Directions in Conservation Biology
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REVIEW

Directions in conservation biology

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Summary

1. Conservation biology has two threads : the small-population paradigm which deals with the effect of smallness on the persistence of a population, and the declining-population paradigm which deals with the cause of smallness and its cure.

2. The processes relevant to the small-population paradigm are amenable to theoretical examination because they generalize across species and are subsumed by an inclusive higher category : stochasticity.

3. In contrast, the processes relevant to the declining-population paradigm are essentially humdrum, being not one but many. So far they have defied tight generalization and hence are of scant theoretical interest.

4. The small-population paradigm has not yet contributed significantly to conserving endangered species in the wild because it treats an effect (smallness) as if it were a cause. It provides an answer only to a trivial question: how long will the population persist if nothing unusual happens? Rather, its major contribution has been to captive breeding and to the design of reserve systems.

5. The declining-population paradigm, on the other hand, is that relevant to most problems of conservation. It summons an investigation to discover the cause of the decline and to prescribe its antidote. Hence, at least at our current level of understanding, it evokes only an ecological investigation which, although utilizing the rigour of tight hypotheses and careful experimentation, is essentially a one-off study of little theoretical interest.

6. The principal contribution of the small-population paradigm is the theoretical underpinning that it imparted to conservation biology, even though most of that theory presently bears tenuous relevance to the specific problems of aiding a species in trouble. It would contribute immeasurably more if some of the theoretical momentum so generated were channelled into providing a theory of driven population declines, thereby liberating the declining-population paradigm from the inefficiency of case-by-case ecological investigations and recovery operations.

7. The declining-population paradigm is urgently in need of more theory. The small-population paradigm needs more practice. Each has much to learn from the other. A cautious intermixing of the two might well lead to a reduction in the rate at which species are presently going extinct.

Key-words: conservation biology, demographic stochasticity, effective population size, environmental stochasticity, extinction, genetic drift, inbreeding, minimum viable population, population viability analysis.

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Introduction

My purpose in this overview is to chart recent research into the processes of extinction and to gauge the extent to which it has contributed, potentially and actually, to slowing the loss of species. In so doing, I will be retracing some of the lines already sketched by Simberloff's (1988) stimulating review. The thesis advanced by this paper, however, is that conservation biology is presently advancing on two separate fronts with little overlap and that neither approach is likely to achieve its stated purposes alone. I will therefore characterize these alternative approaches to conservation and suggest how they might be reconciled.

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Figure 1 is a graphic example of the extinction problem. It shows for each $2 \times 2°$ block ($c.$ 50 000 km^2) of Australia the number of mammalian species that were there at European colonization, but which are not there now (Woinarski & Braithwaite 1990). Each loss represents a 'local' extinction. If a species disappears from all the blocks of its former range it is, by definition, extinct in the wild. '[T]he extinction problem has little to do with the death rattle of the final actor. The curtain in the last act is but a punctuation mark—it is not interesting in itself. What biologists want to know about is the process of decline in range and numbers' (Soulé 1983, p. 112). Or put another way, 'There is no fundamental distinction to be made between the extinction of a local population and the extinction of a species other than this that the species becomes extinct with the extinction of the last local population' (Andrewartha & Birch 1954, p. 665). That happened over this period of 200 years to 17 species of mammals in Australia, to 1–3 species of birds depending on classification, to no species of reptiles, to several species of frogs, and to 117 species of vascular plants.

An alternative and more familiar process of extinction is the loss of species whose ranges occupy islands, either literally or figuratively. These species tend to disappear as units rather than being progressively eroded out of a region. However, for all extinctions, potential and actual, ranging from the loss of species distributed across continents to loss of point endemics, we can ask the same questions: how do we investigate and explain such losses, and how do we use this knowledge to prevent further losses?

The saving of a species from extinction has always been a paramount responsibility within the field of biology, but since the mid-sixties there has been a rising tide of interest in this area, reaching a flood in the 1980s. There is no doubt that the new conservation biology differed from the old, but the sense of that difference is less easily defined. Gavin (1986) reckoned it as between applied and pure research orientations. I refuse to recognize that awkward distinction, and prefer to differentiate between creative and trivial research, which is not the same thing.

TWO CONSERVATION PARADIGMS

I see it differently from Gavin (1986) while acknowledging that he has secured a portion of the truth. A reading of the literature on conservation biology over the last 20 years will reveal that the field is advancing on two fronts backed by two sets of ideas that have little overlap. These have most of the characteristics of what Kuhn (1970) called scientific paradigms, and that word is used here without pejorative connotation. The first set of ideas, called here the small-population paradigm, deals with the risk of extinction inherent in low numbers. The second set, the declining-population paradigm, is concerned instead with the processes by which populations are driven to extinction
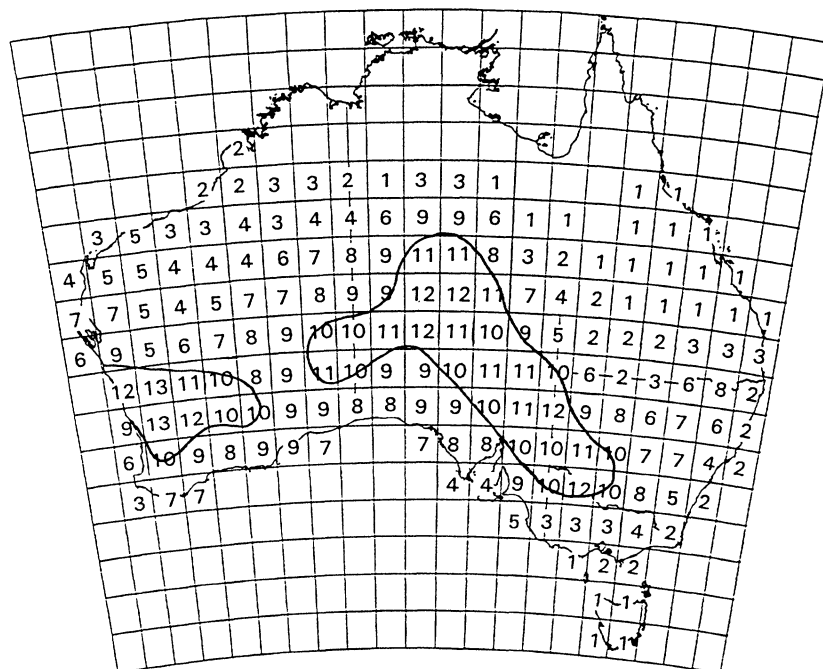
Fig. 1. The number of mammalian species that were in each 2x2° block ($c.$ 50 000 km^2) of Australia at European colonization but which are not there now (Woinarski & Braithwaite 1990). Contours enclose regions which lost 10 or more species from each block.

by agents external to them. They will be discussed at length later, but here follows a summary of their characteristics.

The small-population paradigm

The new ideas about conservation emerging in the 1980s were almost without exception produced from within a small-population paradigm, the concern being with the population consequences of rareness or smallness as such. The Soulé books (Soulé & Wilcox 1980; Frankel & Soulé 1981; Soulé 1986, 1987a) are its display cabinet. This paradigm deals largely with the population genetics and population dynamics problems faced by a population at risk of extinction because its numbers are small and those numbers are capped. A population on a small island, or its analogue in a zoo, transmits the appropriate image.

An integration is supplied to at least part of the small-population paradigm by the 'extinction vortex', that compound snare of positive feedback loops by which inbreeding depression, demographic stochasticity and genetic drift might combine to render a small population smaller. The small-population paradigm is well served by theory—this is its strength—but its links to actuality are as yet poorly developed. It tends to fall over when pressed too hastily into service.

The declining-population paradigm

The declining-population paradigm encapsulates the alternative approach that began earlier and runs parallel with the other. It focuses on ways of detecting, diagnosing and halting a population decline. By this paradigm the problem is seen as a population in trouble because something external to it has changed, the current size of the population being of no great relevance. The research effort is aimed at determining why the population is declining and what might be done about it.

Because the declining-population paradigm is rooted in empiricism it provides most of the means by which practical conservation problems might be solved. Its weakness lies in an almost total lack of theoretical underpinning. It comprises mainly case-by-case ecological investigations and recovery operations, often short on scientific rigour, that provide few opportunities for advancing our general understanding of the processes of extinction.

The small-population paradigm: theory

We first examine the tools of trade and the concerns characterizing the small-population paradigm, asking what would be the major worries exercising the minds of people charged with managing small populations with capped numbers. It may be noted that my list is jerky, a set of disparate ideas being abutted with little logic to their sequence or attention to their integration. At least in part that is the nature of the beast because the small-population paradigm is largely defined by its methods.

DEMOGRAPHIC STOCHASTICITY

The dynamics of a small population are governed by the specific fortunes of each of its few individuals. In contrast, the dynamics of a large population are governed by the law of averages. In a very small population, if a female produces male offspring for three consecutive years and then dies herself, the population may die out.

It is easy enough to simulate that kind of effect with a computer's generator of random numbers grinding out draws from binomial distributions, but easier to approximate it by considering the amount of stochastic variance in rate of increase r attributable to the demographic behaviour of an average animal. This quantity is here symbolized V_{d1}. For a mammal with a moderate intrinsic rate of increase, that individual contribution to the variance of the demographic behaviour of the population is about $V_{d1} = \frac{1}{2}$. The variance in rate of increase of a population of size N is simply $V_d = V_{d1}/N$. Table 1 shows that a population, which would increase at $r = 0.3$ if it had a stable age distribution appropriate to fixed schedules of age-specific fecundity and survival, needs to be moderately large before the rate of increase stabilizes. It shows further that a small population, although 'trying' to increase at $r = 0.3$, is likely to suffer erratic swings of growth and decline that might knock it out before it escaped from the pit of low numbers.

It is an established result (e.g. MacArthur & Wilson

Table 1. The effect of demographic stochasticity on rate of increase for various population sizes N, where expected $r = 0.3$ and $V_{d1} = \frac{1}{2}$. V_d is the variance in rate of increase r caused by demographic stochasticity and s_d is the corresponding standard deviation

N	V_d	s_d	95% Confidence limits of r	
			lower	upper
1	0·500	0·707	—	—
2	0·250	0·500	−6·053	6·053
3	0·167	0·408	−1·457	2·057
4	0·125	0·354	−0·825	1·425
5	0·100	0·316	−0·578	1·178
10	0·0500	0·224	−0·206	0·806
20	0·0250	0·158	−0·031	0·631
30	0·0167	0·129	0·036	0·564
40	0·0125	0·112	0·074	0·526
50	0·0100	0·100	0·100	0·500
100	0·00500	0·071	0·161	0·439
200	0·00250	0·050	0·202	0·398
300	0·00167	0·041	0·220	0·380
400	0·00125	0·035	0·231	0·369
500	0·00100	0·032	0·238	0·362

1967; Lande, in press) that, under demographic stochasticity alone, mean time to extinction increases with intrinsic rate of increase r_m and, if the population is bounded above as in the logistic or truncated exponential model, persistence time exhibits an upwardly concave trend (nearly exponential) when regressed on that upper bound K (Fig. 2: dashed line).

ENVIRONMENTAL STOCHASTICITY

May (1973a) introduced the term environmental stochasticity to cover the effect of environmental fluctuation upon a population's higher order demographic parameters, differentiating it from demographic stochasticity which reflects the uncertainty of individual fortunes.

The effect on rate of increase of the environment fluctuating from year to year will be much the same whether the population is large or small. It is measured as V_e, the variance in r resulting from variation of environmental conditions with time. It is likely to act on r mainly through its effect upon renewable resources, grass eaten by herbivores for example. Food supply in a variable environment can range from one year to the next between the extremes of dearth and plenitude irrespective of how many animals are eating it. Robertson (1987), for example, showed that over 4 years the herb layer averaged across 800 km² of the Australian arid zone varied between 8 kg ha⁻¹ and 1150 kg ha⁻¹ as a consequence largely of the highly variable annual rainfall. When translated to the dynamics of the associated herbivores (Bayliss 1987; Caughley 1987), those levels of plant biomass would cause the annual rate of increase of red kangaroos to vary between $r = -1.49$ and $r = 0.40$.

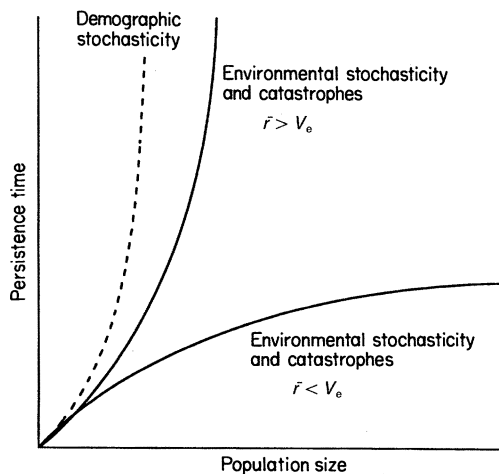The environment may also influence a population's rate of increase by working directly upon mortality without resources acting in intermediary fashion. Avalanche, predation and fire are such agents.

Year-to-year environmental variation acting upon the dynamics of a population can produce counter-intuitive effects. May (1971, 1973b) warned that even in the simplest of population models the addition of environmental stochasticity may generate fluctuations in numbers that become progressively more severe. When $V_e > 2\bar{r}$ 'the probability for the system to become extinct tends to unity as time tends to infinity' (May 1971), even when the population has a positive $\bar{r}$. Figure 3 illustrates these effects: the trend of a modelled population whose successive rates of increase are random draws from a normal distribution of zero mean and unit variance. The position of the origin may be viewed as arbitrary because the trace can be raised or lowered by adjusting its starting value.

May (1971, Appendix 4) showed that under the influence of environmental variability the mean rate of increase over the long run $\tilde{r}$ is lower than the mean $\bar{r}$ of its annual rates. Lande (in press) gives the relationship as

$$\tilde{r} = \bar{r} - V_e/2.$$

He showed that the curve relating population persistence time to carrying capacity will curve upward (as with demographic stochasticity) if $\bar{r} > V_e$, but will be convex if $\bar{r} < V_e$, and that the scaling rules are similar in kind where the environmental stochasticity comes in the form of randomly timed catastrophes (Fig. 2).

Those findings overtake the previous consensus (e.g. Ewens *et al.* 1987; Goodman 1987; Pimm & Gilpin 1989; Soulé & Kohm 1989; Hedrick & Miller 1992; Stacey & Taper 1992) that random catastrophes pose a greater threat to a population than does environmental stochasticity, which in turn is more important than demographic stochasticity. In fact, the relative importance of the first two depends on the details of frequency and force of catastrophes as against the variance in r imparted by environmental stochasticity. In the light of these findings the artificial distinction between catastrophes and environmental variation might perhaps be allowed to wither away.

Fig. 2. The shape of the curves relating persistence time to population size under the influences of demographic and environmental stochasticity, as determined by Lande (in press). V_e is the variance in rate of increase attributable to environmental fluctuations and $\bar{r}$ is mean rate of increase.
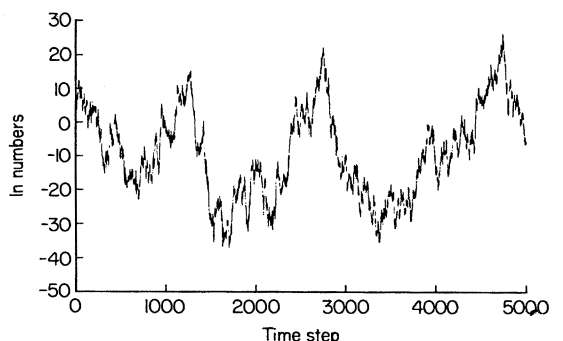
Fig. 3. The trend of a modelled population whose successive rates of increase r are drawn at random from a normal distribution with zero mean and unit variance.

It may be noted further that a comparison between the independent effects of demographic stochasticity and environmental stochasticity has little utility. The first is always operating and the second simply adds to that background variation in *r*.

HETEROZYGOSITY AND FITNESS

If p_{ij} is the frequency of allele *i* at locus *j* in the population as a whole, the proportion of individuals heterozygous at that locus may be estimated as

$$h_j = 1 - \Sigma p_{ij}^2$$

providing that the number of individuals n_j examined for locus *j* is greater than 30. If fewer, h_j is under-estimated, but can be adjusted by multiplying by the small sample corrector $2n_j/(2n_j - 1)$. Mean heterozygosity is then estimated as

$$H = (1/L)\Sigma h_j$$

where *L* is the number of loci examined. *H* is the proportion of loci heterozygous in an average individual. It varies considerably between species for reasons that are not understood. *H* must not be confused with *P*, the proportion of polymorphic loci within the population's genome. *P* usually exceeds *H* by a factor of about 4. Unfortunately, the estimate of *P* is unstable because it increases with sample size, and it is less closely related to additive genetic variance than is *H*. For these reasons it is not considered further. Table 2 gives a frequency distribution of *H* for mammals. The data are from the compilation of Nevo, Beiles & Ben-Shlomo (1984), but averaged values of *H* are used here where a species is represented

Table 2. Frequency distribution of mean heterozygosity *H* for 169 mammalian species. Data are from Nevo *et al.* (1984)

H	Number of species	% of species
0·000–0·009	30*	18
0·010–0·019	22	13
0·020–0·029	26	15
0·030–0·039	17	10
0·040–0·049	21	12
0·050–0·059	13	8
0·060–0·069	7	4
0·070–0·079	8	5
0·080–0·089	9	5
0·090–0·099	4	2
0·100–0·109	2	1
0·110–0·119	2	1
0·120–0·129	2	1
0·130–0·139	2	1
0·140–0·149	1	<1
0·150–0·159	2	1
0·160–0·169	0	0
0·170–0·179	0	0
0·180–0·189	1	<1

*Eighteen of these 30 (60%) returned *H* = 0·000. The cheetah is not included in this sample.

by more than one value in that list. These frequencies should not be taken too literally. They probably indicate accurately enough the spread of heterozygosity within mammals, but the one-dimensional electrophoretic isozyme analyses upon which these levels are based always miss some allelic variation (Lewontin 1991).

Nevo *et al.* (1984) examined published levels of *H* within 1111 species of plants and animals with an average of 23 loci and 200 individuals per species. They searched for patterns according to taxonomic group, life history traits, habitat, range, behaviour and whether the species occurred on islands or on a mainland. There were certainly clear differences between some taxonomic groups (on average vertebrates have half the heterozygosity of invertebrates, for example), but most of the other comparisons returned statistically non-significant results and those that were significant reflected trivial absolute differences.

Data from ontological studies suggest strongly that individuals with more heterozygosity are fitter than individuals of the same cohort with less heterozygosity (Schaal & Levin 1976; Soulé 1980; Frankel & Soulé 1981; Allendorf & Leary 1986; Ledig 1986; Danzmann, Ferguson & Allendorf 1988). The mechanism need be no more complex than the exposure of and selection against recessive semi-lethals. There is good evidence that individuals within a population that has recently lost some of its heterozygosity are less fit on average than individuals of the same species within populations that have not suffered a recent loss of heterozygosity. Again the mechanism is most likely selection against individuals with recessive semi-lethals exposed by the increased homozygosity. However, there is scant evidence that the individuals of a species with more heterozygosity are fitter than those of another species with less heterozygosity, both values of *H* being at equilibrium (Lande & Barrowclough 1987). One might expect that the first two observations necessarily invalidate the third and that argument has been made, but it is not a logical necessity. The species with the lower mean heterozygosity has not necessarily fixed more semi-lethals than a species with a higher mean heterozygosity. Most loci of most species are occupied by only one kind of electrophoretically detectable allele anyway. There is little utility, given our present level of knowledge, of debating whether a species of *Drosophila* with *H* = 0·14 is less likely to die out than a mammalian species with *H* = 0·04

GENETIC DRIFT

The number of different alleles at a locus in the population as a whole will tend to decrease in the absence of immigration and mutation. Heterozygosity thus decreases also. Its rate of decline is a function of population size *N*, the proportion of heterozygous loci

in the population as a whole being reduced by the fraction $1/(2N)$ per generation (Wright 1931). Over one generation H changes according to

$$H_1 = H_0[1 - 1/(2N)]$$

and over t generations

$$H_t = H_0[1 - 1/(2N)]^t.$$

After $t = 2N$ generations the population's heterozygosity will have dropped to 0.37 (i.e. e^{-1}) of its initial value at time $t = 0$. The loss of additive genetic variance is exactly analogous and conforms to the same equations. With the inclusion of mutation the mean heterozygosity (and additive genetic variance) will change over one generation by an amount ΔH according to

$$\Delta H = -H/2N + m$$

where m is the input of heterozygosity by mutation. Its equilibrium is solved by setting ΔH to zero:

$$H^* = 2Nm$$

which indicates that for any population size N there will be an equilibrium H^* between mutational input to additive genetic variance and loss of it from drift. What varies however is the value of H^*, the equilibrium value to which H converges. It will be higher when N is large and lower when N is small.

INBREEDING

Inbreeding is mating between close relatives. It is intuitively obvious that the smaller the population the more frequent such matings. Inbreeding reduces heterozygosity of the offspring below that of the population as a whole. In the case of mating between full sibs of unrelated parents, the drop in heterozygosity below that of the population will, on average, be 25% or, put the other way, their offspring's heterozygosity H will be only about three-quarters that of an individual taken at random from the population. For the offspring of half-sibs, themselves born of unrelated parents, the loss is $12\frac{1}{2}\%$. Those percentages are inbreeding coefficients F, defined as the probability that the two alleles at any given locus on an individual's chromosomes are identical by descent. If mating continues for further generations in like vein, as for an inbred line, the inbreeding coefficient rises progressively. F can lie anywhere between 0 (complete outbreeding) to 1 (complete inbreeding). In the latter case all individuals of the line are homozygous: $H = 0$. Much time is spent in well-run zoos, and rightly, compiling breeding registers allowing the calculation of pedigrees leading to estimation of inbreeding coefficients. Ballou (1983) describes the methodology. DNA fingerprinting can be used in the absence of a pedigree to give a rough index of relatedness (Brock & White 1992).

Both genetic drift and inbreeding reduce heterozygosity. For both the rate of loss accelerates with declining numbers. Beyond that their effects differ. The probability that genetic drift will fix a given allele is dependent upon its initial frequency and the size of the population. Inbreeding effects are more predictable and directional. 'The effect of inbreeding on a trait, in the presence of recessives, will be a shift in the average expression of the character towards the homozygous recessive phenotype' (Frankel & Soulé 1981, p. 63).

INBREEDING DEPRESSION

The exposure of recessives by inbreeding can decrease fitness if some of those recessives are deleterious. A disproportionate number of recessives at polymorphic loci are, in fact, deleterious for the simple reason that their expression is less often exposed to selection than those of the dominant alleles (Frankel & Soulé 1981). The mechanisms of inbreeding depression are poorly understood. Studies on *Drosophila* suggest that the homozygosity of deleterious recessives accounts for only about half of the observed loss of fitness (Hedrick 1992). The gene pools of most populations contain many of these sub-lethal recessives (the genetic load), about enough to kill an individual three times over (but see Ewens 1992) if by chance they all occur in homozygous form and are therefore all expressed in its phenotype. Thus, a decline in heterozygosity tends to lead to a decline in fitness.

The following sequence may be triggered if a population becomes too small.

1. The frequency of mating between close relatives rises.
2. Which leads to reduced heterozygosity in the offspring.
3. Which exposes the effect of semi-lethal recessive alleles.
4. Which reduces fecundity and increases mortality.
5. Which causes the population to become smaller yet, and that trend may continue to extinction.

This is the 'extinction vortex' produced by a positive feedback loop (the worse it gets the worse it gets) between the size of the population and the average fitness of its members. The population must be held at low numbers for several generations before the effect of that five-fold process manifests. A short bout of low numbers has little effect on heterozygosity. Loss of fitness during inbreeding can be traced largely to the process of fixation (i.e. reduction of alleles at a locus to one type) of deleterious recessive alleles.

Frankel & Soulé (1981, p. 65) point out that inbreeding depression shows up preferentially in the so-called fitness characters (e.g. fecundity, age at first breeding, juvenile mortality) because these are the traits that typically display dominance or overdominance. The traits that are least affected by

inbreeding are those that vary widely, but have little influence on or association with reproduction and viability.

Inbreeding depression is an ever-present worry with the typically small populations housed in zoos. It is a lesser problem in natural populations because mating between close relatives is uncommon and individuals often actively avoid mating with close relatives (Ralls, Harvey & Lyles 1986).

Ralls, Brugger & Ballou (1979) produced the first comprehensive account of inbreeding depression in captive populations of mammals. In an elegant later paper, Ralls, Ballou & Templeton (1988) estimated inbreeding depression of progeny according to the degree of relatedness of the parents. They used the theoretically predicted model

$$S = e^{-(A+BF)}$$

where S is the juvenile survival rate: the proportion of newborn surviving to (in this case) 180 days, or to half the age of sexual maturity for small mammals. A is the instantaneous rate of juvenile mortality for progeny of unrelated parents. It may be viewed as the control against which the effect of inbreeding is gauged. B is the additional instantaneous rate of mortality imparted to A when the line is completely inbred (i.e. $F = 1$ and $H = 0$) and F is the inbreeding coefficient. That equation is linearized as $\ln S = -A - BF$ which allows a regression estimate of A and B.

The cost of inbreeding i is estimated for a given F (standardized by Ralls *et al.* (1988) to an arbitrary $F = 0.25$) as

$$i = 1 - \left[\frac{\text{Survival rate when } F = 0.25 : e^{-(A+0.25B)}}{\text{Survival rate at } F = 0.0 : e^{-(A)}}\right]$$

$$= 1 - e^{-0.25B}.$$

Cost of inbreeding i is the proportionate decline in survival caused by inbreeding of a given magnitude. Thus, the relationship can be written also as $i = (S_0 - S_F)/S_0$, where S is the proportion of a cohort surviving at the end of the juvenile stage and the subscripts give the coefficients of inbreeding. Cost of inbreeding i is sometimes erroneously interpreted as the proportion by which mortality is higher in offspring of inbred matings than in offspring of matings between unrelated parents. That statistic is i multiplied by $S_0/(1 - S_0)$.

Ralls *et al.* (1988) gathered an impressive database of juvenile mortality for individuals of known pedigree in 38 species across seven mammalian orders. Consequently, they could estimate A, B and i for each species. A ranged between 0·03 and 1·1 with a mean of 0·33; B ranged between −0·68 and 15·16 with a mean of 2·33; and i, the inbreeding cost of mating between full sibs or between parent and offspring, lay between −0·19 and 0·98 with mean also of 0·33.

Close inbreeding usually leads to inbreeding depression, but not invariably (see Ralls *et al.* 1988, Fig. 3). Some species have such a low B that prodigious inbreeding is needed to generate a detectable effect. It is worth remembering that $F = 0.25$ is a huge coefficient of inbreeding for natural populations; even $F = 0.025$ would be cause for comment. Nor does low heterozygosity necessarily lead to inbreeding depression. Note that on average an individual bird or mammal is electrophoretically heterozygous at less than 5% of loci (Nevo 1978). A population that has weathered a bout of inbreeding may come out of it with fitness enhanced because inbreeding exposes deleterious recessives and allows them to be purged from the gene pool. That is precisely the method used by animal breeders to remove deleterious alleles.

METAPOPULATIONS

A metapopulation is a population of populations (Hanski & Gilpin 1991). Wright (1940) laid the groundwork for a genetic theory of metapopulations, while Andrewartha & Birch (1954, Ch.14) did the same for metapopulation dynamics: 'A natural population occupying any considerable area will be made up of a number of local populations or colonies. In different localities the trend may be going in different directions at the same time.' They emphasized the influence of dispersal on the number of patches occupied at any given time.

Huffaker (1958) studied experimentally the effect of metapopulation structure on the stability of predator–prey systems, using two species of mite, one a predator and the other a herbivore, inhabiting a universe of oranges spread out on a tray. A previous set of experiments of prey–predator interaction within a single patch (Gause 1934; Gause, Smaragdova & Witt 1936) led to the conclusion that such systems were self-annihilating except in unusual circumstances. Huffaker found Gause's conclusions held for the predator but not the prey within simple metapopulations. However, the prey and predator co-existed if the number of local populations were expanded and the spacing between them increased. Nachman (1991) experimented similarly with mites on cucumbers to show that demographic stochasticity performed an important role in counteracting the synchronizing effect of density-dependent dispersal that would otherwise generate cyclicity.

Andrewartha & Birch (1954) simply described the world as they saw it and did not present a mathematical model of the metapopulation processes they described. That task was left to Levins (1969, 1970). In the notation of Hanski (1991):

$$dp/dt = mp(1-p) - ep$$

where p (i.e. patch) is the number of patches of favourable habitat occupied by a local population at time t. The variable e (i.e. extinction) is the rate at which

those local populations die out and m (i.e. migration) is the rate of colonization of patches. His model presages an equilibrium p^*, at $p^* = 1 - e/m$. It is interesting to note that those equations are structural homologues of Wright's (1940) equations describing the change in the frequency of a non-dominant gene within a metapopulation.

Levins' model is actually a logistic (Hanski & Gilpin 1991) as can be seen by rewriting his equation as

$$\mathrm{d}p/\mathrm{d}t = (m-e)p[1-p/(1-e/m)]$$

$1-e/m$ being equivalent to carrying capacity K, and $m-e$ to intrinsic rate of increase r_m, of the logistic equation.

Metapopulation structure has many implications for conservation biology. It forms the conceptual framework for designing a reserve system and for managing populations whose habitat is fragmented. It has marked effects on the genetics of the constituent local populations (typically a reduction in heterozygosity) and on their dynamics (the ability of the metapopulation to 'rescue' declining local populations by dispersal from larger local populations).

These days a zoo system acts as a metapopulation for some species, particularly those endangered in the wild. By maintaining a register of pedigrees, and by judicious interchange of individuals or semen between zoos, it is possible to beat the constraints of effective population size (genetic) and minimum viable population (genetic). Stanley Price (1989) gives a good example from captive breeding of the endangered Arabian oryx *Oryx leucoryx*.

MINIMUM VIABLE POPULATION, MVP

The notion of a size below which a population is at imminent risk of extinction was discussed by, amongst others, Soulé (1980), Shaffer (1981, 1987), Gilpin & Soulé (1986), and Lande & Barrowclough (1987). Soulé (1987b) and later elaborations (Soulé & Kohm 1989; p. 38, Soulé & Mills 1992) give a brief, but useful review of its history. It is critically important to planning reserves, and has wide application to small and fragmented populations outside a reserve system. It must be kept continually in mind for populations in zoos.

Minimum viable population size has two aspects: genetics and population dynamics. Franklin (1980) offers two estimates for the size of populations needed to conserve genetic variance (the much quoted 50/500 rules). He pointed out that animal production studies indicate that inbreeding is kept to a tolerable level with a population of 50 individuals and that this level was probably high enough to stave off inbreeding depression.

He suggested 500 as a lower limit for allowing free rein to evolutionary processes. It should be noted first that this is an effective population size (genetic) rather than the actual size of the population, which would have to be three or four times larger to score $N_e = 500$. Secondly, it is not a unique size at which loss of genetic variance by drift is balanced by mutational input. That equation balances at any population size, as indicated earlier under the heading 'Genetic drift'. What varies is the amount of additive genetic variance at equilibrium with population size. Franklin's 500 relates instead to the amount of genetic variance one might wish to retain. He chose as his standard an amount of genetic variance equal to the amount of environmental variance expressed in the phenotype of a totally homozygous population. He calculated from information on the genetics of bristle number in *Drosophila* reviewed by Lande (1976) that this variance would be retained by an effective population size (genetic) of 500 (see Lande & Barrowclough 1987 for a clear exposition). Note that the extrapolation of this number to other species carries three implicit assumptions: that the targeted amount of genetic variance is appropriate, that it is independent of H, and that the number of bristles on a fruit fly serves as a surrogate for other traits in other taxa.

The 50/500 rules are purely genetic concepts which have nothing to do with the size of a caribou population sufficient to cope with freezing rain in two successive winters. As Soulé (1987b, p. 5) emphasized, there can be no single rule of thumb nor magic number for the safe minimum size of populations subject to varying influences on their dynamics. First, any estimate is specific to its envelope of prediction: probability of persistence and time interval. Both must be specified. Secondly, populations are subject to differing levels of environmental stochasticity and have differing behavioural and demographic adaptations for coping with it. If generalization is possible it must await the accumulation of case studies and experimental manipulations of population size (Boyce 1992).

EFFECTIVE POPULATION SIZE, N_e

The previous sections on genetic drift and inbreeding were written as if all individuals in the population were equivalent. Thus, the N of the drift equations made no distinction between breeders and juveniles. In fact, the proportion of genetic variance lost by random genetic drift may be higher than the computed theoretical $1/(2N)$ per generation because that formulation is correct only for an 'ideal population'. In this sense 'ideal' means that family size is distributed as a Poisson variate, sex ratio is 50:50, generations do not overlap, mating is strictly at random and the population is stable in size. This introduces the notion of effective population size in the genetic sense, the size of an ideal population that loses genetic variance at the same rate as does the real population. The population's effective size (genetic) will be less than its census size (Wright 1938), except in special and

unusual circumstances, typically by a factor of 2–4 in birds and mammals and by rather more in fish and invertebrates. As a first approximation, effective population size is equal to or less than the number of breeding individuals.

Genetic drift is minimized when sex ratio is 50:50. Effective population size (genetic) in terms of sex ratio is given by

$$N_e = 4N_mN_f/(N_m+N_f).$$

(Wright 1940), where N_m and N_f are the numbers of males and females. The reason lies in the relatedness of the offspring. Using the example of Frankel & Soulé (1981, p. 38), consider a population of zebra comprising one male and nine females. The offspring would necessarily be related to each other as either half-sibs or full sibs. However, if the population were five males and five females the offspring would, on average, be less closely related and, hence, would be less likely to lose an allele during transfer of genetic material from parents to progeny. The effective population size (genetic) is controlled by the number of the less common sex. For the example of one male and nine females, $N_e = 1\cdot6$. With one male and an indefinitely large number of females $N_e = 4N_m = 4$ (Wright 1940).

A second cause of the disparity between census size and effective size is the differences among individuals in the number of offspring they contribute to the next generation. In the ideal population their contribution has a Poisson distribution, the fundamental property of which is that variance equals mean. Should the variance of offspring production among individuals exceed the mean number of offspring produced per individual the effective population size will be smaller than the census size. In the unlikely event of variance being less than the mean the effective population size is greater than the census size and the population is coping better genetically than one might naively have expected. The effective population size N_e corrected for this demographic character was first provided by Wright (1940). It is here given in the form suggested by Lande & Barrowclough (1987):

$$N_e = (NF-1)/[F+(s^2/F)-1]$$

where F is the mean lifetime production of offspring per individual and s^2 is the variance of production among individuals. It indicates that when mean and variance are equal $N_e = N$ (within the tolerance of the small-sample correction) for this component of effective population size. Since males and females sometimes differ in mean and variance of offspring production that equation is often solved for each sex separately and the sex-specific N_e values summed.

Similarly, the effective population size (genetic) of a fluctuating population is not the arithmetic mean but close to the harmonic mean (Wright 1940):

$$N_e \approx n/\Sigma(1/N_i)$$

where there are n individual censuses each yielding an independent estimate N_i, because the overall leakage of genetic variance is controlled disproportionately more by smaller than by larger numbers. Lande & Barrowclough (1987) give the exact formulation.

Then there are the additional effects of overlapping generations, dispersal and dispersion on effective population size. These, and those discussed above can with courage be linked one to another to finally produce an N_e that describes the genetically effective size of the population. Reed, Doerr & Walters (1986), Chepko-Sade et al. (1987) and Harris & Allendorf (1989) provided useful discussions and several examples of this methodology.

To round out this section it is necessary to consider the other way in which the structure of a population influences the chance that it will die out: the effects of unbalanced age distribution and sex ratio on its rate of increase. Derivations will be given elsewhere. As far as I can determine, this aspect seems not to have been considered overtly within the literature of the small-population paradigm although it is tacit, for example, in Goodman (1980). An effective population size (demographic) may be defined by analogy with an effective population size (genetic) as the size of an 'ideal' population with an even sex ratio and a stable age distribution that has the same net change in numbers over a year as the population of interest. It will be symbolized N_{ed}, and to avoid confusion its genetic analogue will be N_{eg} from here on. Effective population size (demographic) may be calculated as the observed population size N multiplied by the ratio of its net annual increment ΔN to the net annual increment ΔN_I of an ideal population of the same size. Thus, correcting for the sex ratio of a birth-pulse population

$$N_{ed} = N(P_fpb+p-1)/(\tfrac{1}{2}pb+p-1)$$

in which, at the beginning of the year starting immediately after the annual birth pulse, a population of size N contains a proportion P_f of females; p is the probability of surviving the subsequent year averaged over individuals of all ages, and b is number of live births produced per female at the birth pulse terminating that year.

A disparate sex ratio may have a significant effect on a population's ability to increase from low numbers, enhancing that ability when females predominate and depressing it when males dominate. Mammalian populations that crash because they eat out their food, or because a drought cut it from under them, often end the population slide with a preponderance of females. They are thus in better shape demographically to recover from the decline than if parity of sex ratio were retained.

Variation in age distribution has a parallel effect on rate of increase. We will follow the demographic convention of considering only the female segment of the population and assume that males do much the

same thing. Analogous equations can be written for the male segment, but in practice they are not used because of the difficulty of determining paternity for the calculation of male fecundity. Thus, N is now only the females and N_x is the number of females aged x at the beginning of the year. The age-specific variables p_x and m_x are, respectively, survival rate of females that started the year at age x and their average production of female live births at the end of the year. The age distribution is symbolized f_x and scaled such that $f_x = 1$ for the newborn age class. For a population with a stable age distribution (i.e. the 'ideal' population) $f_x = l_x e^{-rx}$ to an acceptable level of approximation, where l_x is survivorship at age x and r is the exponential rate of increase. Thus, effective population size (demographic), in terms of the age distribution, is

$$N_{ed} = N[\Sigma f_x(p_x m_x + p_x - 1)]/[\Sigma l_x e^{-rx}(p_x m_x + p_x - 1)].$$

If f_x exceeds $l_x e^{-rx}$ in age classes where m_x is above average (and, consequently, it will be lower than $l_x e^{-rx}$ at the less fecund ages) then ΔN will be higher than that produced by the stable age distribution, and N_{ed} will thus be higher than N. The population is acting demographically as if it were larger. Where more of the animals are loaded into the less fecund age classes the converse applies: ΔN and hence N_{ed} are lower than expected.

The point to note is that N_{eg} and N_{ed} tell different stories. The context is critical if one or other is applied because what is good for a population's genetics may be bad for its demography. The population structure minimizing genetic drift may hinder the population's ability to recover from low numbers.

Population viability analysis, PVA

Population viability analysis returns an estimate of the expected time to extinction of a population with given characteristics, or alternatively the chance of its dying out over a specified interval. It has been reviewed recently by Boyce (1992). Here I simply profile the subject.

Richter-Dyn & Goel (1972) manipulated the mathematics of population growth according to the stochastic birth–death process to provide an explicit estimate T of mean time to extinction. It allowed for birth rate and death rate varying as a function of population size, which itself had a reflecting ceiling K. Goodman (1987) modified this persistence model to allow for environmental variance in r. For a population initially at carrying capacity K, Goodman's equation takes the form

$$T = \sum_{x=1}^{K} \sum_{y=x}^{K} \frac{2}{y[yV_{(y)} - r_{(z)}]} \prod_{z=x}^{y-1} \frac{V_{(z)}z + r_{(z)}}{V_{(z)}z - r_{(z)}}$$

where $r_{(z)}$ is the mean instantaneous rate of increase when the population comprises z individuals, and $V_{(z)}$

is the variance in r (both demographic and environmental) at that population size.

Estimating V for each population size is virtually impossible, estimating size-specific r is very difficult, but one has a fighting chance of estimating an approximate value of the average growth rate. Furthermore, if one assumes that V is only the environmental variation in r, demographic variance being inconsequential, V can legitimately be declared constant. That is what Belovsky (1987) did, simplifying Goodman's equation by dropping out the z subscripts:

$$T = \sum_{x=1}^{K} \sum_{y=x}^{K} \frac{2}{y[yV_e - r]} \prod_{z=x}^{y-1} \frac{V_e z + r}{V_e z - r}.$$

He used this equation to test Goodman's model against Brown's (1971) and Patterson's (1984) information on the persistence of populations of small mammals stranded on the top of mountains (of which more later).

Lande (in press) presented a further simplification in which it is assumed that the population's rate of increase is constant except at K where it is zero. By the approximation provided by the diffusion model

$$T = \frac{2}{V_e c} \left(\frac{K^c - 1}{c} - \ln K \right)$$

where $c = 2r/V_e - 1$. Many more estimators of persistence time are to be found in the literature, but the above three sufficiently sketch the field.

Simulation modelling is an alternative way of gauging a population's viability. The most widely used computer program is VORTEX, which by April 1992 had evolved into Version 5.1. It is a taut piece of programming designed for IBM-compatible desktop computers. I have inferred its substance from the manual (Lacy & Kreeger 1992), from a recently published description of the program (Lacy 1993), from the 'readme' file of version 5.1, and by running it.

The user describes to the program the characteristics of the population of interest and the program replies with the probability of extinction over intervals of time. It assumes that rates of fecundity and mortality are independent of age after first breeding, it accepts mortality rates for each age class before that, and it is interested in the mating system of the species. The user specifies the proportion of females breeding and the proportion of breeders that produce litters of $1, 2, \ldots, 5$. The program insists upon receiving an estimate of carrying capacity K. Genetics (optional) are considered as an infinite-allele model of genetic variation, each founder having two unique alleles. The user may tell the program how much inbreeding depression to expect from loss of heterozygosity. A linear decline in available habitat (treated as a falling K) may be specified where appropriate. Reduction in numbers by harvesting, or supplementation of numbers as from a captive breeding program, are allowable options. Metapopulation structure is catered for.

Environmental variation may be imparted optionally to the carrying capacity and to rates of mortality and fecundity. 'Catastrophes' of specified frequency and intensity may be invoked to act on the specified rates of fecundity and mortality. Fecundity (but not mortality) may be rendered density-dependent by ascribing values to the constants of a fourth-order polynomial relating fecundity to population size.

The engine of this program is a Monte Carlo simulation algorithm. The probability of an individual dying or reproducing is a random draw from a binomial distribution with mean p (entered by the user) and variance $p(1-p)/N$, where N is the size of the simulated population at each computed step. Environmental variance in K is normally distributed. At default values these characteristics adds up to a stochastic model, partially age-structured, of exponential increase truncated at a reflecting boundary specified by the user. The program is run many times, each run providing a different population trajectory because a computer's generator of random numbers seldom repeats itself. The probability of extinction after a given number of computational steps is estimated as the complement of the proportion of those runs still nurturing an extant population.

This program delivers what its documentation promises with exemplary computational efficiency. The quite separate question—to what extent does it assist in the task of saving a species at risk—is addressed further on under 'Persistence'.

CAPTIVE BREEDING

Captive breeding might seem to be outside the general scope of conservation biology, or at the most peripheral to it, but it is in fact one of the icons of the small-population paradigm. It is seen as one of the most powerful tools available for rescuing a species that has declined to very low density or which is threatened for other reasons. The first book on conservation biology edited by Soulé (Soulé & Wilcox 1980) devoted four chapters to it.

There are few examples of captive breeding or translocation being used to re-establish a species in the wild. Probably the best is the rescue of the southern white rhino Ceratotherium simum (Burchell). By the turn of the century it had declined to about 10 animals. Strict protection in the Umfolozi Game Reserve had it increasing to 120 individuals by 1930 and it has subsequently been translocated to many parts of southern Africa with surplus animals being sent to zoos and wildlife parks throughout the world (Smithers 1983). It is now secure.

Stanley Price (1989) laid down the principles of successful re-introduction. There must be a feasibility study to determine what agents reduced the original population and whether they are still operating, whether suitable habitat remains, and whether funds are available to do the job properly. Secondly, there

should be a preparation phase identifying a suitable site for liberation, a suitable stock and an appropriate strategy. Thirdly, the actual release must be planned carefully to coincide with the optimum season and time of day. And fourthly, the re-introduced population must be monitored for several years.

Zoo populations share all the characteristics defining the small-population paradigm. Anyone working with them must to be conversant with such things as demographic stochasticity, minimum viable population, genetic drift, effective population size and inbreeding depression (Seal 1985). Not surprisingly, there is a nexus of interest between those people working with captive populations and those contributing to the theory of genetics of small populations.

DECISION ANALYSIS

Decision analysis makes explicit the logic by which a decision is reach under conditions of uncertainty. Its methods are outlined by Keeney & Raiffa (1976), and by Nisbett & Ross (1980). There are two distinct steps in a decision analysis. The first is critical. The researcher estimates the probabilities of various events occurring over a given time interval and also the probabilities of each of the several potential consequences of those events. This 'risk assessment' had considerable currency during the 1970s [see in particular the review of Speed (1985) in the context of predicting malfunction of nuclear reactors, an outcome of similar seriousness to loss of species]. In the second step those probabilities are analysed according to the multiplication and addition rules of probability theory to adjudicate between options. Only that second step is rightly termed decision analysis. It requires no additional information and is essentially a mechanical application of the rules of probability. The real work is done at the risk assessment stage.

The aptness of a recommendation provided by a decision analysis is evaluated by examining the risk assessment upon which it operates. The following list is an elaboration of Speed's (1985).

1. Is each probability estimated from the frequency of observed occurrences?
2. Are the probabilities taken from previous similar cases?
3. Are most of the component probabilities estimated but a few supplied by subjective judgment?
4. Are all the probabilities supplied by subjective judgment?
5. Is the list of possible events complete and are those events independent of each other?
6. Is the list of potential consequences complete for each event?

Maguire (1986) advocated decision analysis for deciding between different options for managing an endangered species. I have drawn on that helpful account. The methodology has the virtue of simplicity.

Suppose that over a specified interval a population faces a probability p(storm) $= 0.2$ of being hit by a devastating storm that will reduce its size substantially, perhaps to a size from which it cannot recover. The probability of extinction by such an event is labelled pE(storm). The expected probability E(pE) of its dying out over the specified interval is then

$$E(pE) = [p(\text{storm}) \times pE(\text{storm})]$$
$$+ [p(\text{no storm}) \times pE(\text{no storm})].$$

Having established the frequency of such storms to allow an estimate of p(storm), the probability of no storm is filled in mechanically as p(no storm) $= 1 - p$(storm), but the remaining two variables requires independent information. Let us say that we have such information, that it is a certainty that the storm will wipe out the population— pE(storm) $= 1$—and that the chances of its dropping out due to other influences if there is no storm is pE(no storm) $= 0.08$. The probability of extinction under all specified (if restricted) circumstances can now be estimated as

$$E(pE) = (0.2 \times 1) + (0.8 \times 0.08) = 0.26.$$

In arriving at 0.26 as the probability of extinction over a specified time interval we needed three independent items of information: those leading to the estimates of 0.2, 1 and 0.08.

That thought experiment can be extended to any number of influences on the survival of a population. Maguire (1986) demonstrated the utility of this approach for exploring the problem of whether an endangered species would be more secure if managed as one large population or as two smaller populations, a variant of the SLOSS problem discussed later in the context of designing a reserve system. Table 3 is a mild modification of Maguire's Fig. 1. The probabilities of the first option are those given above for a single large population. It is assumed that the two smaller populations would be established in the same climatic

Table 3. Decision tree for managing a species as one large versus two small populations, where the random events are catastrophic storms that reduce populations below the point of recovery. Modified from Maguire (1986). An event has a probability p of occurring over a specified interval, there is a probability pE that the population will go extinct if that event occurs, and the expected probability of extinction over the time interval is E(pE)

Option	p Event	pE	E(p)E
Single large population	0.2 storm	1.0	0.26
	0.8 no storm	0.08	
Two smaller populations	0.04 storm in both	1.0	0.19
	0.16 storm in Pop 1	0.3	
	0.16 storm in Pop 2	0.3	
	0.64 storm in neither	0.09	

zone as that of the single large population, but that they would not be so close together that the same storm could take out both. The event probabilities of the second option are related to those of the first as $0.04 = (0.2)(0.2)$, $0.16 = 0.2 - 0.4$ and $0.64 = 1 - (0.04 + 0.16 + 0.16)$. The values of pE $= 0.3$ represent the probability that the species would extinguish if reduced to a single small population and it requires information independent of the other probabilities. The probability of extinction, given that neither small population is hurricaned, is $0.09 = (0.3)(0.3)$. Thus, by the logic outlined for the single large population, the expected probability of extinction of the species managed as two small populations is

$$E(pE) = (0.04 \times 1) + (0.16 \times 0.3) + (0.16 \times 0.3)$$
$$+ (0.64 \times 0.09) = 0.19$$

as against the E(pE) of 0.26 for one large population. On that basis we might concluded that the two smaller populations provide the safer option in this particular imaginary example.

This methodology is effective. The only weakness that needs a mention here is its penchant to perform rather like an analogue computer: a twiddling of the knobs (a shift of the risk assessment probabilities) provides almost any result one might wish. Note that the example, simplistic as it is, required four independent data, one giving the frequency of storms and three the probabilities of extinction under specified circumstances. If the input probabilities are faulty or if the dominant agents of risk have been deduced erroneously, the decision will inevitably be wrong.

DESIGN OF RESERVES

Much of the theory of reserve design takes its inspiration from island biogeography, particularly from the early formative publications of MacArthur & Wilson (1963, 1967). It linked the species-area relationship with the dynamic determinants of an equilibrium number of species. The species-area curve had been around for a long time (see May 1975 for a review of species–area relationships)—a ten-fold increase in area is associated with a doubling of species—but it was usually envisaged at a scale of metres rather than kilometres. Islands and national parks are of similar scale and dwarf standard vegetation quadrats. The idea that the equilibrium number of species on an island is a function of migration rate and extinction rate, which themselves vary according to distance from a source of immigrants and the size of the island, is so intuitively appealing that more than theoretical ecologists paid attention. These powerful ideas were easily transformed mentally to 'island' reserves of habitat in a sea of agriculture. Many park planners and managers took the point. Diamond (1975, 1976), Whitcomb *et al.* (1976), Simberloff & Abele (1976) and Margules, Higgs & Rafe (1982) explored and

debated the relevance of island biogeography theory to designing a reserve system.

Reserves share with zoos the characteristics of the small-population paradigm. The populations preserved therein are usually in no immediate danger, but being often small and always capped in numbers, they are potential prey over the mid-term and longer to the deleterious genetic and demographic hazards outlined earlier. These concerns lead to a tight set of questions.

1. How big must a reserve be to retain for a given number of years a given proportion of the species that originally lived there (Diamond 1975)?

2. Will a single large reserve preserve more species over time than several small reserves? This one often goes under the delightful acronym SLOSS (Single Large Or Several Small). Discussion and debate are provided by Järvinen (1982), Lahti & Ranta (1985) and Robinson & Quinn (1992). This question is easily confused with another that is a superficially similar, but trivial: will a single large reserve, or two smaller reserves summing to the same area as the larger, hold the more species?

3. Should a reserve be long and thin to maximize environmental heterogeneity or should it be compact to maximize the ratio of area to circumference (Diamond 1975; Wilson & Willis 1975)?

4. Should reserves ideally be linked by corridors of habitat to facilitate gene flow between them and to encourage metapopulation dynamics whereby a reserve hosting a local extinction may be recolonized from another reserve? There are theoretical and empirical arguments for and against (Wilson & Willis 1975; Fahrig & Merriam 1985; Simberloff & Cox 1987; Noss 1987; Nicholls & Margules 1991; Harrison 1992).

A related but different question addresses the problem of where reserves should be, and how many there should be, to capture either a maximum number of species or a given proportion of the species resident in a region (Pressey *et al.* 1993). Two competing approaches have emerged, a methodology based on first selecting sites rich in species and then filling gaps in coverage (Kirkpatrick 1983; Kirkpatrick & Harwood 1983; Scott *et al.* 1988) and the second based upon first selecting sites containing rare species (Margules & Nicholls 1987; Margules, Nicholls & Pressey 1988; Pressey & Nicholls 1989b, 1991; Pressey, Bedward & Nicholls 1990; Rebelo & Siegried 1992) and then filling gaps. A limited comparison of these two approaches suggested that they tend to converge upon much the same estimate of required number of reserves and total area of reserves (Pressey & Nicholls 1989a). Two benefits flow from the use of these algorithms. First, they lead to an explicit decision from which any variation is open to inspection. Secondly, they invoke a designed field survey to discover what species are where, museum specimens (usually taken from a road and usually yielding out-of-date infor-

mation) providing data on distribution inadequate for these purposes. These methods target places rather than species as the units of reservation, enabling the simultaneous consideration of the many species constituting a biota.

The declining-population paradigm: theory

The dominant tenet of the declining-population paradigm is distilled easily enough: the contraction of the range of a species and the decline in the number of its members has a tangible cause which with skill may be identified and defeated. There is an agent of decline: small population size is not of itself a cause.

One of the more distressing characteristics of the declining-population paradigm is its dearth of theoretical underpinning. The reason is intrinsic in part because agents of decline are multitudinous and their effects not easily generalized, but it reflects also a general reluctance or inability to think beyond the immediate problem. The taxonomy of the species of concern is tacitly considered a more important trait than the process under investigation. Such studies are easily identified by a glance at the list of references at the end of the paper. There are, however, two areas of theory to which the declining-population paradigm can lay claim. The first covers causes of extinction, the second the means by which agents of decline may be identified.

THE EVIL QUARTET

Diamond (1984a, 1989) investigated recent extinctions and found that their agents of decline, where known, could be classed under four headings:

1. Overkill.
2. Habitat destruction and fragmentation.
3. Impact of introduced species.
4. Chains of extinction.

Diamond called these 'the evil quartet'.

Overkill

Overkill results from hunting at a rate above the maximum sustained yield. The most susceptible species are those with low intrinsic rates of increase (i.e. large mammals such as whales, elephants and rhinos) because these have little bounce-back built into their dynamics. Hence, although usually having a high standing biomass when unharvested they have a low maximum sustained yield which is easily exceeded. They are rendered even more vulnerable if valued either as food or as an easily marketable commodity.

In the latter case they clash head-on with an economic imperative known as the discount rate. It is the factor by which future earnings must be discounted to estimate their present value. The effect of this calculation on deciding for purely business reasons

whether to go for a sustained yield, or whether to take the whole population as quickly as possible and re-invest the earnings in an enterprise paying a higher rate of return, is outlined at book length by Clark (1976). May (1976) identified it as the reason why the whaling industry never tried for a sustained yield. We may presently be watching the same economics coming into play against rhino and elephants. Contrary to expectation this mechanism operates most forcefully in a fully informed and free market, particularly if the stock of animals or plants is owned privately.

Also at risk from overkill are those species inhabiting islands whose area (as viewed with a hunter's eye) is finite; particularly if the luck of the draw in the sweep-stake lottery of over-water dispersal has excluded co-evolved predators. The species is then likely to be both accessible and tame. Olson (1989) drove that point home with a distressing surfeit of examples.

To summarize, a species face-to-face with a hunting culture is in grave danger if valuable, insular or big.

Habitat destruction and fragmentation

Habitat may be degraded by such agents as a change of fire regime or grazing by sheep and goats. Sometimes the habitat is eliminated as in the draining of a wetland or the cutting down of a patch of forest.

More commonly the habitat becomes fragmented, a large tract being converted piecemeal into another land-use. The process may be viewed almost anywhere in the world by inspecting aerial photographs taken several years apart. Graetz, Fisher & Wilson (1992) provided vivid examples from satellite imagery compared between 1972 and 1992. Loss of habitat by a given proportion does not increase the vulnerability of a species, nor decrease the number of its members, by that same proportion, except in the special case of habitat being cleared from the edge inward. More commonly the modification acts to produce a patch-work pattern as it erodes the tract of habitat from inside and changes microclimates (Saunders, Hobbs & Margules 1991). From the air it looks like a thin layer of colloidal suspension. At first the areas occupied by the new land-use form islands—the discontinuous phase of a colloidal suspension. At this stage there may well be both an increase in the number of species using the total area, because the additional habitat attracts new species, and an increase in the numbers of those resident species that benefit from the increased length of edge. However, that period is usually short-lived. As the process gains momentum those desert islands multiply and enlarge. Hobbs *et al.* (1992, Fig. 4.1) provide a graphic four-stage example. Quite suddenly the system flips, the new land-use providing the continuous phase and the original habitat the discontinuous phase. The vulnerability of species then increases quantally.

Some information is available on the effect of fragmentation upon rate of local extinction in tropical forest (Willis 1980; Lovejoy *et al.* 1984, 1986; Diamond, Bishop & van Balen 1987), semi-arid woodland (Hobbs *et al.* 1992) and temperate forest (Diamond 1984b), but very little from other ecosystems. There is, however, a considerable literature on fragmentation caused by rising post-Pleistocene temperature that constricted temperate habitats to the tops of mountains (e.g. Brown 1971; Patterson 1984), and rising sea levels that marooned species on continental islands (e.g. Hope 1973; Wilcox 1978; Diamond 1983). Many such studies have provided astonishingly clean and lavish data, in some cases including fossil evidence of which species were originally on the islands (e.g. Hope 1973) and in one case a detailed chronology (Wilcox 1978). Diamond's (1984a) pivotal paper provided an analysis of these and other fragmentation studies to show that of the various factors influencing rate of local extinction, population size was clearly dominant.

Impact of introduced species

Diamond's third agent of decline is the alien species introduced intentionally or unintentionally by people and which proceeds to exterminate native species by competing with them, preying upon them, or destroying their habitat. Atkinson (1989) provided an excellent review, well documented. Table 4 indicates the extent of the problem.

Chains of extinction

In this category are the secondary extinctions, the extinction of one species caused by the extinction of another upon which it depends. Diamond (1989) gave as an example the extinction or near extinction of endemic Hawaiian plants of the genus *Hibiscadelphus* as a consequence of the extinction of several species of their pollinators, the Hawaiian honey creepers. There are numerous examples of predators and scavengers dying out after the species providing their food died out.

MINIMUM VIABLE POPULATION AND POPULATION VIABILITY ANALYSIS

Minimum viable population size as viewed from within the declining-population paradigm is a single pregnant female. Since that is a trite answer the question is seldom put. The notion has scant relevance because management actions are concentrated on increasing the size of a small population as quickly as possible. Likewise a small population whose size is capped is something of an oxymoron within the declining-population paradigm.

Population viability analysis (PVA) provides an estimate of how long a population will persist if

Table 4. Causes of extinction in three groups of vertebrates (after Atkinson 1989)

Vertebrate group	Place	Number of species/subspecies	Causes Alien animals	Other causes	Unknown
Reptile and amphibian extinctions since AD 1600	World	30 spp.	22	3	5
Reptile and amphibian extinctions since AD 1000	New Zealand	14 spp.	9	4	1
Bird extinctions or near extinctions since AD 1840	New Zealand	31 spp. and sub-species	23	2	6

nothing external to it changes. Recent elaborations of this methodology have moved in the direction of the declining-population paradigm by factoring in a contracting habitat but ignoring all other influences upon a population's rate of increase. These tend to provide the unremarkable but erroneous insight that a reduction in habitat by 20% reduces the capped size of the population by 20%. Boosting standard PVA with a factor only for contracting habitat (PHVA), in recognition that the population is in decline, does not convert it to an investigative instrument.

GENETICS

The small-population paradigm, dealing with the special problems faced by a small capped population, emphasizes the possible genetic problems that might arise therefrom. The declining-population paradigm sees the smallness of a population as a pathological condition that must be treated to get the population out of that danger zone as fast as possible. Thus, the genetics problems that might be faced by a population held at small size for several generations are not embraced by the declining-population paradigm because that state is precisely what management actions are designed to avert.

SCIENTIFIC METHOD

The essence of the declining-population paradigm is that the answer is not known. However, the question is defined precisely—why is this species declining and what might be done about it?—and we have had more than 400 years of advice on how such questions might be answered expeditiously. The quickest and surest way to identifying an agent of decline is to proceed down the hypothetico-deductive path. The problem is then within reach of the phalanx of powerful analytical methods typified by the Analysis of Variance. The steps go rather like this:

1. Study the natural history of the species to gain a knowledge of, and feel for, its ecology, context and status.
2. When confident that this background knowledge

is adequate to avoid silly mistakes, list all conceivable agents of decline.
3. Measure their levels where the species now is and also where the species used to be. Test one set against the other. Any contrast in the right direction identifies a putative agent of decline.
4. Test the hypotheses so produced by experiment to confirm that the putative agent is causally linked to the decline, not simply associated with it.

Agents can be tested singly or in groups, and sequentially or concurrently, according to the complexity of the system and the design. The most important steps are 1 and 4. Because the effect of putative agents are often confounded, step 4 is critical. However, without step 1 the wrong hypotheses may be selected. These prescriptions are amply validated by the results of research and management directed at the endangered red-cockaded woodpecker *Picoides borealis* (Walters 1991).

The influence of habitat upon the decline of a species is particularly tricky to diagnose. There are examples of the researchers assuming that the endangered remnants of a once widespread species have settled upon the most favourable habitat left and that their present lifestyles and feeding habits represent normality. The history of the New Zealand takahe rail *Notornis mantelli* (Caughley 1989), of the Hawaiian goose *Branta sanvicensis* (Kear & Berger 1980) and of the Lord Howe woodhen *Tricholimnas sylvestris* (Miller & Mullette 1985), informs us that the assumption is dubious and dangerous. A safer preliminary hypothesis would conjecture that the species ends up, not in the habitat most favourable to it, but in the habitat least favourable to the agent of decline.

Use of the small-population paradigm on wild populations

The management actions or tools of trade needed to avert extinctions, as seen from within the small-population paradigm, were outlined by Maguire, Seal & Brussard (1987), presented here as Table 5. They are described as 'intensive management strategies for endangered animal populations, both wild and cap-

Table 5. Examples of management interventions for critically endangered animal species as suggested by Maguire *et al.* (1987)

Wild populations and habitat only
- Translocating individuals or genetic material
- Raising carrying capacity (e.g. artificial feeding)
- Restricting dispersal (e.g. fencing)
- Fostering and cross-fostering young
- Reducing mortality (e.g. vaccination, parasite, predator, poaching control)
- Culling
- Preserving habitat
- Restoring habitat

Captive populations only
- Maintaining captive breeding populations for reintroduction and/or perpetual captivity
- Genetic and demographic management
- Maintaining gametes or embryos in 'miniature zoos' (i.e. freezers)

Captive and wild populations
- Re-introduction of captive-reared individuals or genetic material to occupied or unoccupied habitat
- Continued capture of wild individuals or genetic material for captive propagation

tive' and clearly the authors had in mind the kind of problems faced by small capped populations in nature reserves or zoos.

Woodruff (1989) offers another list of management actions here reproduced in part as Table 6. It has the same flavour as the previous set, again emphasizing genetic considerations. Whereas these can be and are tightly prescribed, given that the population is small and its members amenable to hands-on control, those management actions aimed at minimizing ecological problems faced by more dispersed populations are simply listed without elaboration. Neither his table nor text explain how to 'manage interacting species including: pollinators, prey species, predators, parasites, competitors' nor what 'manage' might mean in this context.

Thorne & Oakleaf (1991, Table 1) give similar advice, this being a six-step prescription entitled 'Steps in an effective endangered species management programme.'

Each of these three tables prescribe treatment, but none mentions diagnosis or cause. That may be

because the latter was seen as a separate issue outside the authors' brief or it may reflect the problem being perceived as low numbers as such and, hence, too obvious to mention.

There are, in fact, few examples of the tools of trade of the small-population paradigm being used to solve problems faced by wild populations. That may reflects only their newness. The rest of this section provides a commentary on most of the presently available examples.

PERSISTENCE

This heading subsumes the topics of minimum viable population, population viability analysis and to some extent effective population size, all of which are closely linked conceptually.

Minimum viable population

I can find no example of the idea of minimum viable population size being applied, as against talked about,

Table 6. Management practices identified by Woodruff (1989) as 'the recent application of genetic and ecological theory to the management of threatened populations and species, the results of conservation biology's first decade'

Maximize effective population size (N_e)
Minimize the variance in population growth rate (r)
Attain viable population size as soon as possible
Equalize the genetic contribution of the founders
Monitor and maintain inherent qualitative and quantitative genetic variability
Reduce inbreeding or purge populations of genes responsible for inbreeding depression
Avoid outbreeding depression
Maintain multiple populations (metapopulations)
Avoid selecting for 'type' or for domestication
Facilitate natural behaviour patterns including:
   dispersal and migration
   social and breeding
Manage interacting species including:
   pollinators
   prey species
   predators
   parasites
   competitors

in conserving a species in the wild. That might be just as well because it is a slippery notion, some people thinking of it in terms of the genetics of a population in a stable environment and others considering it in the context of population dynamics in a fluctuating environment.

Effective population size

Effective population size as presently used within the small-population paradigm is relevant to the management of only very small populations or those that remain moderately small for a considerable time. It is conceived largely in terms of genetic drift and founder effects, but it is equally apposite, and probably much more important in the wild, to the demographic behaviour of populations. However, the genetic and demographic effects do not go hand-in-hand. Effects that boost the effective population size (genetic) may erode the effective population size (demographic) and, thereby, slow or stop the population's break-out from low numbers. Effective population size (genetic) is most relevant to managing populations in zoos where maintaining a high effective population size (demographic) has little utility. Both the genetic and demographic versions are relevant to re-establishing a wild population by releasing a small nucleus of animals bred in captivity. The genetic version, for example, dictated the composition of the two founding groups of oryx released in Oman (Stanley Price 1989).

Population viability analysis

The expected staying power of a population can be expressed either as a mean persistence time or as the probability of its persistence for a given number of years. The second has proved more useful because it invokes a test of theory against numerous data on how long populations have persisted in fact. The question can then be transformed, with those findings in tow, to ask whether a reserve of a particular size (implying a population of a particular size) will conserve for the foreseeable future what it was set up to conserve.

Belovsky (1987) took Goodman's (1987) simple model of the effect of environmental stochasticity on persistence time and applied it to Brown's (1971) and Patterson's (1984) data on persistence of species stranded by post-Pleistocene warming on mountain tops in the American south-west. The date of stranding can be set with tolerable confidence at about 10 000 years ago, give or take one or two thousand. Belovsky's testing required several assumptions and approximations about the species and their environment to generate the data necessary to run the model. He had to substitute estimates of r_m derived from a regression restricted to herbivores (Caughley & Krebs

1983) for Goodman's 'average per capita growth rate' (which is not the same thing). Then he estimated density and, hence, population size (area being known) from Peters & Raelson's (1984) rough and ready regressions of carrying-capacity density on body size. Furthermore, variance in r was assumed to be related in simple fashion to variance in rainfall when, in fact, the relationship is complex. Even with these approximations the theory and the facts matched up surprisingly well. The trend of observed against expected probability of persistence was essentially linear although observed persistence tended to fall below that predicted, a little for herbivores and a lot for carnivores. Irrespective, Goodman's model was clearly several steps in the right direction.

Short & Turner (1991) questioned Belovsky's algorithm on the grounds that a species of kangaroo has persisted for the last 8000–10 000 years with a population size of about 2000 on the 233 km^2 Barrow Island off the west coast of Australia, which is a population size a couple of orders of magnitude smaller than the Goodman/Belovsky model would predict as a minimum viable population for that time interval. In fact, the two observations are not necessarily at variance because the distribution of persistence times on replicate islands is likely to have a standard deviation comparable in size to the mean (Goodman 1987) and because the critical comparison would require knowing the proportion of islands (their differing areas partialled out) on which that kangaroo died out over the same period.

Belovsky's comparison of observed and expected, impaired as it may be, is yet adequate to demonstrate that most national parks are too small to discharge their stated function (Schonewald-Cox 1983; Newmark 1985; Salwasser, Schonewald-Cox & Baker 1987; Grumbine 1990). This is a significant finding and entirely a product of the small-population paradigm.

PVA is used also within the small-population paradigm to predict persistence of a threatened species as against deducing persistence retrospectively. Seal (1991) relates how VORTEX (see previously) is used by one of the IUCN specialist groups to estimate the degree of risk faced by a population of concern. PVA used thus answers the question: how long is this population likely to persist if present conditions obtain? It is usually asked only of a population already identified as at risk because it is small. Thus, PVA tends to be used within the small-population paradigm to confirm what is already known (there is a risk), and the gravity of that revelation may well be appropriately emphasised by its emerging as numbers from a computer rather than as a verbal statement of the obvious.

The instantaneous dynamic behaviour of a population—whether in given circumstances it will increase, decrease or remain stable—is entirely a function of its current age distribution interacting with its age-specific schedules of fecundity and mortality. Its

trajectory of numbers reflects the history of those instantaneous events in a general way but the process can be modelled further by changing the age-specific constants to variables drawn from probability distributions. A realistic level of unpredictability is thus added to what the population might do.

A PVA is simply such a population-dynamics simulation modified to answer one question: what is the expected mean persistence time if given life tables and fecundity tables remain constant or change in a systematic manner? It answers the heuristic 'if...then' questions posed by the user changing parameter settings between runs. It is less useful for answering questions about a specific population under threat because the necessary data are seldom available. All threatened species known to me share one characteristic: little is known about their age-specific rates of fecundity and mortality upon which a PVA must necessarily operate. Consequently, the PVAs on them are essentially games played with guesses.

People charged with sorting out the problems posed by species at risk are not always able to avoid an exaggerated impression of what a PVA reveals about a real conservation problem. Note that these mistakes are not made by the writers of these programs [see Lacy (1993) for a clear exposition of the limitations of such software]. They are made by users who, lacking the knowledge that simple answers are being supplied to simple questions, conclude that the simple answer is a full answer to a full question. Hence, there are about as many ecological misunderstandings as valid ecological insights generated by the use of PVA methodology in conservation, not the least of the problems being that PVAs often asks the wrong question. Neither does it help that few PVAs surface above the grey literature. [A cheering exception is Lacy & Clark's (1990) excellent account of applying PVA methodology to a population of the eastern barred bandicoot, a common Tasmanian species with a remnant population in Victoria, Australia.] More commonly they are presented as reports to government departments, the accompanying management recommendations being converted directly into government conservation policy without passing through the bracing cold shower of independent critical review. PVA results have frequently been used to argue for a particular management action. It should be obvious that there is no logical connection between a PVA identifying a level of generalized stochastic threat and the formulation of a recovery plan designed to lower that risk. The PVA does not identify the cause of the problem. The counter-argument (Seal 1991), justifying management recommendations as logical outcomes of PVA methodology, is meretricious.

CAPTIVE BREEDING AND RELEASE

There are only a few examples of a captive breeding programme returning an endangered species to the wild. One is the Lord Howe woodhen, another (the Arabian oryx) is outlined below, and three further examples, not review here, are provided by the Hawaiian goose or nene *Branta sanvicensis* (Kear & Berger 1980), the Indian coney *Geocapromys brownii* (Oliver 1985) and the European otter *Lutra lutra* (Jefferies *et al.* 1986). The most recent treatment of the subject is a Zoological Society of London Symposium edited by Gipps (1991), but even here the emphasis is rather on planning future attempts than reporting past successes. With the increased sophistication of captive breeding evident over the last decade, and an increased sensitivity to the problems of re-introduction, we can expect to see this technique used more frequently in the future.

Example: the Arabian oryx

The Arabian oryx *Oryx leucoryx* can be rated tentatively as a success story (Stanley Price 1989). It became extinct in the wild in 1972 because of overhunting. Fortunately, a breeding colony with nine founders had been established in 1963 at the Phoenix Zoo, Arizona, a site chosen for the close match of its climate to that of the Arabian Peninsula. A further breeding group was established at the Los Angeles Zoo from specimens purchased from the Riyadh Zoo. Surplus animals from Phoenix were dispersed to other US zoos and the aggregated population continued to increase, numbering 105 by the end of 1976.

In 1976 the Omani interior was surveyed by IUCN to gauge the likelihood that a re-introduction would succeed and to find a suitable site. Nineteen animals were imported to Oman between March 1980 and January 1984, mainly from the San Diego Wild Animal Park, and released into a 100 ha enclosure at the planned point of liberation. Two social groups totalling 21 individuals were released, one in 1982 and the other in 1984. Some of the animals wore radio collars. The re-introduced population was monitored closely as it increased at $r = 0.20$ per year.

GENETICS OF WILD POPULATIONS

Despite the prominence granted to genetics within the small-population paradigm there has so far been little application outside zoos. An exception is the work on the cheetah *Acinonyx jubatus* which has been widely quoted within the conservation biology literature as showing a relationship between fitness and equilibrium heterozygosity, as against fitness and a recent decline in heterozygosity. It is therefore examined at length.

Example: the cheetah

The cheetah is a highly specialized hunter of gazelle-sized prey. Where there are no such prey there are no cheetah. It is widespread in Africa south of the Sahara

although patchy in distribution, it is now rare or extinct in North Africa, and its previous distribution in Asia has contracted to a remnant but healthy population in northern Iran (Caro 1991; Sunquist 1991). It hunts by day, does not cache its food, and seldom scavenges as lions and leopards will. Perhaps for those reasons the cheetah has never reached densities achievable by those other large cats. It is adapted to mesic and desert areas, being at highest densities in such places as in and around the Kalahari Desert and in the Serengeti ecosystem. At least until the early 1960s it was the commonest large predator across the Sahel (Thane Riney, personal communication) which stretches for 6500 km (the equivalent of London to Delhi) across Africa under the Sahara.

O'Brien *et al.* (1983b) reported that they were unable to detect any heterozygosity at 47 isozyme loci in 50 cheetah from two areas of southern Africa (Namibia and northern Transvaal) and five cheetah of southern African origin in two US zoos. Two-dimensional electrophoresis of 155 soluble proteins from fibroblasts yielded a mean heterozygosity of $H = 0.013$, which can be compared with the $H = 0.02$ for house mice, analysed for those same proteins in the same laboratory. The two-dimensional soluble protein analysis was limited to six South African cheetah from two zoos in the US and one in the Netherlands. The urgency of the cheetah's conservation problem was highlighted by the authors' belief (p. 461) that the cheetah was restricted to East Africa (i.e. Uganda, Kenya and Tanzania) and South Africa (including Namibia), which is curious in that they quoted Myers (1975) who discussed with maps its distribution and status in 22 countries south of the Sahara.

A discussion point of the previous paper (O'Brien *et al.* 1983b) was elevated to the theme of the next paper (O'Brien *et al.* 1985), as exemplified by its title: 'Genetic basis for species vulnerability in the cheetah'. It reported seven lines of evidence for the conclusion flagged by that title. First, it reviewed the evidence of genetic uniformity presented by the previous paper. Secondly, it provided data on acceptance of reciprocal skin grafts between unrelated individuals, thereby suggesting low genetic variance at the loci controlling the immune response. Thirdly, it reported that, as expected from its low heterozygosity, cheetah inbred in zoos exhibit no inbreeding depression. Fourthly, it suggested that juvenile mortality is excessive both in captivity and in the wild. Fifthly, cheetah were considered highly susceptible to disease; and sixthly, cheetah have a high frequency of sperm abnormalities. On these bases the authors suggested that the cheetah's grim conservation status had a largely genetic origin. These postulates will be examined in turn.

The 1983 results on low heterozygosity at isozyme loci in southern African cheetah were reported again by O'Brien *et al.* (1985) with the difference that the number of loci sampled by one-dimensional elec-

trophoresis was expanded from 47 to 55, but still without detection of polymorphic loci. Subsequently, East African cheetah were examined (O'Brien *et al.* 1987) and these revealed a heterozygosity of $H = 0.014$ at isozyme loci. That paper also reported a previously undetected isozyme polymorphism in southern African cheetah to give a revised $H = 0.0004$ for the population of that region.

O'Brien *et al.* (1985) reported experimental exchange of skin grafts between pairs of unrelated southern African cheetah. There is some confusion over the number. The results of this experiment were first published as an abstract (O'Brien *et al.* 1983a) where the number of unrelated cheetahs exchanging grafts was given as 16. The summary of the 1985 paper announced that '14 reciprocal skin grafts between unrelated cheetahs were accepted' but the paper itself seems to indicate that grafts were exchanged between only 12 unrelated cheetah. Acceptance or rejection was symmetrical within pairs; if one member of the pair accepted a graft from the other then the reciprocal graft also took. The authors concluded thereby that cheetah have a very low level of heterozygosity at the major histocompatibility complex. However that experiment is not without problems. The animals were at three places, described as 'Wildlife Safari', 'Johannesburg', and 'De Wildt'. 'The De Wildt and Johannesburg studies were terminated early (day 23).' It is not made explicit what 'terminated' means here nor why the individual termination dates given in their Table 3 for this sub-set, in contrast to the 23 days given in the text, varied between 14 and 44 days. In either event, the time is insufficient to gauge acceptance or rejection because rejection of the four grafts that were definitely rejected occurred at days 39–49, >41, 46–51, and 70. The six short-term grafts must therefore be eliminated from consideration. O'Brien, Wildt & Bush (1986, p. 72) gave a different account of this experiment: all the grafts were monitored twice a week 'for about eight weeks' and 'Remarkably, all allografts were accepted and indeed were indistinguishable from the autografts throughout the 10-to-12-day period.' However, the hard evidence for a general lack of rejection, after the short-term trials are eliminated, comes down to reciprocal graft acceptance by only one of three pairs of apparently unrelated cheetah (in Oregon) as revealed by O'Brien's *et al.* (1985; Table 3). These trials produced an interesting hypothesis that should be tested by an experiment following the standard rules of experimental design.

The third approach was through detection of inbreeding depression or, more precisely, through the lack of it. Close inbreeding in captivity leading to exposure of deleterious recessives is associated with increased juvenile mortality in many mammalian species (e.g. Ralls *et al.* 1979). However, if the cheetah were essentially homozygous, there would be little or no hidden allelic variation that could be exposed by inbreeding and worked on by selection. O'Brien *et al.*

(1985), therefore, hypothesized that the cheetah, in contrast to most other mammals, would exhibit no inbreeding depression as detected by increased juvenile mortality. They had three sets of mortality data. First, there were 183 births in the De Wildt Cheetah Breeding and Research Centre of the National Zoological Gardens in Pretoria. Sixty-seven of these cubs died before 6 months of age and 116 survived. Their parents were judged unrelated. Then there were 194 births to unrelated parents recorded in the *North American Regional Cheetah Studbook* (died-/survived = 51/143) and 43 births (19/24) to related parents recorded in the same publication. O'Brien *et al.* (1985, p. 1428) interpreted these frequencies to mean that 'there is no significant difference between infant mortalities from inbred and noninbred matings of cheetahs, which is not surprising in the light of the genetic status of the species.' To achieve that result they must have pooled the frequencies of the De Wildt and the studbook (unrelated) samples, testing that combined sample against the studbook (related) sample. That gives a $2 \times 2\chi^2$ of 2·359 and a probability of 0·125 based on 420 births. However, the first rule of pooling is that like must be combined with like. If the De Wildt and the studbook (unrelated) cubs had significantly different rates of mortality, although both coming from unrelated parents, they could not be pooled and tested against a third sample of cubs from related parents that was not spread across those two captive populations. Site and relatedness would be confounded thereby. In fact, the juvenile mortality rate at De Wildt was 0·366 as against 0·263 for the studbook (unrelated) sample. Cubs of apparently unrelated parents in the zoos covered by the studbook survived significantly better than those of apparently unrelated parents at De Wildt. The contingency table χ^2 of 4·199 testing survival between these two samples of cubs produced by unrelated parents yields a probability of 0.040. Consequently, the pooling is invalid for their next step and any comparison between cubs of related and unrelated parents must be limited to the two studbook samples. With these the mortality rate of cubs produced by unrelated parents is 0·263 as before and that from cubs of related parents is 0.442. The χ^2 of 4·591 is significant ($P = 0·032$). In contrast to the findings of O'Brien *et al.* (1985), cheetah apparently have substantial variability at those loci influencing juvenile survival.

The fourth line of evidence came from comparing juvenile mortality of captive cheetah with that of 28 other species in captivity, none related to the cheetah at even the ordinal level (O'Brien *et al.* 1985; Fig. 1). There are two quite separate questions posed by this comparison. First, what does the ranking of juvenile mortality among species in zoos reveal about its ranking among those same species in the wild? Secondly, what does the level of juvenile mortality in the wild reveal about the mean fitness of a given species relative to that of other species in the wild? Comparisons within a species are quite another matter. The second question does not have to be answered unless fitness is redefined to a form unfamiliar to population geneticists. There is no necessary relationship between fitness and juvenile mortality. If there were we would have to concede that a species of oyster with a juvenile mortality rate of order $1-10^{-6}$ is considerably less fit than a species of rhino with juvenile mortality of order $1-10^{0}$. The conclusion is obviously daft when stated in that way but it necessarily follows from assuming that juvenile mortality compares fitness between species. Over its lifetime each individual in a stable population will, on average, replace itself but once. The considerable number of births in excess of that needed for replacement is whittled away mainly by juvenile mortality. As a corollary there must be, and is, a tight correlation among species between fecundity rate and juvenile mortality rate. Hence, a comparison between two wild species, in terms only of birth rate or only of death rate, reveals nothing about how either species is coping. Fitness is the difference between the two, not either alone. That carries over in a general way to species held in captivity, but with these there is a further complication. Some species breed well in zoos, others badly, and yet others start off badly but then improve after their curators learn the tricks of manipulating behaviour, space, shelter, food supply and conspecific company to stimulate successful mating and successful rearing of the young. An interspecies comparison of fitness as revealed by juvenile mortality in a zoo is thus deficient on two counts. The suggestion that the difficulty of breeding a species in a zoo is a consequence of its genetic uniformity (O'Brien *et al.* 1985, Summary) is unsustainable in the absence of corroborative evidence.

The fifth line of evidence concerned susceptibility to disease, this being portrayed as a consequence of low heterozygosity at loci controlling the immune response. The evidence was an outbreak of a coronavirus disease, usually restricted to domestic cats, at a zoo in Oregon. It killed 19 cheetah but none of the 10 lions present. Various diseases affect various species in various ways, irrespective of the heterozygosity of those species. Witness the idiosyncratic selectivity of the Asian rinderpest virus when it swept down through Africa for the first time between 1889 and 1896 (Ford 1971). Establishing a link between susceptibility to disease and heterozygosity requires more disciplined data.

The sixth line of evidence was provided by the cheetah having appreciably lower concentrations of spermatozoa in the ejaculate than do domestic cats. Furthermore, an average of 71% of their sperm are abnormal compared with 29% in domestic cats. However, this would indicate reproductive dysfunction only if linked to a demonstrated low conception rate in the wild. No such evidence has been offered.

These conclusions will be summarized. O'Brien and his colleagues have demonstrated rather low het-

erozygosity at isozyme loci in the southern African and East African cheetah populations. The cheetah thus joins the 30% of other mammalian species with an estimated $H < 0.02$ for that part of the genome (see Table 2). However, heterozygosity is moderate at those loci examined by two-dimensional electrophoresis, being about half that found in people and about two-thirds that found in house mice. In contrast to their published conclusions, the available inbreeding data suggest a high level of heterozygosity at those loci influencing juvenile mortality, a point noted also by Pimm (1991, p. 159) and Hedrick (1992). The experimental exchange of skin grafts is flawed technically, and in its reporting, to the extent that little can be extracted from its results. The information presented on sperm abnormalities, susceptibility to disease, juvenile mortality, maternal neglect, low density and restricted range of distribution, does not reach the minimum standards of evidence or logic sufficient to suggest that the cheetah is biologically impaired and that this is a significant cause of its endangered conservation status. In brief there is a justifiable doubt that 'The species as a whole is suffering from the effects of what we call inbreeding depression' (O'Brien 1991, p. 146).

O'Brien and his colleagues reported their research in an inconsistent fashion. Several authors quoting it subsequently were understandably confused. For example, Hedrick (1992) had 14 unrelated cheetah accepting reciprocal skin grafts, and he restricts the distribution of the species 'to two wild populations in southern and eastern Africa'. If 'The cheetah is an important natural experiment for conservation biology' (Allendorf & Leary 1986, p. 75), what is the hypothesis and where are the controls? A bizarre extrapolation (unless it is simply a droll spoof) was the suggestion (Parnham 1991) that all the cheetah remaining in the wild be transported to Europe and North America where their immune-response problems would receive more sympathetic treatment.

There is no evidence known to me suggesting that the cheetah is in any trouble in the wild except where subject to excessive offtake by hunting and by capture of cubs (see Myers 1975, p. 61), or where its prey has been reduced by cattle grazing, by scrub encroachment, by hunting, and by conversion of savanna to cropping land. Unfortunately, the effect of those influences has increased steadily over the last several decades. Myers (1975) reckoned that cheetah numbers probably halved between 1960 and 1975. How far they dropped further between 1975 and now is anyone's guess.

DECISION ANALYSIS

Decision analysis is commonly employed in making decisions about how a piece of land should be used, but it has seldom been employed, as against advocated, within the small-population paradigm. I

know of only four examples—management of Sumatran rhino (Maguire *et al.* 1987), management of the eastern barred bandicoot (Maguire *et al.* 1990), allocation of resources to breeding tigers in zoos (Maguire & Lacy 1990) and management of grizzly bears (Maguire & Servheen 1992)—and will review the first.

Example: *the Sumatran rhino*

The Sumatran rhino *Dicerorhinus sumatrensis* occurs as small and scattered populations on the Malayan Peninsula, in northern Borneo, in Sumatra, and possibly also in Burma and Thailand. It is rated endangered by the IUCN, being under continuing threat from unauthorized hunting and forest clearing.

Maguire *et al.* (1987) used it to demonstrate the technique of decision analysis applied to deciding between competing recovery plans. Their methodology is the same as that reviewed earlier in this paper. Table 7 shows their decision tree for a set of management options ranging from status quo management at one extreme to captive breeding at the other. The option of increased support for control of poaching, leading to a postulated pE of 0.45, comprised funding 10 additional rangers and five vehicles, the effort being split between northern Borneo and Sumatra. The 'expanded reserve' called for doubling the size of an existing national park, the 'dam' condition reflecting a tentative suggestion that the same area might be used for a hydroelectric project. 'Fencing' referred to a scheme to 'fence an area in an existing or new reserve, managing the resultant high density of rhino with supplemental feeding and vet-

Table 7. Decision tree for managing Sumatran rhino (modified from Maguire *et al.* 1987). An event has a probability p of occurring over a specified interval, there is a probability pE that the population will go extinct if that event occurs, and the expected probability of extinction over the time interval is E(pE)

Action	p Event	pE	E(p)E
Status quo	0.1 epidemic	0.95	0.86
	0.9 no epidemic	0.85	
Control poaching	0.2 increased support	0.45	0.84
	0.3 no change	0.86	
	0.5 decreased support	0.98	
New reserve	0.6 timber harvest	0.9	0.69
	0.4 protected	0.37	
Expand reserve	0.1 dam	0.9	0.53
	0.2 timber harvest	0.9	
	0.7 protected	0.37	
Fencing	0.2 disease	0.95	0.55
	0.8 no disease	0.45	
Translocation	0.1 success	0.75	0.93
	0.9 failure	0.95	
Captive breeding	0.8 success	0	0.19
	0.2 failure	0.95	

erinary care, as in the successful South African rhino ranches.'

One could criticize elements of this model. The interactions between strategies are not fully explored (a point the authors made themselves), and changes in degree (e.g. increased support for control of poaching) are considered at only one arbitrary level. However, that would be ungracious. Neither need we be concerned about the correctness of the probabilities attached to the various actions and events while the table is used simply to demonstrate a method of numerical analysis. In fact those probabilities 'are subjective, but represent a synthesis of opinion from rhino biologists and managers' (Maguire *et al.* 1987, p. 144).

There is no problem with this paper as summarized so far, but it then changes character. Under the subheading 'Evaluating management options' the probabilities of this analytical exercise become authentic. 'Translocating animals among wild subpopulations is far too risky to be recommended. Its E(*p*E) is even higher (0·93) than for the status quo (0·86). The chances of success and attendant benefits to the populations are not high enough to outweigh the loss of translocated animals if the programme fails. Captive breeding is the most promising option in terms of minimizing the expected probability of extinction for the species, with an E(*p*E) of 0·19. Even if removal of rhinos to captivity raises *p*E for the wild population, the chances of successful captive breeding are high enough to justify this option' (Maguire *et al.* 1987, pp. 152–153). The authors fell victim to the common fallacy of imagining that their thought experiment was the real world. On the basis of their data-free analysis they confidently recommended captive breeding as the correct option for conserving this species.

Nigel Leader-Williams, a rhino ecologist who presumably was absent while the 'synthesis of opinion from rhino biologists and managers' referred to above was formulated, injected data into the argument (Leader-Williams, in press). He showed that there were several examples of successful protection of wild rhino populations in both Africa and Asia through adequate funding and concentration of effort, that rates of increase of protected rhino populations are substantially higher in the wild than in zoos, and that the annual cost of protecting a rhino in the wild ($575 per individual or $230 per km² for black rhinos in Africa in 1980) is around one-third the cost of maintaining it in a zoo. Furthermore, 'over the past five years, two zoos or zoo partnerships have apparently spent $2·5 million just to catch so called 'doomed' Sumatran rhinos, with the aim of establishing captive breeding populations of Sumatran rhinos in zoos (see Nardelli 1988). This sum does not include the costs of maintaining the rhinos in zoos once captured. Losses of rhinos during capture (three deaths) and particularly during post-capture (six deaths) have been high … To date (May 1991), 21 rhinos are in captivity and there have been no births, except to one female who

was pregnant when captured. In contrast, at $230 km⁻², $2·5 million could effectively protect 700 km² of prime rhino habitat … for nearly two decades. At their apparent normal densities of 0·1 rhino km⁻², an area of this size could hold a population of 70 Sumatran rhinos (see Nardelli 1988) which, with a rate of increase of 0·06 per year shown by all other rhino species given adequate protection [in the wild], would be expected to give birth to 90 calves in this period.'

Use of the declining-population paradigm on wild populations

The manner of the declining-population paradigm is to identify the agents of a population decline and by opposing end them. Ideally, the process comprises a minimum of five steps.

1. Use scientific method to deduce both why the population declined and which agent caused the decline. Do not assume that the answer is already provided by folk wisdom, lay or scientific.
2. Remove or neutralize the agent of decline.
3. Release a probe group to confirm that the cause of decline has been deduced correctly [see Griffith *et al.* (1989) and Short *et al.* (1992) for examples)].
4. If so, restock unoccupied areas by translocation or, if the remnant population is too low to risk further reduction, breed up a protected stock as fast as possible, as near to the problem site as possible, and release it as soon as possible.
5. Monitor the subsequent re-establishment.

All the salient points can be made by two examples, one that got it right and one that got it wrong.

Example: the Lord Howe woodhen

The Lord Howe woodhen *Tricholimnas sylvestris* is a rail about the size of a barnyard fowl. It lives on the 25 km² Lord Howe Island in the south-west Pacific, the nearest dry land from there being the Australian coastline 570 km to the west. Lord Howe was one of the few Pacific islands, and the only one uplifted high, apparently not discovered by Polynesians, Melanesians or Micronesians before European contact, and which therefore suffered none of those multitudinous extinctions, caused directly or indirectly by people, that had significant ecological and social impact in the Pacific over the first millennium AD. Olson (1989, p. 52) judged from sub-fossil evidence that almost every oceanic island had its own species of flightless rail and that 'Extrapolating from the number and size of islands in Oceania, we may expect that hundreds of species of flightless rails have been exterminated in the Pacific in the past 2,000 years or less. Exclusive of continental islands, New Zealand, and the Solomons, only fourteen species of flightless rails, of all the hundreds predicted, were recorded in the historic period

in the Pacific, and all but three of these are already probably extinct.' I relate this considered opinion to make the point that the continued existence of the Lord Howe woodhen is no trivial issue, that if yet another vertebrate species is to be lost the Lord Howe woodhen should be well down the list of those nominated for benign neglect.

People first saw Lord Howe Island in February 1788 (*Supply*, ship of the line, lieutenant Henry Lidgbird Ball master) at which time it was home to 13 species of land birds of which nine became extinct over the next 70 or so years. The history of the Lord Howe woodhen is related by Miller & Mullette (1985) and by Fullagar (1985).

In the late eighteenth and the first half of the nineteenth century the island was visited regularly for water and food (which no doubt included woodhen) by sailing ships, mainly Yankee whalers. It was settled in 1834. Pigs were introduced about 1800, dogs and cats before 1845 and goats before 1851; the black rat *Rattus rattus* came ashore from a ship-wreck in 1918. By 1853 the woodhen's range was restricted to the mountainous parts of the island and by 1920 it had apparently contracted to the summits of Mt Gower, an 825 m (2700 ft) mountain almost surrounded by near vertical cliffs rising out of the sea, and Mt Lidgbird (765 m), an even more difficult summit to get at. Mt Lidgbird can virtually be ignored. Almost all the birds were on the summit plateau of Mt Gower (25 ha) which is clothed by dripping moss-forest hidden by the perpetual cloud of high Pacific peaks, a very different place from the coastal flats that once provided the bird's preferred habitat. The acute conservation problem posed by this species was not recognized until 1969 after which the population was monitored every year. Numbers were stable at between 8 and 10 breeding pairs, although in one year they went down to 6 pairs. No more than 10 territories could be fitted into the available space and so it is reasonably certain that this alpine population, and probably the entire species, did not exceed that size over the 60 years between 1920 and 1980. Over the previous 60 years it could hardly have been more than double that.

Miller lived on the island between May 1978 and May 1980. In that time he methodically tested and rejected several hypotheses as to why the birds were presently restricted to the mountain tops. He quickly rejected by way of elegantly designed comparisons, and a huge amount of grinding field work, the standard ones implicating food supply and habitat. The popular and obvious nomination for the agent responsible for contraction of population size and range was the black rat which had been implicated in the extinction of several species of birds on other islands. However, Miller showed that in this case the rat was not to blame. Trapping at seven sites around the island and on the summit of Mt. Gower revealed that rats were more common on Mt Gower than on any other

sampled part of the island, all comparisons being statistically significant. He finally narrowed down the culprit to be the feral pig which will kill and eat incubating birds, and will destroy the nest and eggs. He discovered from his mapping of distributions that these two species do not overlap, even though the boundaries of each may approach within a few map metres. That is, close in two dimensions but not always so in three. Pigs are physically incapable of accomplishing the minor mountaineering feat of reaching the summits of Mt Gower and Mt Lidgbird. The birds are entirely capable of making that journey in the reverse direction.

The pigs were shot out in the period 1979–1981 as a consequence of Miller's reports to his Department. In 1980 a breeding program was instituted because direct stocking into unoccupied range with wild woodhens from the tiny remnant Mt Gower population was considered too risky. It was run by Glen Fraser, a young New Zealander imported for his expertise in breeding endangered New Zealand birds. The breeding facility seeded with three pairs from Mt Gower was built at sea level within a large enclosure of closed-canopy lowland palm forest. Visitors were not welcomed.

Thirteen chicks were reared in the first season of captivity, 19 in the second and 34 in the third. The tell-tale signs of inbreeding depression did not appear. Twenty-two progeny from the breeding programme were released in four batches at one location between May 1981 and June 1982 and that nucleus was monitored closely. By February 1983 some had commenced breeding. The rest of the birds were then released and the captive breeding terminated at the end of 1983. By 1987 and today (1993), the population appeared stable at about 180 birds, 50-60 breeding pairs, and that number seems to saturate all the suitable habitat on the island, mainly palm forest. The operation cost around $A300 000 ($US200 000) at the value of the 1985 dollar.

Although this population is almost the type example of that inspiring the small-population paradigm (a very small population with numbers capped), most of the conservation biology applied to it aligned with the declining-population paradigm. Not to be ignored, however, is the application of the small-population paradigm at the captive breeding stage. The success of this rescue is largely attributable to the two paradigms coming together at the right time and in the right way. The steps followed for the woodhen—diagnose the agent of decline, neutralize the agent of decline, re-establish the species of concern—may serve for almost any other troubled species.

Example: the California condor

The California condor *Gymnogyps californianus* ranged last century from the British Columbia in the north to Arizona and New Mexico in the south, but

its range had contracted by 1940 to a small area north of Los Angeles. Koford (1953) estimate from sightings that only about 60 individuals survived in the early 1950s. Annual surveys by simultaneous observation of known concentrations were begun in 1965, but abandoned in 1981 as subject to unacceptable error. Photographic identifications were then used to generate a total count of 19–21 birds in 1983 (Snyder & Johnson 1985). The decline continued until the last eight wild condors were caught in 1985 and added to the captive flock.

The initial decline may have been caused by shooting and loss of habitat, but the evidence is anecdotal and flaky. Inadequate food supply was suggested as a cause of the decline during the 1960s, and so deer killed by cars and trucks were cached at condor feeding stations between 1971 and 1973 to alleviate the perceived shortage of food (Wilbur, Carrier & Borneman 1974). That charity was run for too short a time to gauge its effectiveness.

The connection between toxic organochlorines and eggshell thinning was established in the late 1960s, but the resultant flurry of studies focused on birds that ate fish and other birds, scavengers such as the condor being assumed less at risk. The possibility of a causal link between environmental toxins and the later decline of the condor was recognized in the mid-1970s but not investigated for many years (Kiff 1989). Eggshell from condors had been collected from the late 1960s but for various reasons, including mishaps to the samples, analyses were delayed until the mid-1970s. They showed that the shells were thinner and their structure different from shells collected before 1944. Over the same period measured DDE levels rose markedly.

There was little agreement as to why the eggs often broke. Even the monitoring activities themselves were suspected as the cause. The evidence for organochlorines was circumstantial but it led Kiff (1989) to conclude that 'DDE contamination probably had a very serious impact on the breeding success of the remnant population in the 1960s, leading to a subsequent decline in the number of individuals added to the pool of breeding adults in the 1970s.' The US banned DDT in 1972. The few eggs measured after 1975 had thicker shells which led to the tentative conclusion that the agent causing the latter-day decline had been identified and cancelled. However, in March 1986 an egg laid by the last female to attempt breeding in the wild was found broken. Its thin shell was suspiciously reminiscent of the 'DDE thin-eggshell syndrome'. In the meantime, analysis of tissue from wild condors found dead in the early 1980s revealed that three of the five had died from lead poisoning, probably from ingesting bullet fragments in dead deer. Other condors had elevated lead levels in their blood (Wiemeyer *et al.* 1988). The identification of yet another toxin in the condor's food led to provision of 'clean' carcasses at feeding stations just before the last condor was taken into captivity.

The sorry story of the California condor stresses the paramount need to determine, not assume, the causes of a decline, to view correlations not as results but as testable hypotheses, and to investigate and exonerate suspected causal agents by disciplined application of scientific method. As Macnab (1983) emphasized, management actions are experiments and should be run as experiments according to the standard rules of experimental design. Otherwise nothing is learned from them.

Discussion

The only convincing example of the small-population paradigm and the declining-population paradigm coming together to solve a conservation problem is the rescue of the Lord Howe woodhen. Diagnosis and treatment (declining-population paradigm) were combined with on-site captive breeding (small-population paradigm) to provide a heartening success. Another example of on-site breeding being used to tackle a conservation problem (the Hawaiian goose) has not so far been successful because it lacked the diagnostic steps of the declining-population paradigm. 'We still do not know, other than in general terms, what brought the species so low, and so cannot be sure that the hazards have been removed or are being effectively controlled' (Kear & Berger 1980).

The account of the conservation problems faced by the Hamilton population of the eastern barred bandicoot *Perameles gunnii*, alluded to previously, is published under the title Management and Conservation of Small Populations (Clark & Seebeck 1990). It comes to us from the direction of the small-population paradigm and so includes a competent PVA, and a thorough genetic analysis. As viewed from the vantage of the declining-population paradigm, however, the problem is not that the population is small (a few hundred) but that it is declining at 25% per year. Although the cause of the decline has been speculated upon (predation by cats, by foxes and by dogs; traffic accidents; and pesticides) no rigorous study has been launched to determine the dominant cause of the decline or the relative contributions of various agents of decline. A little experimental investigation of the causes of the decline might usefully be hooked into the current protocol supplied by the small-population paradigm.

A further area in which the two paradigms might concatenate with advantage is in the overlap zone between metapopulation structure (small-population paradigm) and habitat fragmentation (declining-population paradigm). The two are linked conceptually in that both deal with patches, static in size and number over time for metapopulations, and dynamic in size and number over time for habitat fragmentation. The deduced ecological effects of metapopulation structure come largely from theory backed by some experimentation (see previously). The

endangering effects of habitat fragmentation are deduced mainly from observation (see previously). May (1991) integrated the two ideas by considering habitat fragmentation as equivalent to a collapsing metapopulation, modifying the patch dynamics equations accordingly. He pointed out the parallels between such a composite model and that depicting the eradication of infectious diseases by vaccination of a proportion of the 'patches' (i.e. hosts) harbouring infection (Anderson & May 1985). Studies of the effects of habitat fragmentation on endangerment of species will clearly benefit from an injection of such theory from both the small-population paradigm and from epidemiology.

The small-population paradigm has contributed significantly to the theory underpinning the genetics and dynamics of small populations whose numbers are capped. The application of that theory has so far been restricted largely to captive breeding and to the design of reserve systems. It has had little to say about populations driven to extinction and it has confused that issue by labelling those declines 'deterministic', as if the processes were a mirror image of those stochastic events affecting small populations. In addition to supplying the wrong meaning (the processes of driven extinction lacks none of the stochastic elements of the small-population paradigm) that word has an unfortunate connotation: stochastic models are mathematically elegant and theoretically exciting; deterministic models are their poor cousins. 'Deterministic' is enough to scare off potentially helpful applied mathematicians jealous of their professional reputations, and so the processes of driven extinction attract scant theoretical attention.

Much of the small-population paradigm is taken up by the genetics of small but stable populations. About half of conservation biology published as books in the 1980s (Soulé & Wilcox 1980, Frankel & Soulé 1981; Schonewald-Cox *et al.* 1983; Soulé 1986, 1987a; Western & Pearl 1989) was about genetics (1010 of 2162 pages). I would have guessed higher because the chapters on genetics are the ones that stay in the memory. They were intellectually taut, replete with general principles, and a joy to read. The ecological offerings with few exceptions were no less important, but rather less memorable.

There is a danger that a student might take that emphasis literally and conclude that genetic malfunction was a significant cause of extinction in the wild. The literature of conservation genetics provides no hint that it might be otherwise. Yet no instance of extinction by genetic malfunction has been reported, whereas the examples of driven extinction are plentiful. Genetic thinking often intrudes where it is not relevant and where it sometimes obscures the real issues—an interesting case of overdominance. An example is the penchant for converting willy-nilly the size N of a population under study to its effective population size (genetic) N_{eg}. That makes sense only

where the dynamics of the population are under control and where the population is expected to stay at low numbers for a long time, as for example in a zoo. However, if the problem were a wild population so small that it could easily die out by chance in the near future, or if the aim were to get a population back up to a safe level as soon as possible, the relevant population sizes are first N and second N_{ed} (the demographically effective population size). Demographic stochasticity acts upon N_{ed} over a short interval and on N over the short term. N_{eg} is irrelevant. Yet N_{eg} is the metric most often invoked whatever the context, even though demographic and environmental stochasticity usually pose more immediate and potent dangers than do genetic drift and inbreeding depression (Lande 1988); and external agents forcing a decline are manifestly more prevalent and dangerous than either.

That is not an argument for less conservation genetics, but for more of it. There is no shortage of ideas in that area, but an acute shortage of information. As indicated earlier, with the notable exception of its application to captive breeding, conservation genetics has not transplanted comfortably to the field. There is an urgent need to elucidate the physiological and genetic basis of inbreeding depression and to test the hypothesis, often stated as a finding, that equilibrium heterozygosity and species vulnerability are causally related.

A high proportion of the blunders in diagnosis of conservation problems within the declining-population paradigm can be traced directly to faulty scientific method: equating association and correlation with causality, failing to identify and cut through confounding of factors, failing to replicate, failing to balance, failing to control. Most of this springs from a creed that the rules of design, and the analyses appropriate to them, might have a place within the pages of a scientific journal, but can be dispensed with by practical people solving practical problems. A highly recommended antidote to that derangement is Underwood's (1990) paper on the logic of experiments in ecology and management. The squeamish will be relieved to hear that it contains no equations.

Study of the causes of extinction has been restricted almost entirely to within the declining-population paradigm, the work of Jared Diamond being preeminent. The small-population paradigm has contributed only the 'extinction vortex', the physiology of a population's death rattle. Despite some advances in determining and classifying the reasons why species go extinct, a comprehensive theory of extinction is not yet on the horizon. It may not be logically possible to unite such disparate, messy and singular events within a neat and non-trivial theory, but the effort should be made even if ultimately unsuccessful. Even a negative result would be a useful insight.

One rewarding line of investigation is the study of past extinctions and near extinctions. The research of

Diamond and Atkinson points the way. People seem to be implicated in most post-Pleistocene extinctions. 'As far as I know, no biologist has documented the extinction of a continental species of a plant or animal caused solely by non-human agencies such as competition, disease or environmental perturbation in situations unaffected by man' (Soulé 1983, p. 112). I am even less informed. I cannot recollect hearing of a non-anthropogenic extinction of an island species (as against an island population) occurring within the last 8000 years. The fossil record informs us that species die out over geologic time without human intervention on both islands and continents, but how recently; or more to the point, what is the background rate of non-anthropogenic extinction? That information can be extracted most easily from regions discovered by people only a short time ago, where their arrival is dated beyond dispute, and where abundant dateable subfossils might relate the history of faunal composition over the Quaternary centuries preceding human settlement. Those criteria eliminate most of the world beyond New Zealand and (possibly) Madagascar. These two countries should be the focus of research directed at determining the background rate of extinction. The Americas will provided an additional opportunity when the chronology of human settlement there is finally sorted out.

Because extinction is a serious issue there is a heavy burden laid upon researchers and managers to think carefully and act responsibly. Theoreticians also. Otherwise they may preside over the extinction of yet another species. Mind experiments of the 'if...then' variety are fundamental to science. They explore the logical implications of simple assumptions. The process is deductive. After the behaviour of a simple model is explored its assumptions may be multiplied and elaborated. If the outcome of the more elaborate model is similar in kind to that of the simple model (for example, when exponential population growth is replaced by logistic, or where a single-species model is replaced by the analogous two-species model incorporating the dynamics of the species' food supply) we become more hopeful that we are onto something, that the outcome may be robust. We may, however, still be well away from reality because the simple model, and even its elaborations, may differ in kind from real systems. The essence of the thought experiment is that only the qualitative outcome is of interest. For example, does environmental stochasticity generate a convex or a concave regression of persistence time T on carrying capacity K? Or are those outcomes dependent on the settings of the constants? Are these conclusions dependent on the structure of the model? Does K as considered here have any objective or useful meaning in the context of a real population?

There is commonly a gap in the chain of logic stretching from a simple thought experiment to a management action. It skips at the point where a quali-

tative conclusion is applied in quantitative form to a specific problem. The constants of a model (which itself may or may not be structurally appropriate) are assigned values, often by guessing, and the output presented as a specific management recommendation. Conservation biology does not hold a monopoly on this malaise—it is an epidemic within wildlife management and fisheries management, for example—but in the field of conservation, where a faulty recommendation may kill off a species, it must be guarded against with vigilance.

The declining-population paradigm is urgently in need of more theory. The small-population paradigm needs more practice. Each has much to learn from the other. In combination they might enlarge our idea of what is possible.

Acknowledgements

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