to environmental perturbation, or are so small that random birth and death events and genetic drift threaten them. The number of species in this set of vulnerable populations will depend on many factors—especially the size of the reserve, because the larger the reserve, the smaller the proportion of species that will have marginal population sizes. If choices must be made among candidate species, then the ecological role of the species is perhaps the most important criterion. Large predators and other keystone or mutualistic species such as some trees (Chapter 15) and pollinators (Chapters 19 and 21) are automatic targets.

If "Nature knows best" and Nature abhors close inbreeding (Chapter 3), then managers should too. Every rule has its exceptions, however, including this one—especially when a past history of inbreeding requires extreme remedies, even the purging of certain recessive deleterious genes from a group (Chapter 6).

Chapters 4 and 5 suggest that any measurable increase in homozygosity is likely to extract a cost in the currencies of immediate fitness and long-term adaptability. But even more dangerous than inbreeding depression is the kind of "depression" that afflicts some agencies when it appears that a population is too small to save. The point is that even when numbers are very low recovery is possible, given the will and the resources. Theory indicates that most genetic variation (though not the rare alleles) can be saved from even a handful of individuals. In such cases, it is usually financial resources, not genetic resources, that is the limiting factor.

Finally, genetic systems are too heterogeneous to allow generalizations about the dangers of outbreeding depression in specific cases. Managers of threatened or endangered species, whether the species are captive or managed in the wild, must be aware of the hazards discussed above and in the following chapters. The problems range from meiotic disturbances and sterility in the F_1 generation to quantitative reduction in developmental homeostasis, viability, local adaptation, and host recognition, to inappropriate timing of reproduction (Templeton, Chapter 6). Anticipating all the potential problems may be impossible, but chromosomal surveys and autecological studies could eliminate most sources of failure.

M.S.

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CHAPTER 2

MINIMUM VIABLE POPULATIONS: PROCESSES OF SPECIES EXTINCTION

Michael E. Gilpin and Michael E. Soulé

The term "minimum viable population" has come into vogue, possibly because of an injunction from the Congress of the United States (National Forest Management Act of 1976) to the U.S. Forest Service to maintain "viable populations" of all native vertebrate species in each National Forest. The term implies that there is some threshold for the number of individuals, or some multivariate set of thresholds and limits, that will insure (at some acceptable level of risk) that a population will persist in a viable state for a given interval of time. This chapter introduces the term "population vulnerability analysis" (PVA) for analyses that estimate minimum viable populations (MVPs). That is, MVP is the product, and PVA the process.

Earlier investigations of the MVP problem, including MacArthur and Wilson (1967), Richter-Dyn and Goel (1972), and Leigh (1975), emphasized a demographic approach, in which the expected lifetime of a population was the objective. Their work was based on birth and death branching processes, and they found that there were critical "floors" for size, below which the population would quickly go extinct. A second, more recent body of work (for reviews see Frankel and Soulé, 1981, and Schoenwald-Cox et al., 1983) has focused on genetic aspects of the population extinction question; again, the findings support the existence of critical factors of population size and population structure, below which inbreeding and loss of selectable variation become a problem for the continued survival of the population.

A paper by Mark Shaffer (1981) appears to have been the first to take an overall systems perspective. He began by distinguishing deterministic extinction from chance or stochastic extinction. Then he distinguished four separate forces, or kinds of variation, that independently contribute to population extinction. The first two, demographic stochasticity and genetic stochasticity, were just mentioned. The second two, identified by Shaffer, were environmental stochasticity (environmental shocks received by all members of a population), and catastrophes. Despite this comprehensive view, however, Shaffer used only a combination of demographic and environmental stochasticities in his study of the grizzly bear (Shaffer, 1983).

Estimating MVPs is complex. Efforts to consider all four effects jointly and, beyond these, to consider the consequences of habitat fragmentation, have been slow to develop. As elaborated in this chapter, the probability of extinction cannot be pegged to population size alone. In addition, each situation will have a set of "minima" (Salwasser et al., 1984; Soulé, in press), depending on the life history of the species (population), the temporal and spatial distribution of its resources, and its level of genetic variation. In other words, there will be no "magic number," no single MVP that is universally applicable to all species.

These considerations might appear to undermine the idea of MVPs, but we would not agree that the term should be abandoned. First, it is already widely used, and it focuses attention on a critical problem. Second, its disappearance or repudiation would have deleterious effects on conservation. Third, it conveys three important concepts:

1. It defines the single species population as the unit of study.[1]
2. The term "viability" stresses that we are concerned with the persistence of the population over some relatively long temporal interval.
3. The idea of "minimum" suggests that there are critical aspects of the "population-in-its-environment," whether involving its total size, its distribution, or some feature of its genetics, that govern its probabilistic decay from existence to extinction.

In this chapter we take a conceptual, pluralistic overview of the process of species extinction. We do this to provide a framework for the integration of ecological, population dynamic, and population genetic models that must be brought to bear on this vital issue. We base our efforts on the seminal analysis of Shaffer (1981), but we go beyond Shaffer in two important ways. We not only identify the components of population vulnerability analysis (PVA), but we examine the feedback loops by which the decay in one factor (such as population size) can exacerbate not only itself but also the behavior of other factors (such as inbreeding and fragmentation).[2]

A general conceptual model of the viable population problem is important to a number of concerns in conservation biology. Captive populations must be kept above the MVP that assures retention of genetic variation and fitness. Ecological preserves must be large enough to provide for the minimum viability of their important species. MVP also plays a role in the emerging discipline of ecosystem restoration, because it may govern the composition and timing of the reintroduction of extirpated species. The following general model of viability and its decay is intended to be a guide for further work, not a solution, *per se*, of the MVP problem.

[1] But, as will be discussed later in the chapter, this unit may not apply to systems of patch populations (i.e., "metapopulations").

THE DYNAMICS OF SPECIES VIABILITY

Species extinction is a systems phenomenon, involving the interaction of processes and states. In the remainder of this chapter, we will attempt to disentangle some of the major processes that lead to species extinction. At the very least, our exercise provides a checklist for assessing the completeness of a PVA for a particular species. Our approach conceptually integrates the more exact and quantitative models for the demographic, environmental, and genetic mechanisms that contribute to species extinction (Soulé, in press).

For reasons that will become apparent, we view PVA to be based on three interacting fields, the states of which are constantly changing and interacting. "Population phenotype" (PP) includes all of the physical, chemical and biological manifestations of the population. A second field, the environment (E), is the context. It includes all aspects of the abiotic and biotic factors that influence the population. Together, these two fields determine the third field: the "population structure and fitness" (PSF). This is the field in which the dynamic consequences of the interactions of population phenotype and the environment are manifested. Table 1 lists the components of each of these fields, and Figure 1 represents their overlaps and interactions schematically.

[2] We see population vulnerability analysis as a touchstone for the validity of much of the extant theory in population biology. PVA implicitly asks whether current theory is sufficiently accurate and comprehensive to make long-term predictions as to the probable persistence of populations.

TABLE 1. Components of the three fields of population vulnerability analysis (PVA).

Field	Components of the field[a]
Population phenotype (PP)	Morphology: Variation of sizes, shapes, and patterns Geographic and temporal variation Physiology: Metabolism Metabolic efficiency Reproduction Disease resistance Behavior (intra- and interspecific): Courtship and breeding Social behavior Interspecific interactions Behavior (distribution): Dispersal Migration Habitat selection
Environment (E)	Habitat quantity Habitat quality: Abundance (density) of resources Abundance of interacting species Patterns of disturbance (duration, frequency, severity, and spatial scale of disturbances)
Population structure and fitness (PSF)	Dynamics of spatial distribution: Patch distribution Metapopulation structure and fragmentation Age structure Size structure Sex ratio Saturation density Growth rate (r) Variance of r: Individual Within patches Between patches

[a]The categories listed for each field are meant to be suggestive, not comprehensive.

Population phenotype

The population phenotype field can be divided into four sections. Although these sections are somewhat artificial, they have heuristic value. The first section of this field is the most concrete; it is the *morphology* section. Here we are dealing with the tangible, physical, static representations of size, volume, color, texture, and shape of morphological structures. These are things that can be stored in museum cabinets.

The other three sections or components of the PP field are dynamic; they are the physiological and behavioral processes of individuals. The physiology section is *metabolism* in the broadest sense, including nutrition, coordination, reproductive processes such as gametogenesis, and many aspects of disease resistance. A third section includes sexual, courtship, breeding, and social *behaviors*. It also includes the behavioral aspects of interactions with other species (prey, predators and competitors). The fourth section includes those behaviors (migration, taxes, dispersal) that govern the choice of and *movement* between different habitats.

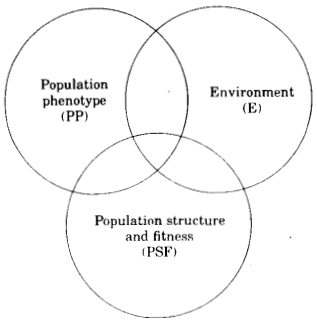

FIGURE 1. The three interacting fields of analysis in population biology: population phenotype (PP), environment (E), and population structure and fitness (PSF). See text for complete discussion.

Environment

It is useful to examine the environment field from two perspectives—*quality* and *quantity*, because a deterioration of either can extinguish a population. While both quantity and quality contribute to the number of individuals that will be found in the associated region, each does it in a different way (though this distinction may collapse under certain circumstances, as discussed below). For terrestrial species, "quantity" refers to the amount of habitat available to the population,

everything else being equal. Environment or habitat quantity scales the total population size and may influence aspects of its distribution, including how it responds to a perturbation of environment quality.

Environment quality comprises everything extrinsic that determines the adaptedness of the species including the relative fitness of the individuals. It interacts with the population phenotype to establish the population density or carrying capacity. Quality includes the states of the physical environment, the abundance of resources (food, nutrients, shelter, mutualists, breeding sites), and the kinds and numbers of interacting species (competitors, predators, herbivores, and disease organisms). Environmental quality also has dynamic components; it includes the patterns of change in all of these factors. In other words, quality depends on disturbance dynamics as much as it does on average conditions.

From the viewpoint of population vulnerability analysis, the disturbance regime where a population lives is often the most important aspect of the environment. This is because many of the populations that are subject to PVA will be restricted to island-like habitats such as parks and refuges, and they will be unable to escape to other refugia when the environment deteriorates (Peters and Darling, 1985). Therefore, a PVA must anticipate the "worst-case" eventualities for each targeted population in each place.

The complex nature of environmental quality notwithstanding, a good naturalist is often able to summarize his or her habitat knowledge of a species, using such categories as "marginal" to "central," or "good, medium, and bad." Such subjective judgments about environment quality will often be our best and only guides to the relative merits of particular sites.

Population structure and fitness

Population phenotype and the environment interact to produce what we call PSF. These are the measurable aspects of population structure and fitness: the population's age structure, sex ratio, size structure, and the distribution of individuals over time and space. In this field we also include the dynamic changes in these variables, which give the population growth rate (r) and the saturation density of the population.

DETERMINISTIC AND STOCHASTIC EXTINCTIONS

The goal of a population vulnerability analysis is to establish a minimum viable population that reduces the risk of an extinction to an acceptable level. In this quest, it helps to classify extinctions into the two kinds mentioned above, deterministic and stochastic. Deterministic extinctions are those that result from some inexorable change or force from which there is no hope of escape. Deforestation and glaciation are two such forces for many species of trees. For monkeys, the disappearance of all the trees in a region is such a force. For the species-specific parasites of monkeys, the death of all the monkeys is such a force.

A deterministic extinction occurs when something essential is removed (such as space, shelter, or food), or when something lethal is introduced (such as too many cats or hydrogen ions). Deterministic extinctions can be linked. For example, the extermination of the dodo bird by humans doomed a Mauritian plant species to extinction (Temple, 1977), because only by passage through the gut of the dodo could the seeds of this plant be primed for germination. Loss of habitat has already been a major factor in the extinction of a number of tropical plant species (Gentry, Chapter 8). The total extinction of a species due to habitat loss, however, can only occur (1) when the species is a local endemic, (2) when it is already on the verge of extinction, or (3) when humans or global events cause wholesale habitat destruction.

Stochastic extinctions are those that result from normal, random changes or environmental perturbations. Usually such perturbations thin a population but do not destroy it; once thinned, however, the population is at an increased risk from the same or from a different kind of random event. The smaller a population, the greater its vulnerability to such perturbations. Also, the shorter the interval between such events, the more likely the population will be pushed over the brink before it can recover to a safe size.

The following section will emphasize stochastic extinctions, because the processes involved are more subtle and more difficult to observe and defend against. As with most dichotomies, however, this one can be misleading. Many extinctions are the result of a deterministic event that brings the population into a size range where rather frequent or probable stochastic events can easily terminate it. For example, habitat destruction or overharvesting will reduce a population to the point where a stochastic extinction is inevitable (see Soulé and Simberloff, 1986, for examples).

THE FOUR EXTINCTION VORTICES

We are proposing that any environmental change can set up positive feedback loops of biological and environmental interactions that have further negative impacts on the population, possibly leading to its extinction. We refer to these event trains as "extinction vortices." The

first question is, "What are the changes that can bring a population under the influence of one of these vortices?"

Figure 2 attempts to capture such events—where a major loss of habitat causes reductions in population size, N, and in population distribution, D.[3] The consequences of reductions in N and D are shown at the bottom of Figure 2. One possible outcome is the immediate (deterministic) extinction of the population. If the population survives, it will probably be more fragmented and will suffer an increase in demographic stochasticity, and an increased chance of stochastic extinction.

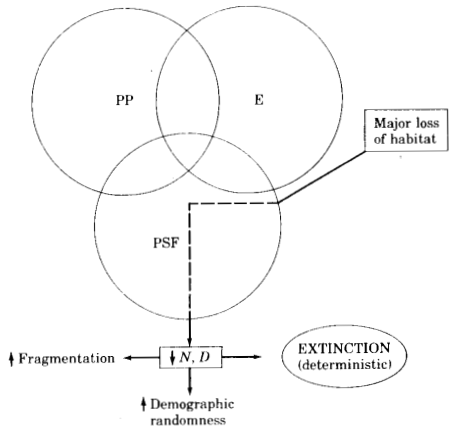

FIGURE 2. Consequences of a major loss of habitat. The population suffers reductions in population size (N) and in distribution (D). One possible outcome is the immediate deterministic extinction of the population. Even if the population survives, it will be increasingly vulnerable to stochastic extinction. ($\uparrow$ = increase; $\downarrow$ = decrease.)

[3] For the purposes of this chapter we characterize the population distribution with a single variable, D, where this is a summary statistic that describes the intensity of the fragmentation—i.e., the number of patches and the degree of their isolation. We recognize that fragmentation has both genetic and population dynamic consequences (Gilpin, in press a) and that its description may require more than a scalar variable.

Demographic stochasticity

"Demographic stochasticity" is often the immediate precursor of extinction. It is defined as the chance variation in individual birth and death. It is assumed to be independent for each individual. In a small population, extinction can occur accidentally (randomly) because of high death rates or low birth rates. A population is not safe from such a chance failure in recruitment until it has a large number of members. For an organism that reproduces by fission, such as a microorganism, MacArthur and Wilson (1967) found a critical limit of 10 individuals. For a dioecious organism with a more complex life history, including a prereproductive period, Shaffer (1981) obtained numbers in the range of 50. Whenever a population is drawn down to this size, it is in danger of falling prey to demographic stochasticity.

Very small populations are therefore in extreme jeopardy. In Chapter 12, Lovejoy et al. show that 10-ha patches of Amazonian forest are large enough to contain only one or two groups of red howler monkeys (*Alouatta seniculus*), with up to about 10 individuals in each group. Such groups are not likely to persist for more than a few generations, according to population biology theory.

Environmental stochasticity

There are two general routes to the domain of strong demographic stochasticity. The first is a decrease in habitat quantity. The second is disturbance, or a deterioration in environmental quality. Most changes in quality fall under the heading of environmental stochasticity.

"Environmental stochasticity" is the random series of environmental changes. In models, a frequent assumption about environmental variation is that all individuals in a patch feel these perturbations in an equivalent way, and that regardless of the initial population size, a series of negative blows can reduce the population by orders of magnitude and bring it to a state where demographic stochasticity can take hold. The distribution of the species in space, however, may tend to ameliorate this, because a large range may introduce ecological heterogeneity that buffers the action of the perturbations.

Figure 3 adds two additional state variables. It suggests that a perturbation can reduce r (for example, by reducing per capita resources, and via mortality by affecting age structure). It also shows that the genetic effective population size, N_e, is likely to be reduced.

Both effects—a lower N_e and increased demographic stochasticity—have consequences or "outputs," to use systems theory terminology. The output of a lower N_e is an increase in genetic drift and inbreeding.

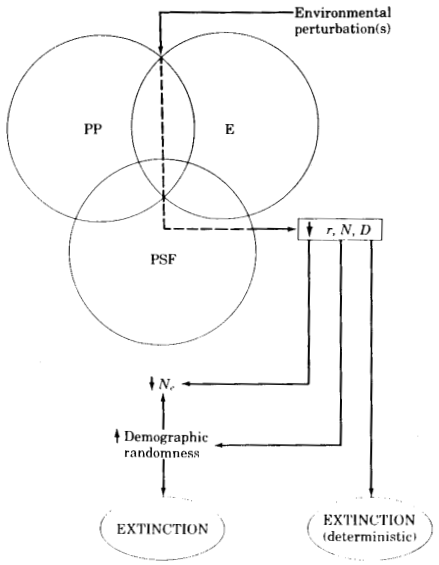

FIGURE 3. The effects of one or more environmental perturbations. These can affect the population's growth rate *(r)* and also reduce the genetic effective population size *(N_e)*.

while the output of increased demographic stochasticity is manifested as an increase in the variance of the population growth rate, denoted *Var (r)*.

Var (r) has been employed to calculate extinction probabilities. This study was pioneered by Feller (1957) and was developed further by MacArthur and Wilson (1967), Richter-Dyn and Goel (1972), Leigh (1981), and Goodman (in press). *Var (r)* should not be viewed as a simple manifestation of any one ecological factor. Rather, it is the distillation of all of the forces acting on and through a population. We place *Var (r)* under PSF, because it is neither part of the population phenotype, *per se*, nor is it part of the environment. Rather, it is a statistic that depends on the interaction of these two fields over time.

These outputs lead to further changes of state in the field of PP—where they can modify the population's genetics and phenetics—and then to the field of PSF, where population dynamics are affected, with the possibility of deleterious effects on N, N_e, r, and *Var (r)*. We distinguish four such loops: the R, D, F, and A vortices. It should also be emphasized that the pathways of responses we are sketching can be traveled multiple times and can become complex and interconnected.

The R vortex

The R vortex is triggered when chance lowerings of N and increases of *Var (r)* make the population vulnerable to further disturbances, in turn reducing N further and increasing *Var (r)*. That is, the severity of the impact of a disturbance may be exacerbated by the current states of N and r, with a series of otherwise similar disturbances having progressively more serious consequences on the population (Figure 4). An example of this would be a case where the early disturbances alter the age structure of a population in such a way as to make it more vulnerable to subsequent disturbances. Or, an alteration

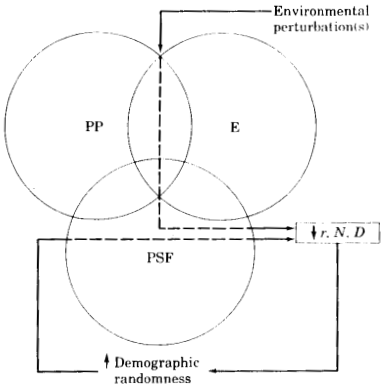

FIGURE 4. The demographic (R) vortex is triggered when chance lowerings of N and increases of *Var (r)* make the population vulnerable to further disturbances, which in turn reduce N further and increase *Var (r)* still more.

of the sex ratio away from 50:50 could lead to even greater variation in birth and death rates (due, for example, to increased difficulty in finding mates). The effects of low N over a number of generations can, in a sense, be cumulative. That is, the probability that a small population will be extinguished by demographic stochasticity alone increases at a rate that is greater than the linear sum of the per generation probabilities of extinction.

The D Vortex

A lowering of N and an increase in $Var\ (r)$ can alter the spatial distribution of a population and can increase the patchiness of its distribution (Figure 5). Fragmentation has a number of detrimental implications. First, because the probability of extinction of a local patch varies inversely with the population size on the patch (Gilpin and Diamond, 1976), more fragmented distributions are likely to increase further the rate of local extinction, further exacerbating the problem of isolation.

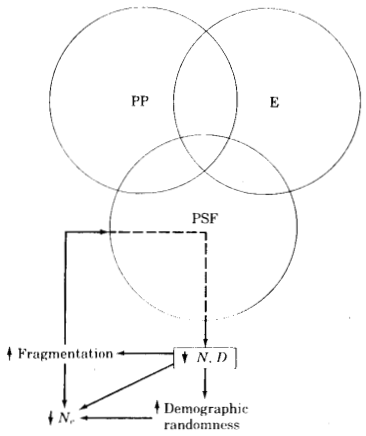

FIGURE 5. The fragmentation (D) vortex. The effects of the R vortex can alter the spatial distribution of a population, introducing or increasing fragmentation. More fragmented distributions increase the likelihood of local extinctions, as well as having detrimental effects on N_e.

Second, and less obviously, fragmentation with turnover of patch populations has profound negative consequences for N_e, potentially producing effective population sizes that are orders of magnitude lower than the actual total census count (Maruyama and Kimura, 1980; Gilpin, in press b).

The F vortex

A decrease in N_e can have far-reaching consequences for a population, especially if the low N_e persists for many generations. If severe enough, such reduced sizes can lead to the initiation of two genetically-based vortices. Both vortices are the consequences of increased genetic drift and the loss of heterozygosity and genetic variance.

The F vortex is predicated on the well-established connection between inbreeding and inbreeding depression (Frankel and Soulé, 1981; Ralls and Ballou, 1983) and also on the frequent, direct relationship between individual heterozygosity and individual fitness (Allendorf and Leary, Chapter 4; Ledig, Chapter 5). Inbreeding depression and the loss of heterozygosity probably undermine most components of the population phenotype, including metabolic efficiency, growth rate, reproductive physiology, and disease resistance. In turn, these affect the schedule of births and deaths that determine r in the PSF field. These effects are relatively "hard," or absolute, because their impact is more or less independent of the environment. Lower r and N further reduce N_e, completing this feedback loop (Figure 6), and increasing the probability of extinction via this vortex and the others.

The A vortex

The last vortex is also the consequence of genetic drift and loss of genetic variance, but it manifests itself differently. Genetic drift can affect the precision with which selection can "tune" a population to its environment. Any process or event that decreases N_e will also reduce the efficacy of stabilizing and directional selection, in turn causing an increasing and accelerating lack of fit between the population phenotype (PP) and the environment it faces (E). This reduces r and N still further, draws the population still deeper into a vicious cycle, and exacerbates all of the other vortices at the same time.

For example, a reduction in fitness (from either the F or A vortices, or both) may increase fragmentation, with all of the derivative effects mentioned above. In part, this may occur because marginal habitats or patches become submarginal as fitness deteriorates. Second, and

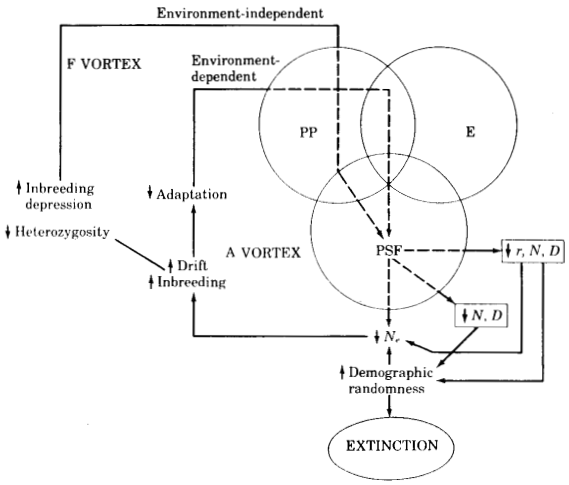

FIGURE 6. The inbreeding (F) and adaptation (A) vortices. Both are the consequences of increased genetic drift and the loss of heterozygosity and genetic variance. The F vortex reflects inbreeding depression and the effects of loss of heterozygosity on phenotype viability (metabolic efficiency, growth rate, reproductive efficiency, disease resistance, etc.) and eventually on r. The A vortex reflects that a decrease in N_e brought about by genetic drift will affect the ability of a population to adapt to its environment, causing an increasing lack of "fit" between the population phenotype (PP) and the environment (E).

probably more important, loss of genetic variance may make it increasingly unlikely for a population to track environmental changes genetically (Frankel and Soulé, 1981; Futuyma, 1983). The A vortex has the longest time scale, meaning that the other vortices will often lead to extinction before the population suffers serious genetic erosion and loss of adaptation.

To briefly illustrate the interaction of these positive feedback vortices, we use the case of the howler monkey mentioned earlier in this chapter and described in Chapter 12. Assuming that the monkeys are all confined to those 10-ha patches of forest, the consequences include

an overall decline in population size. Each unit of the fragmented population is now isolated. The small sizes of these units mean that they are prone to severe demographic stochasticity and a very high probability of extinction. Small population size and isolation also produce a low effective size and a high level of inbreeding and loss of heterozygosity. In addition, edge effects and other characteristics of the fragmented habitat change the selective pressures on the monkeys. Assuming that one generation is equivalent to one cycle through a vortex, the longer that a population of monkeys survives, the more it cycles through the F and A vortices. Each cycle further reduces its fitness, increasing mortality and decreasing natality. This exacerbates demographic stochasticity (the R vortex), lowering N_e still further. As populations go extinct, the probability of gene flow and recolonization of empty patches is further reduced, exacerbating the D vortex as well as the F and A vortices.

SUMMARY

The foregoing treatment of population vulnerability analysis is general, but we hope it has been exhaustive at the level of systems identification. But even if this systems model is comprehensive, it is far from being a universal protocol for the estimation of minimum viable populations in specific cases.

One reason for this lack of precision is that species will differ in their vulnerability to the four vortices. To some extent, it is possible to predict the vulnerability to these vortices using taxonomic, ecological, and body size categories. For example, small-bodied, high-r insects, fish and rodents—especially those with relatively long-range juvenile dispersal—are unlikely to suffer from the F and A vortices. Usually their total population sizes and their rates of gene flow are great enough to avoid the loss of genetic variation. Besides, their local populations are relatively susceptible to capture by the R vortex because r and $Var\ (r)$ are positively correlated. In other words, extinctions of patch populations of small annual species are likely to occur before the F and A vortices are fully engaged (for example see Ehrlich, 1983).

At the other extreme, large, iteroparous organisms can persist at low population levels for generations, because of longevity and because they are physiologically buffered from short-term environmental changes. Therefore, trees and large vertebrates have relatively high probabilities of being sucked into the F and A vortices.

Lineages have been episodically exposed to one or more of these vortices during their evolution, and therefore may have evolved some kind of "resistance" to them. For example, the high dispersal rates

and high r characteristic of many small organisms may be evolutionary adaptations that minimize the threats of frequent fragmentation (D vortex) and local extinctions (R vortex). At the other extreme, it is conceivable that large-bodied, high trophic level species with chronically low N_e might evolve some resistance to inbreeding depression, such as allelic and physiological redundancy. To our knowledge, however, little evidence exists for such adaptations.

We hope that further elaboration and improvement of this systems approach will lend itself to the management of rare or endangered populations. At this point, our approach can be used as a conceptual checklist for those responsible for the viability of particular species, and as a guide to gathering the sorts of data that may be most useful in assessing the connectedness of the scheme we have sketched.

SUGGESTED READINGS

Frankel, O.H. and M.E. Soulé. 1981. *Conservation and Evolution.* Cambridge University Press, Cambridge. Chapters 4 and 5 give some of the background of genetic thinking regarding minimum viable population sizes. For several reasons the "magic number" of 500 may be simplistic, but it is still considered to be about the right order of magnitude for many applications.

Shaffer, M.L. 1981. Minimum population sizes for species conservation. *BioScience 31:* 131–134.

Soulé, M.E. In press. *Viable Populations.* Cambridge University Press, Cambridge. The chapters in this book should fill in some of the theoretical details of the black boxes in Figures 3–6 of this chapter.

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INBREEDING IN NATURAL POPULATIONS OF BIRDS AND MAMMALS

Katherine Ralls, Paul H. Harvey and Anna Marie Lyles

Mating between close relatives increases the proportion of loci at which offspring are homozygous because such pairs are genetically more similar to each other than are pairs of individuals taken at random from the population. This increase in homozygosity can cause inbreeding depression. Although few data from natural populations are available, studies of captive and experimental animals consistently confirm the ubiquity and magnitude of its effects (Ralls and Ballou, 1983).

Packer (1979) lists three reasons why inbreeding depression should be expected to occur. First, "increasing homozygosity increases the chances of a detrimental recessive being expressed." Second, "the heterozygote may sometimes be fitter than either homozygote." And third, "an increase in homozygosity decreases the variability between offspring, with the effect that the chance of any one of an individual's progeny surviving a sudden environmental change may be reduced" (Williams, 1966).

Outbreeding can also entail costs. For example, outbreeding may require dispersal over an unfamiliar environment where the risks of mortality are high; and there may sometimes be genetic costs as well (Templeton, Chapter 6). The costs of inbreeding versus those of outbreeding will vary from species to species, depending on a range of

and high r characteristic of many small organisms may be evolutionary adaptations that minimize the threats of frequent fragmentation (D vortex) and local extinctions (R vortex). At the other extreme, it is conceivable that large-bodied, high trophic level species with chronically low N_e might evolve some resistance to inbreeding depression, such as allelic and physiological redundancy. To our knowledge, however, little evidence exists for such adaptations.

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INBREEDING IN NATURAL POPULATIONS OF BIRDS AND MAMMALS

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Mating between close relatives increases the proportion of loci at which offspring are homozygous because such pairs are genetically more similar to each other than are pairs of individuals taken at random from the population. This increase in homozygosity can cause inbreeding depression. Although few data from natural populations are available, studies of captive and experimental animals consistently confirm the ubiquity and magnitude of its effects (Ralls and Ballou, 1983).

Packer (1979) lists three reasons why inbreeding depression should be expected to occur. First, "increasing homozygosity increases the chances of a detrimental recessive being expressed." Second, "the heterozygote may sometimes be fitter than either homozygote." And third, "an increase in homozygosity decreases the variability between offspring, with the effect that the chance of any one of an individual's progeny surviving a sudden environmental change may be reduced" (Williams, 1966).

Outbreeding can also entail costs. For example, outbreeding may require dispersal over an unfamiliar environment where the risks of mortality are high; and there may sometimes be genetic costs as well (Templeton, Chapter 6). The costs of inbreeding versus those of outbreeding will vary from species to species, depending on a range of