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Anthropogenic, Ecological and Genetic Factors in Extinction and Conservation

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Abstract. Anthropogenic factors constitute the primary deterministic causes of species declines, endangerment and extinction: land development, overexploitation, species translocations and introductions, and pollution. The primary anthropogenic factors produce ecological and genetic effects contributing to extinction risk. Ecological factors include environmental stochasticity, random catastrophes, and metapopulation dynamics (local extinction and colonization) that are intensified by habitat destruction and fragmentation. Genetic factors include hybridization with nonadapted gene pools, and selective breeding and harvesting. In small populations stochastic factors are especially important, including the ecological factors of Allee effect, edge effects, and demographic stochasticity, and the genetic factors of inbreeding depression, loss of genetic variability, and fixation of new deleterious mutations. All factors affecting extinction risk are expressed, and can be evaluated, through their operation on population dynamics.

Key words: conservation, demography, ecology, extinction risk, genetics.

Introduction

Many plant and animal species around the world are in danger of extinction, largely as a result of human activities. Planning regions often include multiple threatened and endangered species, which implies that effective conservation and restoration must be done in the context of comprehensive landscape and ecosystem approaches that consider biodiversity and large-scale ecological processes. Species and population-based approaches should nevertheless play an essential role in the development and monitoring of regional conservation and restoration strategies to ensure proper management of ecologically important species, or those that indicate ecosystem health. Analyzing factors that contribute to the extinction risk of particular species therefore remains of central importance even within landscape and ecosystem approaches to conservation and restoration.

The primary causes of species declines, endangerment and extinction are anthropogenic: land development, overexploitation, species translocations and introductions, and pollution. Anthropogenic factors produce ecological and genetic effects that contribute to extinction risk. For example, land development causes habitat fragmentation, isolation of small populations, and intensification of metapopulation dynamics. All factors affecting extinction risk are ultimately manifested, and can be evaluated, in terms of population dynamics. This article reviews anthropogenic, ecological and genetic factors contributing to extinction risk, briefly discussing their relative importance and interactions in the context of conservation planning.

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Anthropogenic factors

Land development

Human population growth and economic activity convert vast natural areas for settlement, agriculture and forestry. This produces ecological effects of habitat destruction, degradation, and fragmentation which are among the most important causes of species declines and extinctions. Habitat destruction contributes to extinction risk of three-quarters of the threatened mammals of the Australasia and the Americas and more than half of the endangered birds of the world (Groombridge 1992).

Overexploitation

Unregulated economic competition

Inadequate regulation of competing resource extractors, especially in open-access fisheries and forestry, is one of

the major causes of overexploitation and depletion of renewable resources (Ludwig et al. 1993; Rosenberg et al. 1993). About half of the fisheries in Europe and the United States were recently classified as overexploited (Rosenberg et al. 1993). Hunting and international trade contributes to the extinction risk of over half of the threatened mammals of Australasia and the Americas and over one-third of the threatened birds of the world (Groombridge 1992) and has caused local extinctions of many forest-dwelling mammals and birds even in areas where habitat is largely intact (Redford 1992).

Economic discounting

A nearly universal economic practice is discounting of future profits. Annual discount rates employed by governments and corporations exploiting natural resources are often in the range of 5% to 10% or higher. Clark (1973, 1990) showed that in many cases there is a critical discount rate above which the optimal strategy from a narrow economic viewpoint is immediate harvesting of the population to extinction (liquidation of the resource). In simple deterministic models with a constant profit per individual harvested, the critical discount rate equals the maximum per capita rate of population growth, r_{max}, because money in the bank grows faster than the population (May 1976). Organisms with long generation time and/or low fecundity, such many species of trees, parrots, sea turtles, and whales, have r_{max} below the prevailing discount rate and are frequently threatened by overexploitation.

Stochastic fluctuations in population size reduce sustainable harvests (Beddington and May 1977; Lande et al. 1995). Optimal harvesting strategies that reduce extinction risk as well as maximize sustainable harvests generally involve threshold population sizes below which no harvesting occurs (allowing the population to increase at the maximum natural rate when it is below the threshold), and above which harvesting occurs as fast as possible (Lande et al. 1994, 1995, 1997).

Introduction of exotic species

Numerous species are transported and released in foreign environments deliberately and accidentally in biological control, ornamental plantings, live animal trade, and in private and commercial transport. Introduced species of parasites, predators and competitors seriously affect about one-fifth of the endangered mammals of Australia, Asia and the Americas, and birds of the world (Groombridge 1992; Dobson and May 1986). Introduced rats caused extinctions of many island-endemic birds (Atkinson 1989). In some Hawaiian National Parks, up to half of the plant species are non-native (Vitousek 1988) and pose a serious risk for the endangered flora.

Pollution

Agricultural and industrial pollution produce both localized and diffuse effects. Long-lasting pesticides, such as DDT, become concentrated in terrestrial and aquatic food chains, and have endangered several birds of prey such as the American bald eagle and peregrine falcon. Although bans on most long-lasting pesticides in the USA helped recovery of both these species in the USA, such pesticides are still used in many countries. About 4% of endangered birds of the world and 2.5% of mammals of Australasia and the Americas are at risk from pollution (Groombridge 1992). These figures underestimate the extent of morbidity, mortality, and fertility impairment caused by pesticides in many non-endangered species.

Acid rain has had intense regional effects on terrestrial plant communities in western Europe and on freshwater ecosystems in the eastern USA. In Germany, about one-fourth of the native species of ferns and flowering plants are endangered or extinct, with about 5% affected by air and soil pollution, and 5% by water pollution (ODEC 1991).

Ecological factors

Environmental fluctuations and catastrophes

Unexploited vertebrate populations fluctuate through time with coefficients of variation in annual abundance usually in the range of 20% to 80% or more (Pimm 1991). Exploited populations also are highly variable (Myers et al. 1995), not only due to environmental stochasticity, but because commonly used exploitation strategies, such as constant effort or constant rate harvesting, tend to reduce population stability (Beddington and May 1977; May et al. 1978). Catastrophes are extreme environmental fluctuations that suddenly reduced the population by a large fraction, usually caused by extreme climatic conditions, such as droughts, floods, or extreme temperatures, or by disease outbreaks (Young 1994).

In stochastic population models with either normal environmental fluctuations in population growth rate or random catastrophes, for a population initially near carrying capacity the mean time to extinction, T, scales asymptotically (for sufficiently large populations) as the carrying capacity raised to a power. Depending on the magnitude of environmental fluctuations in population growth rate, or on the frequency and magnitude of catastrophes, this power may be either greater than or less than one. Comparing populations with different carrying capacities, under low environmental stochasticity, T increases faster than linearly with increasing carrying capacity, whereas under high environmental stochasticity, T increases less

than linearly with increasing carrying capacity (Lande 1993). Logarithmic scaling of T with initial population size applies to a declining population, in which the mean growth rate is negative, regardless of whether the decline is deterministic or stochastic (Lande 1993). Asymptotic scaling laws for different risk factors are shown in Table 1.

If population subdivision substantially reduces the correlation in environmental stochasticity among localities, e.g. considering one large reserve versus multiple small dispersed reserves with the same total area, then subdivision can increase T. For example, when single populations are subject to major catastrophes, occurring randomly among populations, then population subdivision can clearly be advantageous for persistence (Burkey 1989). Subdivision also can increase persistence in the presence of catastrophic epidemics, not only by reducing the transmission of epidemics among localities, but also by reducing their frequency because many epidemics happen only above a threshold population size or density (Hess 1996). In contrast, if multiple small reserves located in the same general area are subject to nearly identical environmental stochasticity, then subdivision that reduces local population size is likely to decrease persistence by exacerbating edge effects, Allee effects, and demographic stochasticity.

Ecosystem function is likely to be enhanced in large contiguous reserves. Species diversity is expected to be larger, initially, in several small reserves spread over a larger geographic area, but these would tend to suffer more rapid local extinctions than in a single large reserve of the same total size. Designs for nature reserves systems must balance these advantages and disadvantages of subdivision. From a review of data on several natural and artificial archipelagoes, Burkey (1995) concluded that on a single large island the rate of species extinctions is initially faster, but ultimately slower, than on several small islands.

Table 1. Asymptotic scaling laws for mean time to extinction, T, from different ecological and genetic risk factors, as a function of the initial actual size, N, or effective size, N_e, of a population at carrying capacity.

Risk Factor	*Proportional scaling of T*
Declining population[1]	$-(\ln N)/\bar{r}$
Environmental stochasticity[2]	$N^{2\bar{r}/V_e-1}$
Demographic stochasticity[3]	$(1/N)\exp\{2Nr/V_1\}$
Fixation of new mutations[4]	N_e^{1+1/c^2}

[1] In this case only, mean population growth rate, $\bar{r}$, is negative.
[2] Mean and variance of annual growth rate are respectively $\bar{r}$ and V_e.
[3] Mean and variance of individual Malthusian fitness are respectively $\bar{r}$ and V_1.
[4] Coefficient of variation of selection against new mutations is c.

Metapopulation dynamics

Dispersal among local populations on patches or "islands" of suitable habitat also can have demographic advantages and disadvantages for persistence. The main advantage is that dispersers can recolonize suitable habitat after local extinctions, allowing a metapopulation to persist in a balance between local extinction and recolonization (Levins 1970; Hanski and Gilpin 1997). In Levins' (1970) original metapopulation model the equilibrium proportion of islands occupied by a species is $p=1-e/m$ where e is the rate of local extinction and m is the colonization rate. Metapopulation persistence requires that $m>e$. This and other demographic and genetic benefits of dispersal (described below) have spurred interest in various methods of enhancing dispersal, from artificial transport of individuals or germ cells to preservation or creation of habitat corridors connecting islands of suitable habitat. The utility of corridors has rarely been tested (Andreassen et al. 1996), but for many species they may be of little value because of edge effects, suggesting that it may often be necessary to manage the entire matrix between reserves to ensure successful dispersal.

The major demographic disadvantage to dispersal occurs during individual movement through unsuitable habitat in a heterogeneous landscape. For species in which individuals or mated pairs hold exclusive territories or home ranges with small overlap, the basic effects of habitat destruction and fragmentation can be taken into account along with life history, age-structured population dynamics, and individual dispersal behavior (Lande 1987). Identifying the individual territory as the local unit in a metapopulation, local extinction corresponds to the death of an individual, and colonization constitutes successful dispersal into a suitable, unoccupied patch of habitat. In this model, patches of suitable habitat the size of individual territories are assumed to be randomly or evenly distributed across a large region, with no clumping on a spatial scale much larger than the mean individual dispersal distance, and the proportion of the region composed of suitable habitat is h. The equilibrium proportion of suitable habitat occupied by the species is $p=1-(1-k)/h$. The "demographic potential," k, depends on the life history and dispersal behavior of the species and gives the maximum proportion of suitable habitat occupied in a completely suitable region ($p=k$ when $h=1$).

This model reveals two general and robust features of great importance for conservation planning. First, unoccupied suitable habitat may be as important as occupied habitat for long-term persistence of a metapopulation. Continual destruction of unoccupied habitat will doom a metapopulation to early extinction. Second, as the amount of suitable habitat in a region decreases through anthropogenic alterations or natural processes, the

equilibrium occupancy of suitable habitat decreases. Since the population size equals the amount of suitable habitat multiplied by its occupancy, the equilibrium population size generally declines faster than the rate of habitat loss. This implies the existence of an "extinction threshold" or minimum density of suitable habitat in a region necessary for population persistence, $h=1-k$ ($p=0$ when $h\leq 1-k$). A population may become extinct in the presence of suitable habitat if this is too sparsely distributed, as first shown by application of this model to the northern spotted owl in the Pacific Northwest USA (Lande 1988a; Doak 1989; Thomas et al. 1990; McKelvey et al. 1993).

Few attempts have been made to analyze dispersal and stochastic local population dynamics in metapopulation models that could be applied more generally to non-territorial species. Initial work in this direction (Hanski and Gyllenberg 1993; Hanski et al. 1995; Lande et al. 1998) suggests the existence of alternative equilibria: a stable equilibrium with high occupancy of suitable habitat, and an unstable equilibrium with low occupancy of suitable habitat. These alternative equilibria arise because of interactions between local and global population dynamics. At low habitat occupancy, emigration from an occupied patch is not compensated by immigration, which can render isolated populations vulnerable to extinction. Increasing habitat occupancy in the metapopulation increases the number of immigrants to a given site, decreasing the rate of local extinction (the "rescue effect" of Brown and Kodric-Brown 1977) and increasing the probability of successful colonization per immigrant ("establishment effect" of Lande et al. 1998). The unstable equilibrium at low habitat occupancy constitutes a kind of Allee effect at the metapopulation level.

Small population size

Demographic stochasticity

Random individual variation in vital rates of mortality and reproduction, and random variation in adult sex-ratio, cause fluctuations in the per capita growth rate of small populations. The magnitude of these fluctuations is inversely proportional to population size because independent random events among individuals tend to average out in a large population. In contrast to environmental stochasticity which may operate with equal intensity in populations of any size, demographic stochasticity affects small populations most strongly. Demographic stochasticity generally is thought to be of relatively little importance in populations larger than roughly 100 individuals (MacArthur and Wilson 1967; Richter-Dyn and Goel 1972; Lande 1993). However, in small populations demographic stochasticity may be the dominant stochastic factor in population dynamics, posing a greater risk of extinction than environmental stochasticity. For a population initially at carrying capacity, under demographic stochasticity alone the mean time to extinction scales asymptotically almost exponentially with carrying capacity (Table 1). Demographic stochasticity can create a type of Allee effect such that in populations below a small size most population trajectories tend to decrease, resulting in a high probability of extinction (Lande 1998).

Allee effect

In populations below a certain size or density, individuals may suffer reduced fitness from insufficient cooperative interactions with conspecifics. Cooperative social behaviors occur in many animal species, including group defense against predators, physical or chemical conditioning of the environment (e.g. huddling for warmth), communal nesting, and increased per capita efficiency of group foraging. More generally in small or sparsely distributed populations, individuals may have difficulty encountering potential mates. These effects can produce negative rates of population growth in small populations, creating an unstable equilibrium at small population size below which the population tends to decline to extinction (Allee et al. 1949; Andrewartha and Birch 1954). For example, the Lakeside Daisy is a self-incompatible perennial, and the last individuals in Illinois were found to be incompatible and hence incapable of reproduction (DeMauro 1993). After overharvesting, the white abalone off the California coast is declining to extinction because population densities are insufficient to ensure adequate rates of fertilization during broadcast spawning (Malakoff 1997).

Edge effects

Habitat destruction and fragmentation create new edges between habitat types and may reduce habitat quality for considerable distances inside suitable habitat patches, by causing microclimatic alterations and facilitating incursion or invasion of exotic species. For example, clearing tropical rainforests for pastureland causes desiccation and vegetational changes up to hundreds of meters inside remnant forest patches (Lovejoy et al. 1986). Fragmentation of temperate zone forests by agriculture and settlement facilitates the invasion of cowbirds that parasitize the nests of other birds, some of which are endangered (Robinson et al. 1995).

Another type of edge effect arises from dispersal beyond the boundary of suitable habitat. The intrinsic rate of population increase and the rate of individual dispersal into unsuitable regions determine the minimum size of a geographically isolated patch of suitable habitat that can support a stable population, known as the critical patch size. With random dispersal, lethal surroundings, and a low intrinsic rate of increase per generation, the critical patch size is much larger than the average individual

dispersal distance (Kierstead and Slobodkin 1953). More hospitable surroundings, high intrinsic rate of increase, and habitat selection behavior decrease the critical patch size (Okubo 1980; Pease et al. 1989).

Genetic factors

Maladaptive translocation and hybridization

The low rate of interspecific hybridization that often occurs between closely related species may be beneficial in augmenting intraspecific genetic variance and adaptive evolution (Lewontin and Birch 1966; Grant and Price 1981). Artificial habitat disturbance facilitates contact and hybridization between normally non-interbreeding species. Interspecific contact and hybridization also can occur through invasion or introduction of exotic species. Abnormally high rates of interspecific hybridization are likely to be maladaptive because of partial sterility and reduced viability caused by post-zygotic reproductive isolating mechanisms between species (Dobzhansky 1970), which can exert a heavy cost on a rare species hybridizing with a common species (Levin et al. 1996). When partial prezygotic reproductive isolating mechanisms exist, interspecific hybridization may not threaten the demographic stability of a species but can nevertheless destroy its genetic integrity. For example, fragmentation of old-growth forests in the Pacific Northwest USA facilitated range extension of the barred owl which is hybridizing with the northern spotted owl. Molecular genetic evidence indicates that domestic dogs are hybridizing with the endangered Simien jackal (Wayne 1996). With artificial increases in water turbidity that decrease color vision, sexual selection and mate choice, hybridization has reduced the diversity of subspecies and species of cichlid fish in Lake Victoria (Seehausen et al. 1997).

Intraspecific hybridization also can produce maladaptive effects by diluting gene pools adapted to local environmental conditions. This frequently occurs when nonlocal genetic strains are used for restocking game fish and forest trees. Lack of attention to genetic properties of introduced stocks in past decades has resulted in widespread declines in fitness of stocked populations and maladaptive hybridization with remaining wild stocks. This is one of the major factors contributing to massive declines and numerous local extinctions of salmon runs in the Pacific Northwest USA (Nehlsen et al. 1991; Ratner et al. 1997).

Selective breeding and harvesting

Many exploited populations are subject to intense selective harvesting based on individual size and age. This can produce maladaptive evolution in life history and behavior that diminishes the quantity and quality of future harvests. Selective harvesting is thought to be a factor in body size declines of many exploited stocks of anadromous fish (Stokes et al. 1993).

Intense selective pressures can occur during artificial propagation of captive populations, because artificial environments differ substantially from natural ones (Arnold 1995). The resulting evolutionary changes are likely to be maladaptive for populations reintroduced into the wild. Artificial propagation for augmentation and reintroduction should be done in as few generations under as natural conditions as possible. Long-term restocking programs, such as fish hatcheries, may do more harm than good, and should not be viewed as a substitute for habitat restoration (Allendorf and Waples 1996).

Small population size

Inbreeding depression

Matings between closely related individuals tend to reduce offspring fitness due to the expression of (partially) recessive deleterious mutations in homozygous form. In historically large, outcrossing populations inbreeding typically reduces fitness by a few to several percent per 10% increase in the coefficient of inbreeding or consanguinity (Ralls and Ballou 1983; Falconer and Mackay 1996). For domesticated animal species, experimental propagation by continued brother-sister mating generally results in extinction of a large fraction of lines within 5 or 10 generations (Soulé 1980; Frankham 1995a). Species, and populations within a species, differ substantially in the magnitude of inbreeding depression (Soulé 1980; Lacy et al. 1993).

The genetic basis of inbreeding depression is best understood in species of *Drosophila*, in which roughly equal parts are contributed by nearly recessive lethal mutations and by partially recessive (nearly additive) mildly deleterious mutations (Simmons and Crow 1977). Both types of mutations arise at thousands of genetic loci throughout the genome in eukaryotic species (Simmons and Crow 1977). Gradual inbreeding allows natural selection to purge recessive lethal mutations from a population as they become expressed in homozygotes, but it is difficult or impossible for inbreeding to purge the more nearly additive mildly deleterious mutations (Lande and Schemske 1984; Charlesworth and Charlesworth 1987). However, for populations with extremely high inbreeding depression, such as some tree species and gynodioecious plants, it may be difficult to purge even the recessive lethals by close inbreeding because if nearly all the selfed offspring die before reproduction then the population is effectively outcrossed and no purging occurs unless the selfing rate exceeds a threshold value (Lande et al. 1994).

Sudden reduction to very small population size almost

inevitably causes a substantial loss of fitness from inbreeding, unless the population rapidly recovers to a large size thereby allowing selection to reverse the short-term effects of inbreeding and random genetic drift (e.g. Keller et al. 1994). The more gradual the reduction in population size, the greater the opportunity for purging recessive lethal mutations and avoiding a large part of the inbreeding depression. Therefore, inbreeding depression is not simply proportional to the inbreeding coefficient routinely calculated for selectively neutral genes. The rule suggested by Franklin (1980) and Soulé (1980), supported by extensive practical experience in animal and plant breeding, is that inbreeding depression can be largely avoided in populations with effective sizes larger than $N_e=50$. However, inbreeding depression may be more severe in natural environments than in laboratory populations (Jiménez et al. 1994) and in stressful than in optimal environments (Keller et al. 1994; K. Biljsma personal communication).

Inbreeding depression can be readily reversed (at least temporarily) by introduction of several unrelated individuals into an inbred population, and it can be permanently prevented by continued immigration every one or two generations of a single unrelated individual into each local population regardless of their size (Lande and Barrowclough 1987). Such a plan was recently instituted for the endangered Florida panther, motivated by strong circumstantial evidence of inbreeding depression in the small remnant population, and their low genetic divergence from other conspecific populations (Hedrick 1995). Although this genetic augmentation may be necessary to reverse inbreeding effects (and not too high to swamp any local adaptations), the Florida Panther still faces the ecological threats of small population size due to past habitat destruction, and high mortality from automobile collisions.

Loss of genetic variation

Finite population size causes stochastic changes in gene frequencies known as random genetic drift, attributable to Mendelian segregation and variation in family size, which results on average in a loss of genetic variance from a population. A fraction of $1/(2N_e)$ of selectively neutral genetic variance is expected to be lost from a population per generation, where N_e is the effective population size. For wild populations the effective size is usually substantially less than the actual size because of large variance in family size, unequal sex ratio among breeders, and temporal fluctuations in population size (Wright 1969). Accounting for all these factors, the ratio of effective to actual size of wild populations is often on the order of 0.1 (Frankham 1995b). Weakly selected genes become effectively neutral if the magnitude of selection on them is much less than $1/(2N_e)$ (Wright 1969).

To be expected to lose a large fraction of its genetic variance, measured by heterozygosity in molecular genetic polymorphisms or heritable variance in quantitative characters, a population reduced to a small effective size N_e must remain small for at least $2N_e$ generations (Wright 1969). Following such a population "bottleneck," genetic variance can be replenished by immigration and/or mutation. An isolated population that passes through a bottleneck must regain large size and remain large for a long time for mutation to restore normal levels of genetic variance. Metapopulation structure, with frequent local extinction and colonization, can reduce N_e of the metapopulation orders of magnitude below its actual size, mimicking the genetic effects of a population bottleneck (Wright 1940; Maruyama and Kimura 1980; Hedrick 1996).

All types of genetic variance are equally influenced by random genetic drift, but different kinds of genetic variance are replenished at different rates depending on their mutability. Stable populations of different sizes also maintain unequal proportions of different kinds of genetic variance depending on the balance between random genetic drift, mutation and selection (see Table 2). Among populations of comparable size, there may be substantial differences in inbreeding depression (Lacy et al. 1993), in heritable variance of quantitative characters (Bürger and Lande 1994), or in molecular heterozygosity at particular loci (Wright 1969), due to random genetic drift, and differences in population structure, history, and selection. Different types of genetic variance therefore should not be expected to have a close concordance among populations of different size, contrary to the suggestion of Soulé (1980). For example, populations with moderate effective size, on the order of $N_e=10^3$ to 10^4, may maintain low molecular heterozygosity for point mutations, with substantial heritable variance in quantitative characters, and typical inbreeding depression and heterozygosity for microsatellites.

A low dispersal rate of a few individuals exchanged among populations per generation is sufficient to prevent much genetic differentiation at quasineutral loci, such as

Table 2. Mutability, approximate time scale in generations for replenishment, and minimum effective population size (N_e) for maintaining typical levels, of different types of genetic variance in a randomly mating population. Molecular and quantitative variance are assumed to be quasineutral (excluding strongly selected mutations).

Genetic variance	*Mutability*	*Time scale*	*minimum* N_e
Inbreeding depression	high	10^2	50
Quantitative characters	moderate	10^4	5,000
Molecular heterozygosity			
point mutations	low	2×10^4 to 10^5	10^4 to 10^5
microsatellites	high	10^3	500

most molecular genetic polymorphisms (Wright 1969; Crow and Kimura 1970). In contrast, natural selection can maintain adaptive differences among populations even under high levels of dispersal (Endler 1977). Lack of differentiation between populations at molecular genetic loci therefore does not imply lack of adaptive differences. Thus while molecular differentiation among populations is likely to imply adaptive divergence among populations, the converse is not true. It could therefore be a serious mistake to manage populations in different environments as a single unit, simply because no molecular differentiation among them has been detected, especially if morphological, behavioral and physiological characteristics in which the populations might be adaptively differentiated have not been investigated.

Based on experimental estimates of mutability in quantitative characters, Franklin (1980) and Soulé (1980) recommended a minimum effective population size of $N_e = 500$ to maintain typical levels of heritable variance. Recent experimental evidence indicates that a large fraction of the mutational variance in quantitative characters is associated with recessive lethal and semi-lethal effects (Lopez and Lopez-Fanjul 1993a, b; Mackay et al. 1992), such that the quasineutral, potentially adaptive mutational variance is roughly one-tenth as large as previous estimates. Lande (1995) therefore suggested that the Franklin-Soulé number should be increased by a factor of 10, to $N_e = 5{,}000$. Maintenance of rare alleles with major effects on disease resistance may require much larger populations (Roush and McKenzie 1987). Nevertheless, populations that do not meet these simplistic criteria are not necessarily hopeless for conservation. First, if a population is well adapted to a relatively constant environment, then there may not be much need for adaptive evolution. Second, in a small population that recovers to a large size, mutation can restore genetic variance and adaptability (Table 2). There are several examples of populations or species that have recovered after reduction to small numbers, such as the northern elephant seal (Hoelzel et al. 1993) and American bison (Miller 1990 pp. 38–39).

Quantitative (continuously varying) polygenic characters of morphology, behavior and physiology generally are important for current adaptation, future adaptability, and population persistence. Quantitative characters typically are under stabilizing natural selection toward an intermediate optimum phenotype (that may fluctuate with time), such that extreme phenotypes are selected against. Heritable variance in quantitative characters therefore imposes a fitness cost or "genetic load" on a population, which is the price it must pay for future adaptability (Crow and Kimura 1970; Lande and Shannon 1996). Thus there is an optimal level of genetic variance for maintaining both current fitness and future adaptability. Genetic variance in quantitative characters increases fitness and promotes population persistence primarily when environmental change is somewhat predictable, when the optimal phenotype undergoes continued directional change, long-period high amplitude cycles, or substantial temporal autocorrelation (Lande and Shannon 1996).

There is, however, a maximum rate of directional or random environmental change that a population can tolerate by adaptive evolution without becoming extinct, depending on the amount of genetic variability maintained (Lynch and Lande 1993; Bürger and Lynch 1995; Gomulkiewicz and Holt 1995; Lande and Shannon 1996). Rapid, extreme environmental changes, such as anthropogenic global warming, will place a premium on genetic variability and adaptability of populations in fragmented habitats during the coming centuries (see Discussion).

Fixation of new mutations

In contrast to recessive lethal mutations that generally are restricted to low frequencies by natural selection, random genetic drift can fix mildly deleterious mutations in a small population and gradually erode its fitness. Mildly deleterious mutations arise at many loci, with a total genomic rate on the order of one per generation in a variety of organisms. Such mutations produce an average fitness loss of a few to several percent and are only partially dominant (nearly additive). When enough deleterious mutations are fixed, the population becomes genetically inviable ($r_{max} \leq 0$) and extinction rapidly ensues. For a population at carrying capacity in a constant environment, with no demographic stochasticity, the mean time until genetic inviability from fixation of new mutations scales asymptotically as a power of the effective population size at carrying capacity; the power depends on the coefficient of variation of selection against new mutations (Lande 1994, 1995). For realistic distributions of selection on mildly deleterious mutations, the coefficient of variation is on the order of one (e.g. an exponential distribution of mutational effects on fitness), so the power is not very large (see Table 1, Keightley 1994). Nearly neutral mutations, with selection coefficients close to $1/(2N_e)$, do the most damage to a population, because strongly selected mutations rarely become fixed and more weakly selected mutations have relatively little impact on fitness (Lande 1994; Lynch et al. 1995a, b).

Even for very small populations, hundreds of generations may elapse before fixation of new mildly deleterious mutations causes extinction, if population fitness is initially high. In sufficiently large populations, advantageous, compensatory and reverse mutations will completely prevent the erosion of fitness by deleterious mutations. Thus it is only for small populations with low fitness that the extinction risk from fixation of new deleterious mutations is a serious concern within the typical 100 year time scale

of conservation planning. However, for populations of moderate size, with N_e up to few thousand, fixation of new mutations could substantially decrease their *long-term* viability (Lande 1995).

Discussion

The primary anthropogenic causes of species declines produce a series of ecological and genetic effects that ultimately are expressed, and can be evaluated, in terms of population dynamics and extinction risk. Land development causes habitat destruction and fragmentation, which, along with overexploitation and artificial introductions of exotic species, produces population declines, creating small population effects and intensifying metapopulation dynamics. Demographic and genetic factors affecting small populations are involved with population decline in positive feedback loops or "extinction vortices" (Gilpin and Soulé 1986).

Recovery of an endangered species already reduced to small and/or fragmented populations requires management consideration of all the potential risk factors described above, as well as their interactions. Small population effects usually are more a symptom than a cause of incipient extinction, and treating them without addressing the underlying causes of population decline is not likely to prevent extinction (Lande 1988b; Caughley 1994). Scaling laws for mean time to extinction under different risks (Table 1) support the idea that deterministic population declines of anthropogenic origin are generally of much greater importance than stochastic factors as the main causes of species declines prior to their becoming endangered. This is especially important because it is often possible to understand the causes of deterministic declines and to reverse them through restoration and management actions (Caughley and Gunn 1996).

Habitat destruction and fragmentation restrict dispersal, and eliminate species ability to change their geographic distribution, which for many species was the most important mechanism for population persistence in response to long-term climatic alterations (Pease et al. 1989; Peters and Lovejoy 1992). In response to previous periods of global warming and cooling associated with glacial cycles, species often changed their geographic range while maintaining essentially the same phenotype, except perhaps for evolution in body size (e.g. Coope 1979; Smith et al. 1995). Species restricted to isolated habitat fragments and reserves must instead rely either on their limited physiological tolerances, or on evolutionary adaptation *in situ*, to survive rapid global warming and other environmental challenges in the coming centuries. A small proportion of species may be aided by accidental or deliberate artificial transport. For many species persistence during the next millennium will therefore increasingly depend on maintaining ample genetic variation for adaptive evolution, and on having natural or artificial opportunities for dispersal.

Real conservation problems almost never can be solved by a single discipline alone. Conservation biologists specalizing in evolution, demography and community ecology, should have sufficiently broad knowledge and be humble enough to admit that their speciality is not necessarily the most important for solving any given problem. They must be willing to work not only with specialists from other areas of biology, but also with social scientists, economists, political activists and environmental lawyers. This is often the only effective means of conserving and restoring biological diversity against powerful economic and political interests. Although politicians rarely make plans on time scales longer than decades, conservation biologists must begin planning on time scales of centuries, millennia, and longer, if our attempts to preserve a substantial fraction of existing biodiversity are to have any lasting effect.

Ecologists and evolutionary biologists should spend at least part of their time involved in conservation. There is nothing wrong with scientists using their expertise to advocate a political position supported by objective scientific information, provided that they do not distort the information. Scientists are also citizens and human beings, who have a right to be movited not only by a desire for knowledge and truth, but also by aesthetic and emotional reasons to conserve and restore nature. If conservation biologists do not advocate the cause of biological diversity and the environment, then the myopic forces of ignorance and destruction will eventually ruin what remains of the natural world.

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