression was present only in years with low food availability, whereas in adults, inbreeding depression was five times more severe in years with low food availability and large population size.

Demonstrating the importance of inbreeding depression in the wild does not necessarily imply that it will cause natural populations to decline (Caro and Laurenson, 1994). However, recent papers have demonstrated that this may happen. Saccheri et al. (1998) studied the effect of inbreeding on local extinction in a large metapopulation of the Glanville fritillary butterfly (*Melitaea cinxia*) and found that extinction risk increased significantly with decreasing heterozygosity due to inbreeding, even after accounting for the effects of ecological factors. Larval survival, adult longevity, and egg hatching rate were all affected adversely by inbreeding and seem to be the fitness component responsible for the relationship between inbreeding and extinction. An experiment by Nieminen et al. (2001) provided further support to the results of Saccheri et al.'s (1998) field study. Nieminen et al. (2001) established inbred and outbred local populations of the Glanville fritillary at previously unoccupied sites using the same numbers of individuals. The extinction rate was significantly higher in populations established with inbred individuals. Similar evidence for plants is provided by Newman and Pilson (1997). They established experimental populations of the annual plant *Clarkia pulchella* that differed in the relatedness of the founders. All populations were founded by the same number of individuals but persistence time was much lower in those populations whose founders were related. Additional evidence for inbreeding

influencing population dynamics comes from the study of an isolated population of adders in Sweden (Madsen et al., 1999), which declined dramatically in the late 1960s and was on the brink of extinction due to severe inbreeding depression. The introduction of 20 adult male adders from a large and genetically variable population led to a rapid population recovery due to a dramatic increase in recruitment.

The evidence discussed here indicates that inbreeding depression is common in natural populations and can represent a short-term extinction threat to small populations, especially if populations are subject to stressful conditions or to sharp population declines.

Accumulation of Slightly Deleterious Mutations

Under more or less constant environmental conditions, mutations with phenotypic effects are usually deleterious because populations tend to be well adapted to the biotic and abiotic environmental conditions which they experience. A random mutation is likely to disrupt such adaptation. In populations with moderate or large effective sizes, selection is very efficient in eliminating detrimental mutations with large effects on fitness. However, mildly deleterious mutations with selection coefficient $s < 1/2N_e$ are difficult to remove because they behave almost as neutral mutations (Wright, 1931). Thus, small population size hampers selection and increases the role of genetic drift in determining allele frequencies and fates. This increases the chance fixation of some of the deleterious alleles supplied constantly by mutation and results in the reduction of population mean fitness, which eventually leads to population extinction (Muller, 1964). Initially, this process was assumed to represent a threat to asexual populations only because in the absence of recombination, their offspring carry all the mutations present in their parent as well as any newly arisen mutation (Muller, 1964). Mathematical models of this process (Lynch and Gabriel, 1990; Lynch et al., 1993, 1995a) show that the process of mutation accumulation can be divided into three phases. During the first two phases, deleterious mutations accumulate and fitness declines, but population size remains close to carrying capacity. During the third phase, fitness drops below 1 and population size declines. This population decline increases the effect of random genetic drift, which enhances the chance fixation of future deleterious mutations, leading to further fitness decline and reduction in population size. Due to this positive feedback, the final phase of population decline (when growth rate is negative) occurs at an accelerating rate, a process known as "mutational meltdown."

Although recombination can slow down the mutational meltdown to some extent, sexual populations are also at risk of extinction due to mutation accumulation (Lande, 1994; Lynch et al., 1995a). Lande (1994) modeled a randomly mating population with no demographic or environmental stochasticity and considered only unconditionally deleterious mutations of additive effects. He derived analytical approximations for the mean time to extinction for two cases: (a) when all mutations had the same selection coefficient s and (b) when there was variance in s. Lynch et al. (1995a) provided a more detailed analysis of scenario (a) and checked the analytical results using computer simulations. With constant s, the mean time to extinction, $\bar{t}_e$, is an approximately

exponential function of the effective population size. Because the mean time to extinction increases very rapidly with increasing N_e, the fixation of new mutations poses little risk of extinction for populations with N_e of about 100 (Lande, 1994). However, with variance in s, the mean time to extinction increases as a power of N_e. For instance, if s is distributed exponentially, $\bar{t}_e$ is asymptotically proportional to N_e^2. As an increase in $\bar{t}_e$ with population size is now more gradual than for constant s, the risk of extinction is much elevated. For reasonable variance in s (coefficient of variation around 1), the mutational meltdown is predicted to pose a considerable risk of extinction for populations with N_e as large as a few thousand individuals (Lande, 1994). If, as is generally agreed, the ratio of N_e to census population size is around 0.1 to 0.5, moderately sized populations of several thousand individuals may face extinction due to genetic stochasticity.

Unfortunately, there is a paucity of empirical evidence for or against the mutational meltdown. What we have is experimental evidence for the accumulation of deleterious mutations due to genetic drift, but these studies do not directly address the risk of extinction (Zeyl et al., 2001). As of today, only Zeyl et al. (2001) explicitly explored the plausibility of the mutational meltdown. They established 12 replicate populations of the yeast *Saccharomyces cerevisiae* from two isogenic strains whose genome-wide mutation rates differed by approximately two orders of magnitude. They used a transfer protocol that resulted in an effective population size of around 250. After more than 100 daily bottlenecks, yeast populations with elevated mutation rates showed a tendency to decline in size, whereas populations with wild-type mutation rates remained constant. Moreover, there were two actual extinctions among the mutant populations. These results provide support for the mutational meltdown models.

Despite this preliminary empirical support, there are a number of issues that remain unresolved. The first one relates to a controversy about the estimates of per-genome mutation rates, U, and the average fitness cost per mutation, s, used in the meltdown models. The values that have been assumed were based on mutation accumulation experiments using *Drosophila melanogaster*, suggesting values of $U = 1$ and a reduction in fitness of about 1–2% (Lande, 1994; Lynch et al., 1995a). Studies reviewed by Garcia-Dorado et al. (1999) on *D. melanogaster*, as well as on *Caenorhabditis elegans* and *S. cerevisiae*, yielded values of U orders of magnitude less than 1. However, some mutation accumulation experiments (Caballero et al., 2002; Keightley and Caballero, 1997) reported average fitness effects one order of magnitude higher than those reported previously. The assumption of additive effects is also questioned by Garcia-Dorado et al. (1999), who reported estimates of 0.1 for the average coefficient of dominance. The new estimates of U and s would lead to much lower rates of fitness decline, making the mutational meltdown less likely. Caballero et al. (2002) used a combination of mutation accumulation experiments and computer simulations and concluded that a model based on few mutations of large effect was generally consistent with their empirical observations.

Finally, an additional criticism of the existing mutational meltdown models relates to the fact that the models ignore the effect of beneficial and back mutations. Models including these types of mutations suggest that only very small populations would face the risk of extinction due to genetic stochasticity (Poon and Otto, 2000; Whitlock, 2000). Estimates of mutational effects using

mutation accumulation experiments with *Arabidopsis thaliana* indicate that roughly half of the mutations reduce reproductive fitness (Shaw et al., 2002). The genome-wide mutation rate was around 0.1–0.2. These new results suggest that the risk of extinction for small populations may be lower than initially thought. This issue is reviewed in greater detail in Chapter 7.

At the moment it is not possible to draw definite conclusions about the importance of the mutational meltdown process. This will only be possible once the existing controversy over the rate and nature of spontaneous mutations is resolved (Poon and Otto, 2000). The resolution of this question in turn requires knowledge of the distribution of mutational effects and the extent to which these effects are modified by environmental and genetic background. Additionally, it is necessary to better understand the contribution of basic biological features such as generation length and genome size to interspecific differences in the mutation rate (Lynch et al., 1999).

14.4 POPULATION SIZE, HABITAT PATCH SIZE, AND EXTINCTION RISK

The most robust generalization that we can make about population extinction is that small populations face a particularly high risk of extinction. Holsinger (2000) digged up statements to this effect from the writings of Darwin (1859), E.B. Ford (1945), and (not surprisingly) Andrewatha and Birch (1954). More recent empirical support for the extinction-proneness of small populations has been found practically whenever this issue has been examined; Diamond (1984), Newmark (1991, 1995), Ouborg (1993), Burkey (1995), and Fischer and Stöcklon (1997) represent a small sample of the literature covering different kinds of taxa and spatial scales. The high extinction risk of small populations is not surprising because this is the expectation based on several mechanisms of extinction: demographic and environmental stochasticity, Allee effect, inbreeding depression, mutational meltdown, and so forth. Furthermore, as the different mechanisms tend to make populations ever smaller, they reinforce the effect of each other and lead to what Gilpin and Soulé (1986) termed extinction vortices. Gilpin and Soulé (1986) identified four extinction vortices. Two of them, the R and D vortices, involve only demographic and ecological factors (demographic stochasticity and population fragmentation). The two other ones, F and A vortices, consider the feedback among demographic, ecological, and genetic factors. One way of gauging how much our understanding of the interactions among demographic, ecological, and genetic factors has improved in the last decade or so is to evaluate to what extent the current knowledge calls for a reformulation or refinement of the F and A vortices.

As originally formulated, the F vortex is the consequence of reduced fitness due to inbreeding depression and loss of heterozygosity in initially large populations that have been reduced to a small size. The decrease in fitness further reduces population size, which in turn further increases inbreeding depression and loss of heterozygosity, increasing the probability of extinction via this and all other vortices. Theoretical and empirical advances made in the last few years and reviewed earlier indicate that the enhanced vigor that is often associated with increased heterozygosity is most likely due to a reduced homozygosity of

deleterious alleles rather than to heterozygosity per se (see Section 14.3). Furthermore, it is becoming increasingly clear that purging the genetic load leading to inbreeding depression is generally not that efficient in natural populations (Section 14.3). Therefore, the F vortex in the form of inbreeding depression remains a likely mechanism of population extinction.

The A vortex was also attributed to genetic drift and loss of genetic variance, but in this case, Gilpin and Soulé (1986) proposed that a reduction in population size and the increased genetic drift that ensues could reduce the efficiency of stabilizing and directional selections, in turn causing an increasing and accelerating "lack of fit" between the population phenotype and the environment it faces. This was hypothesized to reduce population size and growth rate even further until the population goes extinct. This mechanism was not formulated very precisely, but it is related to the mutational meltdown discussed in Section 14.3. The reduction in the efficiency of stabilizing and directional selections leads to an accumulation of slightly deleterious mutations, which will progressively reduce population growth rate until it becomes negative. Once this happens, the population size will decrease and the rate at which deleterious mutations accumulate will increase further. This feedback mechanism will eventually lead to population extinction. Another mechanism that was proposed for this vortex is loss of genetic variance, which will impair the capacity of populations to track environmental changes.

An additional short-term mechanism could be added to the A vortex. The loss of habitat reduces population sizes and may lead to a loss of variation at MHC loci, making individuals less able to resist infectious diseases. At the same time, habitat destruction might, in some cases, lead to an initial increase in local density, as individuals crowd in the remaining suitable habitat. High density following fragmentation might in turn increase the disease transmission rate (McCallum and Dobson, 2002). Additionally, land degradation increases the opportunity for contact among humans, domesticated animals, and wildlife, also possibly increasing the transmission of diseases (Deem et al., 2001). An increased transmission rate and a lowered disease resistance will further decrease population size and lead to a further decrease in genetic variability at MHC loci. This feedback loop will increase progressively the extinction probability via this and all other vortices.

Delayed Population Responses to Environmental Deterioration

Although it is abundantly clear that small populations exhibit a high rate of extinction, we cannot rest assured that large populations have a low risk of extinction. Consider the familiar deterministic continuous-time logistic model, with growth rate r and carrying capacity K. The equilibrium population size, without any consideration for stochasticity, is given by K. Now, many forms of deterioration in habitat quality affecting the birth and death rates may be reflected in a reduction in the value of r while K remains unchanged (or is only little affected). In this case, the deteriorating environmental conditions are not expected to be reflected in population size until r drops below zero and the population collapses rather abruptly to extinction or, in a metapopulation context, turns from a source population to a sink population. Incidentally, the genetic meltdown models discussed in the previous section envision a similar gradual

decline in r, although now because of an accumulation of deleterious mutations. Although the deterministic logistic model can hardly be considered a realistic description of the dynamics of real populations, the phenomenon we have just outlined occurs in all population models. Things can be even worse, from the perspective of a manager who is trying to read the early signs of approaching trouble, in multispecies models, in which interspecific interactions can compensate for environmental deterioration (Abrams, 2002). The bottom line is that a large population size is not necessarily a reliable indicator of a small risk of extinction.

Even if the equilibrium population size would fairly reflect the environmental conditions, such that a large population would indicate favorable conditions and a low risk of extinction, there are still two other concerns that should not be ignored: (1) the possibility of alternative stable states, which was discussed in Section 14.2, and (2) the time it takes for the population to respond to changing environmental conditions. In other words, in a changing environment the current size of the population to some extent reflects the past rather than the present environmental conditions. If the environment has deteriorated rapidly, the population size is therefore larger than the long-term expected (equilibrium) population size, and evaluation of extinction risk based on population size only would lead to an overly optimistic assessment. Ovaskainen and Hanski's (2002; Hanski and Ovaskainen, 2002) analysis of transient dynamics in metapopulation models demonstrates that the time lag is especially long when the environment is close to the extinction threshold of the species following environmental change (see Section 4.4 in Chapter 4). Thus, whenever the changing environmental conditions lead to a relatively quick change in the parameters that set the extinction threshold, we may expect long transient times in exactly those species that we are most concerned about.

Effect of Habitat Patch Size on Extinction

Assuming constant population density, which implies uniform habitat quality, larger habitat patches have larger expected population sizes than smaller patches. Therefore, other things being equal, we could expect large habitat patches to have populations with a lower risk of extinction than populations in small patches. Although other things are usually by no means equal, and population density varies because of variation in habitat quality and for other reasons, a relationship between habitat patch size and extinction risk has typically been documented whenever this relationship has been examined (Hanski, 1994a,b, 1999b). This finding has been employed in the dynamic theory of island biogeography (MacArthur and Wilson, 1967) and, more recently, in the spatially realistic metapopulation theory (Chapter 4). More generally, the relationship between patch size and extinction risk provides a key rule of thumb for conservation: other things being equal, it is better to conserve a large than a small patch of habitat or to preserve as much of a particular patch as possible. One important caveat relates to the position of a habitat patch in a patch network (Section 4.4). Naturally, if empirical information exists on variation in patch quality, such information should be used in assessing the relative values of different patches (most simply by multiplying true patch area by the population density, estimated on the basis of habitat quality; for an example, see Chapter 20).

If habitat patches of very different sizes are compared, there are likely to be many complementary reasons why large patches have populations with a low risk of extinction. Hanski (1999b) discussed three different scenarios. In the small-population scenario, the reason for a low rate of population extinction in large patches is large population size itself, as discussed in Section 14.2 [Eq. (14.4)]. In the changing environment scenario, large patches support populations with a small extinction risk because the greater environmental heterogeneity in large than small patches reduces the risk of population extinction. Examples are discussed by Kindvall (1996) for a species of bush cricket and by several chapters in Ehrlich and Hanski (2004) for checkerspot butterflies. Finally, in the metapopulation scenario, large patches in fact consist of patch networks for the focal species, and metapopulation dynamics increase the lifetime of the population in the patch as a whole (Holt, 1993). Regardless of the actual reason why large patches of habitat support populations with a low risk of extinction, the conservation implications remain the same.

14.5 LOCAL EXTINCTION IN THE METAPOPULATION CONTEXT

The previous sections discussed the ecological and genetic processes that operate in the extinction of isolated populations. Although habitat fragmentation increases the isolation of populations, few populations are completely isolated. In contrast, innumerable local populations interact regularly via migration with other local populations in metapopulations. It is appropriate to ask what new processes influencing the extinction risk of local populations might operate in metapopulations. Not surprisingly, these new processes relate to migration and gene flow. Migration and gene flow can both increase and decrease local extinction risk.

Migration and Gene Flow Decreasing Extinction Risk

The beneficial effect of migration arises because immigrants from surrounding populations may prevent the extinction of small local populations, a process known as the rescue effect. In the literature on metapopulations, the rescue effect is occasionally extended to cover recolonization following extinction, but more properly the rescue effect refers to processes that reduce the extinction risk in the first place. A demographic rescue occurs because immigration increases the population size, thereby making extinction less likely (Brown and Kodric-Brown, 1977). An extreme case is presented by source–sink systems, where a (true) sink population has a negative growth rate (e.g., due to poor habitat quality) and may only survive with sufficient immigration from one or more source populations (Chapter 16). Immigration reducing extinction risk is also common in the case of small populations inhabiting small habitat patches located close to large populations, a common situation in many metapopulations. Table 14.2 gives an example on the Glanville fritillary butterfly (*M. cinxia*) in the Åland Islands, Southwest Finland, where the butterfly has a metapopulation consisting of several hundred local populations (Hanski, 1999b). Larvae live gregariously, and population sizes are often very small in terms of the number of larval groups, even though populations have tens of

TABLE 14.2 The Rescue Effect Reduces the Risk of Extinction in Small Local Populations of the Glanville Fritillary Butterfly (*Melitaea cinxia*)[a]

Number of larval groups	Extinct	*n*	Average *S*	The rescue effect	
				t	*P*
1	Yes	150	2.55		
	No	76	2.84	−2.97	0.003
2	Yes	46	2.78		
	No	58	3.12	−2.24	0.025
3–5	Yes	46	2.88		
	No	202	2.75	−0.63	0.527
> 5	Yes	14	3.31		
	No	204	2.83	1.42	0.155

[a] Sizes of local populations are given in terms of the number of larval groups in autumn 1993, the numbers of these populations that went extinct and survived, a measure of connectivity (S) to nearby populations, and a t test of the rescue effect, which was measured by the effect of S on extinction (from a logistic regression, which also included the effects of patch area and regional trend in population sizes on extinction; Hanski, 1999b).

butterflies. Comparing the numbers of populations of given size that did or did not go extinct in 1 yr, it is apparent that populations that were well connected to other populations had a lower risk of extinction than more isolated populations (Table 14.2). It also makes sense that this effect was statistically significant in the case of the smallest populations only because the influence of a given amount of immigration in increasing population size is greatest in the case of the smallest populations. Note that large populations have a much smaller risk of extinction than small populations in Table 14.2.

Local populations may be rescued demographically, as we have just discussed, but they may also be rescued genetically. Gene flow may increase the mean population fitness due to heterosis and the arrival of immigrants with high fitness (outbred vigor). Heterosis refers to increased fitness among offspring from crosses among local populations; different populations tend to fix different random subsets of deleterious alleles, which mask each other when populations are crossed (Crow, 1948; Whitlock, 2000). Therefore, initially rare immigrant genomes are at a fitness advantage compared to resident genomes because their descendants are more likely to be heterozygous for deleterious recessive mutations that cause inbreeding depression in the homozygous state (Ingvarsson and Whitlock, 2000; Whitlock et al., 2000).

Several studies have provided fairly conclusive evidence supporting this expectation. Saccheri and Brakefield (2002) carried out an experimental study with the butterfly *Bicyclus anynana*. They focused on the consequences of a single immigration event between pairs of equally inbred local populations. The experiment involved transferring a single virgin female from an inbred (donor) population to another inbred (recipient) population. The spread of the immigrant's and all the residents' genomes was monitored during four consecutive generations by keeping track of the pedigree of all individuals in the treatment populations. They replicated this experimental design and observed a rapid

increase in the share of the initially rare immigrant genomes in local populations. Ball et al. (2000) reported similar evidence for *D. melanogaster*, measuring the relative frequency of immigrant marker alleles in the first and second generations following a transfer to inbred populations. When immigrants were outbred, the mean frequency of the immigrant allele in the first and second generation after migration was significantly higher than its initial frequency. They attributed this result to the initial outbred vigor of immigrant males, but the possibility of heterosis having played a role was not excluded completely.

Ebert et al. (2002) carried out experiments using a natural *Daphnia* water flea metapopulation in which local extinctions and recolonizations, genetic bottlenecks, and local inbreeding are common events. Their results indicate that because of heterosis, gene flow was several times greater than would be predicted from the observed migration rate. Somewhat less conclusive evidence comes from Richards's (2000) experiments with the dioecious plant *Silene alba*, in which isolated populations suffer substantial inbreeding depression. Richards (2000) measured gene flow among experimental populations separated by 20 m and used paternity analysis to assign all seeds to either local males or to immigrants from other nearby experimental populations. When the recipient populations were inbred, unrelated males from the experimental population 20 m away sired more offspring than expected under random mating. This may be due to some form of pollen discrimination that may be influenced by early acting inbreeding depression (Richards, 2000) or to heterosis per se. Incidentally, the rescue effect in Table 14.2 for the Glanville fritillary butterfly could also involve a genetic component, as it is known that inbreeding depression increases the risk of extinction of small populations of this butterfly (Saccheri et al., 1998; Nieminen et al., 2001).

Migration can have a long-term beneficial effect on population persistence. The arrival of migrants from large populations can increase genetic variability in the recipient populations and, thereby, enhance the evolutionary potential of the species as a whole. The extent to which migration can replenish genetic variability depends on population dynamics and the pattern of migration among populations. Populations with positive growth rates can recover lost genetic variability rapidly, but sink populations will only be able to maintain genetic variability when the variance in the migration process is low (Gaggiotti, 1996; Gaggiotti and Smouse, 1996).

Migration and Gene Flow Increasing Extinction Risk

Migration may increase the extinction risk of local populations for several reasons. In the landscape ecological literature, the role of corridors in maintaining viable (meta)populations in fragmented landscapes has been discussed for a long time. Corridors enhance recolonization and the rescue effect (Bennett, 1990; Merriam, 1991; Haas, 1995; Andreassen et al., 1996b; Haddad, 1999a), but it has been pointed out that corridors may also facilitate the spread of disease agents and predators that might actually increase the extinction risk of the focal populations (Simberloff and Cox, 1987; Hess, 1994). More generally, it is well established both theoretically (Hassell et al., 1991; Comins et al., 1992; Nee et al., 1997) and empirically (Huffaker, 1958; Nachman, 1991; Eber and Brandl, 1994; Lei and Hanski, 1998; Schöps et al.,

1998) that specific natural enemies in prey–predator metapopulations may substantially increase the extinction risk of local prey populations.

Just like immigration into small populations may reduce their risk of extinction, emigration from small populations may increase extinction risk (Thomas and Hanski, 1997; Hanski, 1998b). Theoretical studies have elucidated the critical minimum size of habitat patches that would allow the persistence of viable populations (Okubo, 1980); populations in patches smaller than this critical size go extinct because they lose individuals too fast in comparison with the rate of reproduction. However, just like with the rescue effect in saving small populations, it is hard to prove conclusively that small populations go extinct because of emigration, as small populations are likely to go extinct for many other reasons as well. Nonetheless, emigration compromising the viability of local populations is a potentially important consideration in the conservation of some species. For instance, it has been suggested that small reserves for butterflies should not be surrounded by completely open landscape because this will increase the rate of emigration greatly (Kuussaari et al., 1996).

Migration can also have negative genetic effects on population persistence. In principle, gene flow may reintroduce genetic load fast enough to prevent the purging of inbreeding depression, although we are not aware of any clear evidence for this. More importantly, the long-term beneficial effects of migration may be offset by the introduction of maladapted genes, which may lead to a loss of local adaptation in some populations, the appearance of source–sink dynamics, and the evolution of narrow niches (Kirkpatrick and Barton, 1997; Ronce and Kirkpatrick, 2001). This process, called migrational meltdown (Ronce and Kirkpatrick, 2001) because small populations experience a downward spiral of maladaptation and shrinking size, is discussed in the next section.

The introduction of immigrant genomes from a highly divergent population can reduce mean population fitness, a phenomenon known as outbreeding depression (Fig. 14.4). Outbreeding depression will be expressed in the F_1

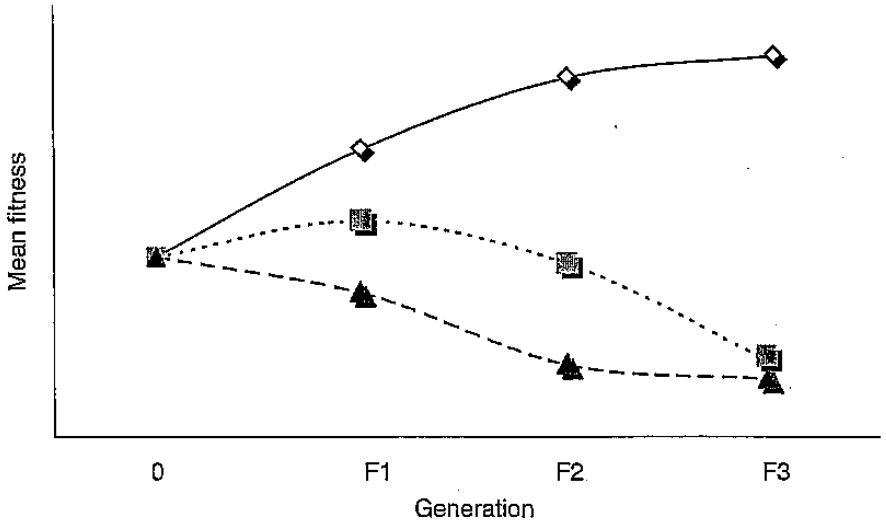

Fig. 14.4 Potential effects of migration on population fitness: (a) heterosis increases fitness (solid line and diamonds), (b) heterosis followed by outbreeding depression leads to a short-lived fitness increase followed by a decline, and (c) outbreeding depression leads to a steady decline in fitness.

generation if the favorable between-population dominance effects (masking the effect of deleterious recessive genes present in the homozygote state in parental lines but in the heterozygote state in the F_1) are outweighed by the loss in favorable additive x additive interactions within populations (Lynch and Walsh, 1998). However, even if this does not occur, outbreeding depression may still be expressed in the F_2 generation or later. The reason for this is that F_1s carry a haploid set of chromosomes from each parental line, and segregation and recombination begin to break apart coadapted genes from a single line in the F_2 generation (Dobzhansky, 1950, 1970). Thus, outbreeding depression is demonstrated when the performance of F_2s is less than the average of immigrants and residents (Lynch and Walsh, 1998). Unfortunately, only few studies of natural populations have tracked the contribution of immigrants beyond the F_1 generation (Marr et al., 2002). The few studies that go beyond F_1 indicate that outbreeding depression may be common in the wild. Marr et al. (2002) showed that the same population of song sparrows in the Mandarte Islands that manifested heterosis among immigrant offspring also displayed signs of outbreeding depression in the F_2 generation. Studies of the tidepool copepod *Tigriopus californicus* show that crosses between populations typically result in F_1 hybrid vigor and F_2 hybrid breakdown for a number of measures related to fitness (Burton 1987, 1990a,b; Edmands and Burton, 1998; Burton et al., 1999). Edmands (1999) showed that the detrimental effects of breaking up coadaptation are magnified by increasing genetic distance between populations. This same effect was shown for the shrub *Lotus scoparius*, but in this case outbreeding depression was already present in the F_1 generation (Montalvo and Ellstrand, 2001). Other plant species for which outbreeding depression has been demonstrated include *Ipomopsis aggregata* (Waser et al., 2000) and *Silene diclinis* (Waldmann, 1999).

14.6 METAPOPULATION EXTINCTION

Not only populations but also metapopulations consisting of many local populations possess a smaller or greater risk of extinction — the metapopulation is extinct when the last remaining local population is extinct. Chapter 4 presents a thorough account of the metapopulation theory, albeit largely from the perspective of one particular class of models, stochastic patch occupancy models. A primary focus of this theory is to dissect the conditions of long-term metapopulation persistence (in deterministic models) and the factors determining the expected lifetime of metapopulations (in stochastic models). Chapter 2 complements this analysis from the perspective of landscape ecology. The spatially realistic metapopulation theory in Chapter 4 is concerned primarily with just one factor in increasing the risk of metapopulation extinction, namely habitat loss and fragmentation, but as we all know, this is currently the main cause of population, metapopulation, and species extinctions. Rather than repeating what has already been written in Chapters 2 and 4 and discussed in the context of particular metapopulations in Chapters 20 and 21, we highlight here one ecological factor that is often critical in metapopulation extinction. We also discuss two genetic processes that have been proposed to

increase the risk of metapopulation extinction, mutational and migrational meltdowns, both of which stem from an interaction between demographic and genetic processes in metapopulation dynamics.

Regional Stochasticity

The counterpart of environmental stochasticity in local populations is regional stochasticity in metapopulations — spatially correlated environmental stochasticity affecting local populations in metapopulations (Hanski, 1991). Just as environmental stochasticity amplifies population fluctuations in local populations and is the major cause of population extinction, regional stochasticity amplifies fluctuations in the size of metapopulations (Fig. 4.11 in Chapter 4 gives a theoretical example and Chapter 21 reviews regional stochasticity in small mammal metapopulations). There is a large literature on spatial synchrony in population dynamics (Ranta et al., 1998; Bjørnstad et al., 1999; Paradis et al., 1999; Engen et al., 2002a) with the same general message. The two mechanisms of spatial synchrony that have been most discussed are migration and regional stochasticity (typically spatially correlated weather conditions influencing birth and death rates). As shown by Lande et al. (1999), even low rates of short-distance migration may affect population synchrony greatly if population regulation is weak. Engen et al. (2002b) examined the probability of quasiextinction for a population distributed continuously in space and affected by regional stochasticity (quasiextinction was defined as the population size dropping below 10% of the carrying capacity). The expected time to quasiextinction decreases with increasing strength of environmental stochasticity, with decreasing rate of migration, and with increasing area within which changes in population size are recorded. The expected population density decreases, and hence the probability of quasiextinction increases, with increasing spatial scale of regional stochasticity.

Metapopulation Meltdown

The accumulation of slightly deleterious mutations can have detrimental effects at the metapopulation level. Higgins and Lynch (2001) extended the mutational meltdown theory described in Section 14.3 to metapopulations using an individual-based model that includes demographic and genetic mechanisms and environmental stochasticity. The metapopulation structure was modeled as a linear array of patches connected by nearest-neighbor (stepping-stone), global (island), or intermediate dispersal. The mutational effect was modeled in such a way that mutations of large effect are almost recessive, whereas those of small effect are almost additive. Results show that for metapopulations with more than a few patches, an accumulation of deleterious mutations accelerates extinction time by many orders of magnitude compared to a globally dispersing metapopulation without mutation accumulation. Moreover, extinction due to mutation accumulation can be quite rapid, on the order of tens of generations. In general, results indicate that the mutational meltdown may be a significant threat to large metapopulations and would exacerbate the effects of habitat loss or fragmentation on metapopulation viability. These conclusions were reached under the assumptions of an

expected genome-wide mutation rate of 1 per generation and unconditionally deleterious mutational effects. As mentioned before, these two assumptions have been placed under close scrutiny, and preliminary evidence indicates that they may not be generally valid.

Migrational Meltdown

Another genetic mechanism for metapopulation extinction stems from the idea that peripheral populations receive gene flow from the center of the species' range. These immigrant genes will typically be adapted to the conditions at the range center and could inhibit adaptation in the periphery (Mayr, 1963). Kirpatrick and Barton (1997) used a quantitative genetic model to study the evolution of a species range in a linear habitat with local migration. The model tracks evolutionary and demographic changes across space and time and assumes that variation in the environment generates patterns of selection that change in space but are constant in time. Among other things, results show that a species' range may contract as the dispersal rate increases and extinction may follow if conditions change too rapidly as one moves across space, even if the species remains perfectly adapted to the habitat at the range center. Ronce and Kirpatrick (2001) also studied the maladaptive effect of migration but they considered a model with two discrete habitat types connected by migration. In this case, an increasing migration rate above a threshold value results in the collapse of the total population size and the complete loss of one of the populations. However, in contrast to Kirpatrick and Barton's (1997) analysis, there is no metapopulation extinction. Ronce and Kirpatrick (2001) attributed this disagreement between the two models to the assumption of infinite space made by Kirpatrick and Barton: the distance traveled by migrants and thus the maladaptation of such migrants to local conditions increase indefinitely with the migration rate. This assumption is unlikely to be valid for real situations and, therefore, complete metapopulation extinction due to migrational meltdown is unlikely to occur.

14.7 CONCLUDING REMARKS

The major causes of population and species extinctions worldwide are habitat loss and interactions among species. The models discussed in this chapter address the adverse effects of habitat loss in terms of the reduced sizes of populations and metapopulations that are the inevitable and direct result of habitat loss. With metapopulation models, we may additionally examine the consequences of habitat loss that occur in the surroundings of the focal population, and which consequences influence the focal population via metapopulation dynamics (Chapter 4).

Considering interactions with other species, it may at first appear surprising that this would be an important cause of population extinction — if this were the case, would such extinctions not have already happened a long time ago? This argument does not hold in two situations: in metapopulations with recurrent extinctions and colonizations (Section 14.5) and when species are spreading into areas where they did not use to occur and become hence

engaged in novel interactions. We all know that such invasions, with often adverse consequences for native species, have become rampant in the modern world, where humans have helped, in one way or another, innumerable species to spread beyond their past geographical ranges. The actual mechanisms of extinction of native species include hybridization with the invasive species (Simberloff, 1994; Wolf et al., 2001; Levin, 2002; Perry et al., 2002). The spreading of *Homo sapiens* itself, in the far past, was the likely cause of extinction of a large fraction of the megafauna in North America, Australia, and many large islands (Martin and Klein, 1984; Caughley and Gunn, 1996) at a time when humans could be placed among other animals in their lack of concern for the survival of other species. No wonder, then, that modern humans are able to hunt and drive many species to extinction or near extinction. Harvesting of populations has been and continues to be a major threat to both terrestrial and marine populations. Models and ecological knowledge could and should be used to guide harvesting of economically valuable populations (Getz and Haight, 1989; Lande et al., 1995), but generally this is not what happens in reality.

Interactions with invasive species, persecution, and harvesting, along with habitat loss, are *the* major ultimate threats to populations and species, and the threats with which most practical conservation efforts have to be concerned. From this perspective, many of the population ecological and genetic mechanisms discussed in this chapter may appear insignificant. Nonetheless, the matter of fact is that increasing numbers of species are being reduced to a state in which the small-population issues (Caughley, 1994) covered here are relevant and interact with the primary causes of threat (Hedrick et al., 1996). Clearly, population biologists alone cannot solve the current extinction crisis, but we can provide improved knowledge of many specific biological issues. Finally, of course, just like the study of population regulation has been of great intrinsic interest to population ecologists for more than a century, so are the inevitable "failures" of regulation in finite populations.

One of the largely open scientific issues in the study of population extinction relates to the current controversy surrounding genome-wide mutation rates and the average effect of deleterious mutations (Section 14.3). Before these questions have been resolved, it is premature to draw definite conclusions about the importance of mutational meltdown in population and metapopulation extinctions. More research on the mutation process underlying the mutational meltdown and more extensive empirical research on the feasibility of this phenomenon are needed. Additionally, models such as that of Higgins and Lynch (2001) should be extended to include beneficial as well as deleterious mutations. Likewise, additional work has to be carried out to evaluate the importance of the genetic rescue effect due to heterosis and, in particular, to understand how outbreeding influences the mean fitness of natural populations. It is likely that the extent of outbreeding depression depends on how inbred the local populations that receive the migrants are. Highly inbred populations whose fitness is very low may react positively to the influx of migrants and show no signs of outbreeding depression at all. However, less inbred populations whose fitness has not been impaired dramatically may show heterosis in the F_1 generation but outbreeding depression in the F_2 and subsequent generations or outright outbreeding depression. Unraveling the

effects of immigration on fitness will require carrying out experiments that follow the fate of the descendants of immigrants beyond the F_2 generation and control for the inbreeding level of the target populations.

We have commented in the introduction and in later sections of this chapter on the changing views about the relative roles of ecological and genetic factors in population and metapopulation extinction. The theoretical and empirical work done in the past decade makes it clear that genetic factors can contribute significantly to population extinction. In particular, there is a rapidly expanding body of literature demonstrating that inbreeding depression in natural populations is often sufficiently severe to have significant consequences for population dynamics and thereby for extinction. The most clear-cut demonstrations of inbreeding increasing the risk of population extinction, such as in the Glanville fritillary butterfly (Saccheri et al., 1998; Nieminen et al., 2001), relate to very small populations. For this reason, some might dismiss the new evidence as of little general importance. However, this is not so in the metapopulation context, where small populations are often frequent and matter for the dynamics of the metapopulation as a whole. This is also the context that shows very clearly how Caughley's (1994) declining-population paradigm and small-population paradigm interact. Very often, habitat loss and fragmentation are the root causes of metapopulation decline (declining-population paradigm), but the actual metapopulation response to environmental changes is largely determined by what happens in the often small local populations (small-population paradigm). The relative roles of genetic and ecological factors in extinction are also likely to vary among taxa with different biologies. For instance, environmental stochasticity is generally the overriding cause of extinction in insects and other invertebrates, whereas inbreeding might be expected to play a relatively greater role in vertebrate populations that are less influenced by random variation in environmental conditions.