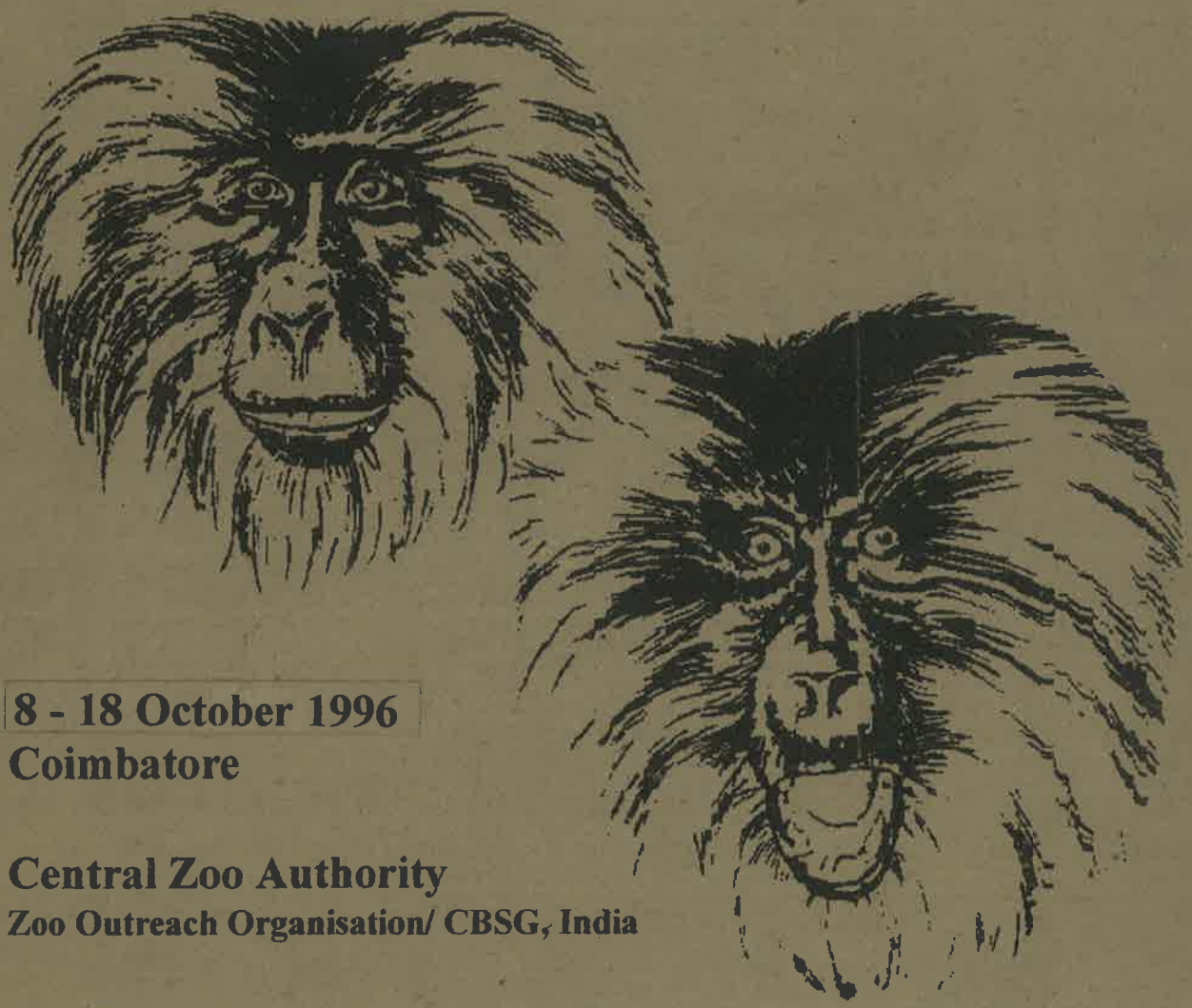
Small Population Dynamics

63/45

Briefing Book

Small Population Dynamics and the Tools of Recovery Training Workshop



8 - 18 October 1996
Coimbatore

Central Zoo Authority
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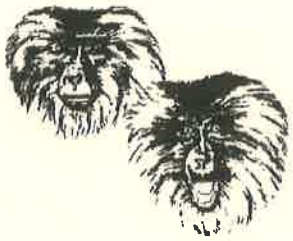

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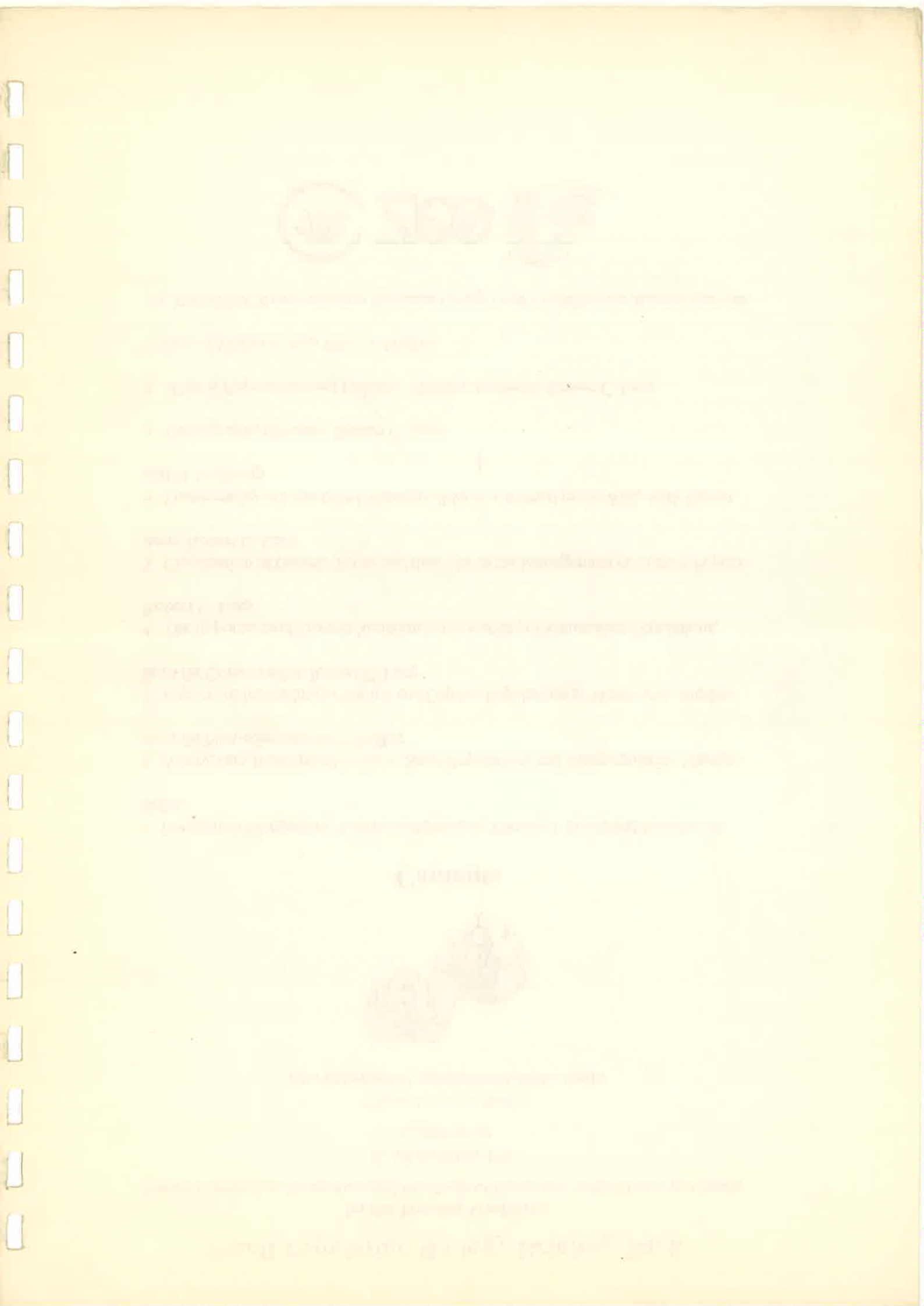

Population management: theory and practice

Int. Zoo Yb. (1988) 27: 26–41

Management of small populations

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It appears that the survival of many species, especially larger vertebrates, will depend upon assistance from captive propagation over the next century or more. Zoos and aquaria can and must serve as arks for vanishing wildlife.

However, captive propagation can truly assist conservation of endangered species only if zoo and aquarium populations are managed genetically and demographically in a manner to reinforce, not replace, wild populations. In the future, conservation strategies will ideally incorporate both captive and wild populations that are interactively managed for mutual support, that is, through regulated interchange of animals or at least genetic and demographic material (e.g. through sperm or embryos). Captive populations can serve as reservoirs of genetic and demographic material that can be infused periodically into remnant wild populations or re-established in vacant wildlands if conditions were suitable. Reciprocally, the wild populations, even if only remnants, will still be subjected to natural selection and thus maintain some

semblance of the characteristic genetic makeup of the species in the wild.

Species are vanishing because a combination of habitat destruction and unsustainable exploitation are reducing and fragmenting wildlife populations. However, the situation is often worse than is indicated by simple numbers because of the particular characteristics of small populations, which are subject to stochastic or random problems that imperil their survival even if the other dangers can be eliminated (Gilpin & Soulé, 1986; Soulé, 1987). As a consequence, even when and where protection of remnant wild populations is feasible, it may not be sufficient to ensure the long-term survival of species. There are three general types of stochastic problems: environmental, demographic and genetic (Gilpin & Soulé, 1986).

Stochastic environmental problems are of several kinds: epidemic, disease, catastrophic events (natural disasters such as earthquakes or floods, fires, wars or, in the captive context, loss of financial or other support) (Dobson & May, 1986). For larger populations, such environmental disturbances may be localised and part of the population might survive (Shaffer, 1987). In general one of the advantages of captivity might be to moderate environmental stochasticity, for example drastic fluctuations in food supply should be less common in captivity than in the wild.

Demographically, stochastic problems afflicting small populations include unexpected failures in reproduction or survival, distortions of age distributions and biases in sex ratio. Severe random fluctuations in birth and death rates will be troublesome in very small populations. The failure of a few ♀♀ to reproduce as expected may have little effect on larger populations, but could be disastrous for a small population with only a few ♀♀. Age distributions will also be more vulnerable to stochastic destabilisation in small populations; extinction through senescence has occurred in a number of captive groups. Another particular problem may be distortions in sex ratio; every zoo manager has been confronted with the phenomenon of a substantial number of consecutive births of predominantly one sex. In a small population such distorted sex ratios can be very disruptive.

Genetically, small populations tend to lose diversity rapidly through the stochastic process of genetic drift, as well as through inbreeding. Genetic diversity is important for both the adaptability of populations and the fitness (survival and fertility) of individuals. The smaller the population, the faster the loss (Appendix 1).

Reduction and fragmentation of small populations, be they captive or wild, convert gene pools into gene puddles that are vulnerable to evaporation in an ecological and evolutionary sense. Genetic and demographic analysis and management can be applied to counteract these problems. In general it may be easier to apply such measures in captivity but the same types of intensive management will increasingly be required in wild sanctuaries which in reality are becoming 'megazoos'.

Multi-institutional population propagation programmes for genetic and demographic management are being developed in several major regions: the Species Survival Plan (SSP) of the AAZPA in North America; the Europäisches Erhaltungszucht Programm (EEP) in Europe; the Joint Management of Species Programme of the British Zoo Federation and the Regional Studbook Programmes of the Anthropoid Ape Advisory Council in Great Britain; the Australasian Species Management Scheme in Australia and New Zealand; the SSP programmes in Japan. It is hoped that eventually these regional programmes can be integrated and co-ordinated internationally. The Captive Breeding Specialist Group of the IUCN Species Survival Commission, in co-operation with the International

Union of Directors of Zoological Gardens, would be appropriate bodies to co-ordinate such co-operation.

These SSP-type programmes will each be based upon a Masterplan for population management. Fundamentally a Masterplan provides *institution-by-institution and animal-by-animal recommendations* for the entire population encompassed by the programme. Individual recommendations should be orientated to well-defined goals and objectives and should reflect attempts at demographic and genetic management. Although the emphasis in this paper is on genetic and demographic management, other considerations such as basic husbandry, behavioural aspects and veterinary care will also be vital to viable captive management programmes. A basic protocol has been developed for this kind of population management in the AAZPA (SSP) programmes (Appendix 3).

Because genetic and demographic problems of small populations are a function of the population's size, a first step in developing a management programme for a captive population is to establish a captive carrying capacity. This will be the optimum number of individuals to be maintained over the long term and will represent a compromise between the minimum necessary for genetic and demographic viability and the maximum that can be accommodated without excluding other taxa from captive programmes.

The lower limit for carrying capacity is the Minimum Viable Population (MVP) size of the captive population. An MVP depends on two sets of factors: the demographic and genetic objectives of the programme and the biological characteristics of the population.

Demographic and genetic objectives include the probability of the population surviving; the kind and amount of genetic diversity to be preserved; the period of time over which this probability of survival and level of diversity are to be sustained.

The perfect programme would ensure a 100% probability of survival and preservation of the total amount of all kinds of genetic diversity in the population for ever. In other words, it would protect against all the problems which confront small populations. Beyond the philosophical observation that nothing is certain or for ever, it is unrealistic to consider such a programme since the size of populations that can be maintained in captivity could never be large enough to achieve its objectives.

A general guideline recommended by some conservation biologists is that captive programmes should attempt to preserve 90% of the average heterozygosity of the founders for 200 years (Soulé *et al.*, 1986). Figure 1 illustrates several examples of minimum effective population sizes that would be necessary to preserve 90% average heterozygosity for 200 years.

Biological characteristics The fact that the MVP size depends on the generation time of the species as well as the number of effective founders leads to consideration of the second set of factors that determine the MVP, the biological characteristics of the population.

1. The generation time: the effect of generation time on the MVP size is based on the fact that genetic diversity is lost generation by generation not year by year. Thus for a given period of time, for example 200 years, a species with a shorter generation time will pass through more generations, have more opportunity to lose genetic diversity and hence require a larger MVP size than a species with a longer generation time.
2. The number of founders: the number of effective founders that establishes a captive population also determines the MVP (Fig. 1). An effective founder is a wild-caught animal that has reproduced to have descendants in the living, managed population. Living animals out

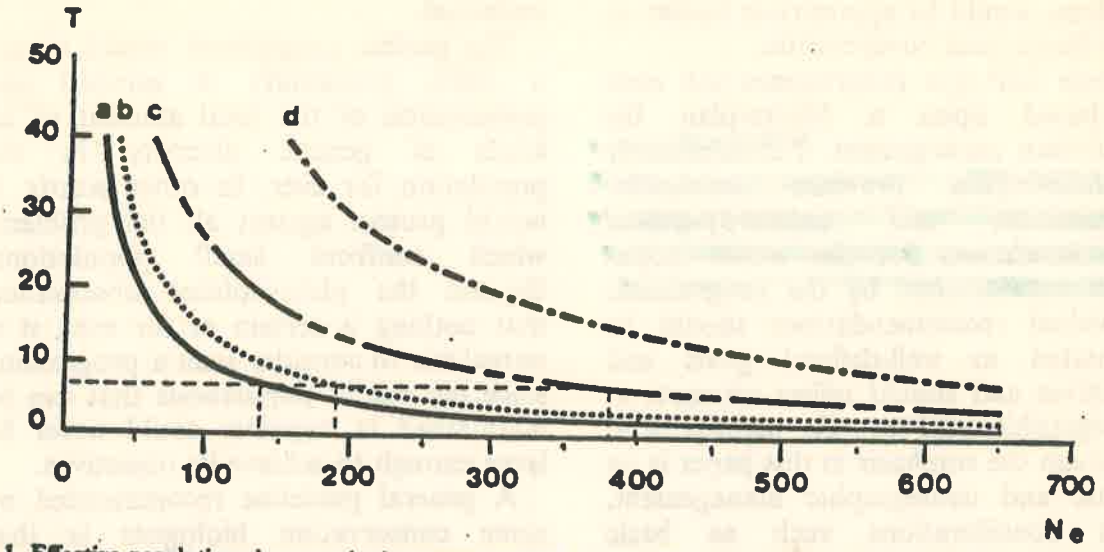


Fig. 1. Effective population sizes required to preserve 90% average heterozygosity for 200 years for populations with various generation times and effective founders. T. generation time; N_e. effective population size; a. no founder effect; b. 20 founders; c. eight founders; d. six founders.

of the wild that have not reproduced may be potential founders but they are not effective until they reproduce. Basically, the fewer the number of founders the larger must be the MVP. There is, however, a point of diminishing returns. For most species increasing the number of effective founders beyond 20–30 (to preserve average heterozygosity) or 30–50 (to preserve rare alleles from the wild population) will not significantly reduce the size of the MVP required for time periods in the order of 200 years (Allendorf, 1986). In most cases founders will not enter the population only at the start of the management programme; periodically, additional founders ('immigrants') will enter the population. As few as one effective wild-caught founder per generation can keep the captive population representative of a wild gene pool.

3. The N_e/N ratio: an MVP prescribes a population size required for some set of objectives. The population size of relevance, however, is not the simple census number (N), rather it is the genetically effective size, denoted by N_e. This is a measure of the way in which the population is reproducing to transmit its genes to the next generation. It is a function of a number of variables: the number of animals that reproduce; the sex ratio of these animals; the mean and variance of their relative number of offspring (lifetime family sizes) (Lande & Barrowclough, 1987). In general the more disparate the sex ratio and family size, the lower N_e will be relative to N. In practical terms, N_e is usually only a fraction of N. Common ratios of N_e in genetically unmanaged populations are 0·2–0·5. As a consequence, actual carrying capacities may have to be several times larger than the N_e necessary to achieve the genetic objectives of the programme. For example, if an N_e of 250 were required for some set of objectives (e.g. maintaining 90% heterozygosity for 200 years), but the N_e ratio of the population was only 0·25, the carrying capacity that would have to be maintained would be 1000 individuals. By managing sex ratios and family sizes, N_e/N ratios can be improved and actual (carrying-capacity) MVPs reduced.

4. Growth rate: until a captive population attains its carrying capacity and while the population size remains small, genetic diversity will be lost at a relatively fast rate. The more rapidly the population can grow to carrying capacity the more it can

N_e and carrying capacity necessary for maintaining the specified amount of genetic diversity for a specified time period

YEARS PER GENERATION:	10	NO. GEN. DURING PERIOD:	20
YEARLY % GROWTH RATE:	1·080	GEN. GROWTH RATE:	2·16
EFFECTIVE NO. FOUNDERS:	25	GEN. EXPON. GROWTH	0·77
ESTIMATED Ne/N RATIO:	0·5		
DESIRED % HET. RETAINED:	0·90		
LENGTH OF TIME PERIOD:	200 YEARS		

Effective Size required to maintain desired amount of original variation for the specified length of time:	199
Carrying capacity necessary to maintain desired amount of the original variation over the time:	238

Table 1. An example of population viability analysis.

reduce the rate at which it loses genetic variation. Maximising the rate of growth will therefore minimise loss of diversity during the growth phase.

It must be emphasised that there is no single or magic number that represents an MVP for all species at any time or for any species all of the time (Soulé, 1987). The MVP size will vary with the circumstances of the programme. Computer software is available to perform the population viability analysis (PVA) that will prescribe MVPs required for various circumstances; some examples of such analysis are presented in Table 1.

A secondary consideration for determination of the MVP is demographic stochasticity which is significant if the MVP prescribed by genetic considerations is fewer than 50–100. Populations smaller than 50 and possibly even 100 may be particularly vulnerable to 'crashes' or extinctions due to random demographic causes such as epidemic diseases, natural disasters or sex ratio distortions (Shaffer, 1987; Soulé, 1987).

The MVP establishes the lower limit for the carrying capacity of a managed captive population. It should be evident from the preceding discussion that it is advantageous for a population to be larger than the MVP size; indeed, in this respect, more is always better. Enlarging any one captive population, however, might well exclude other taxa from the zoo ark and therefore there has to be an upper limit on the carrying capacity for any one taxon in captivity.

The upper limit can be established by:

1. Assessing how much captive habitat (space and resources) is available for taxa with similar captive ecologies (i.e. forms that have equivalent enclosure requirements, resemble each other in terms of public expectation of what should be in a zoo, etc.). A crude measure of the captive habitat available is provided by the number of 'ecologically similar' specimens currently being maintained (Foose & Seal, 1986).
2. Ascertaining how many taxa with similar captive ecologies are in need of assistance by propagation in captivity. In this regard information from the IUCN SSC Specialist Groups will be important. The CBSG is already trying to develop recommendations for captive priorities among several broad groups, including psittacines and primates (Oates, 1985).
3. Allocating the captive habitat to as many taxa as possible while still maintaining an acceptable MVP for each.

Table 2 illustrates an attempt to apply this type of analysis to one group of animals, the large felids. As is the case for every broad category examined in this way so far, there is not enough captive habitat to accommodate acceptable MVPs for all the taxa that will need assistance to survive. This severe limitation of captive habitat argues

SPECIES	EXTANT SPP		SPP IN RDB		CAPTIVE POPULATION		NO. OF SPP IF POPULATION 100		250		500	
Panthera leo	11		1		1079		10		4		2	
Panthera tigris	8		8		1429		14		6		3	
TOTAL lions, tigers		19		9		2508		25		10		5
Panthera onca	8		8		179		2					
Panthera pardus	15		15		503		5		2		1	
Panthera uncia	1		1		312		3		1			
Felis concolor	29		2		280		3		1			
Neofelis nebulosa	4		4		202		2		1			
Acinonyx jubatus	6		6		454		4		2		1	
TOTAL other large felids		63		36		1930		19		8		4
TOTAL		82		45		4438		44		18		9

Table 2. Capacity of captive facilities for the larger felids calculated from data in ISIS and the appropriate studbooks. (An example of analysis to determine capacity of zoos for taxa with similar captive ecologies.)

strongly for the participation of as much of the zoo world as possible in the population management programme and for international co-ordination of the regional efforts.

Once the carrying capacity is established the institution-by-institution and animal-by-animal recommendations must be formulated. Based on genetic and demographic guidelines or criteria for management, these recommendations form the basis of the SSP-type Masterplan. Appendices 1 and 2 describe the kinds of genetic and demographic analysis and models which are needed for population management. Generally the objectives will be to develop a genetically diverse and demographically stable population.

Genetic management objectives will normally be to: (1) adjust the representation of founder lineages to rectify past disparities, that is, there will be an attempt to adjust the existing founder distribution in the population to the target founder distribution that has been established for the population; (2) regulate family sizes and sex ratios to maximise effective size of the population. Until the disparities in founder representation are rectified the management programme will deliberately reproduce from some animals more than from others. When these adjustments are completed, the objective will be for every animal to produce the same number of offspring. An exception to this guideline would be if there were a deliberate decision to have an unequal sex ratio for gregarious species when one family size objective will apply to ♀♀ and another to ♂♂. At carrying capacity each animal during its lifetime will be expected to produce on average two offspring, preferably one of each sex, which in turn survive to reproduce; (3) manage inbreeding coefficients to ensure that survival and fertility are not declining significantly.

Demography management objectives will normally be to: (1) expand the population from its founder or initial size to the carrying capacity as rapidly as possible within the constraints of the genetic guidelines, that is, regulation of family sizes and adjustment of founder representation. In cases of very small populations, demographic considerations will usually override genetic ones (Seal, in press); (2) stabilise the population at the carrying capacity by some combination of regulation of fertility (birth control) and survival (removal or culling). Programmes for regulation of fertility and

survival are based on analysis of life table data.

The institution-by-institution and animal-by-animal recommendations should specify which animals should reproduce when and with which mate to achieve the genetic and demographic objectives. These specifications will normally entail some relocation of animals between institutions to produce better genetic and demographic combinations of mates.

The Masterplan can then also determine what the genetic and demographic expectations are for each individual in the population. Once an individual has fulfilled what is expected or required of it in demographic and genetic terms, it becomes 'surplus' to the management programme. When this occurs, the individual should not reproduce again in or for the managed population.

Finally, it must be stated that genetic and demographic analysis are possible only if adequate data are available. Compilation of such data is the purpose of studbooks and the mission of ISIS and of the regional inventory systems around the world, all of which are thus vital to the conservation programmes of zoos.

APPENDIX 1

BASIC POPULATION GENETICS FOR CAPTIVE MANAGEMENT

Genetic diversity The many thousands of genes carried by an animal, interacting with the environment, ultimately control the structure and function of the organism. The site of a gene on a chromosome or elsewhere in the cell is known as its locus and the terms gene and locus tend to be used interchangeably. Almost all animals are diploid, carrying two copies of each gene which can occur in alternative forms or alleles. Different alleles may produce totally different effects in the organism. Where more than one allele exists there is genetic diversity. The genes of an individual are known as its genome. Collectively all the alleles for all the genes of all the individuals of a population or species constitute its gene pool.

Genetic diversity can occur at both the level of the individual and of the population (Fig. 2). If the two copies of a gene carried by an individual represent different alleles, the individual has genetic diversity and is known as heterozygous. If both copies are the same allele, there is no

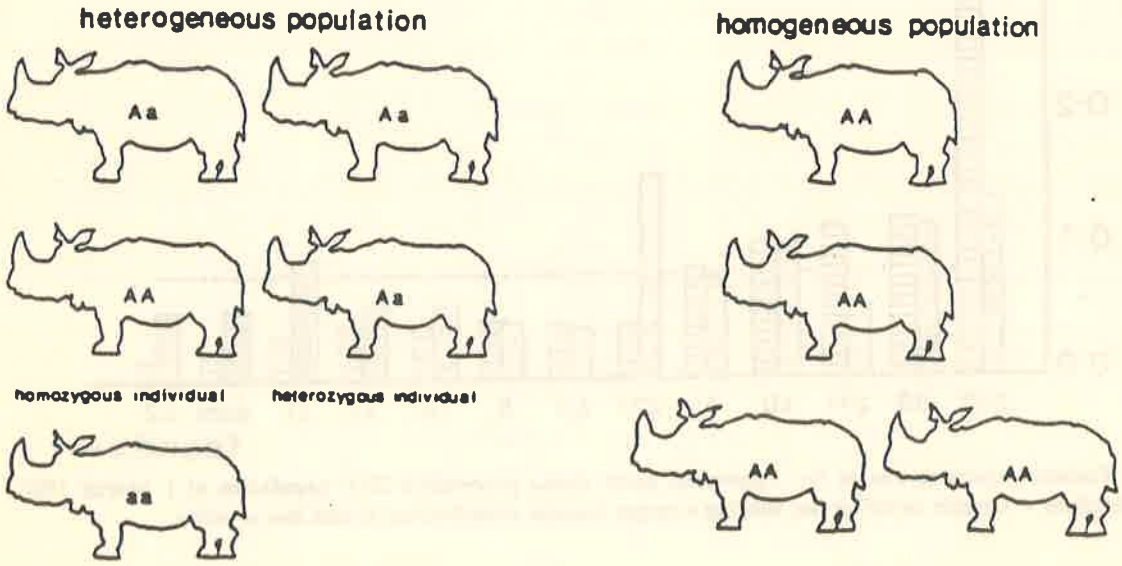


Fig. 2. Genetic diversity at both the individual and population level.

diversity and the individual is known as homozygous. Populations containing genetic diversity will have some heterozygous and some homozygous individuals. The terms heterogenous or polymorphic are sometimes used to describe a population with genetic diversity; homogenous is the analogous term for a population without diversity. At population level genetic diversity can be measured as allelic diversity or as average heterozygosity.

Genetic diversity is important for both individuals and populations. In populations it is needed in order to adapt to changing environments. For individuals heterozygosity is important for maintaining fitness, that is, the ability to survive and reproduce (Allendorf, 1986; Hedrick *et al.*, 1986).

The gene pools of a captive population can best be visualised through the genetic lineages or bloodlines that descend from the founder animals, that is, the animals from the wild that reproduce to have descendants in the living population. For captive populations, the original gene pool consists of all the genes that are carried by the founders. Preserving genetic diversity is thus tantamount to preserving as many as possible of these founder genes in the population for as long as possible.

Genetic drift In small populations, alleles may be lost entirely from the gene pool through genetic drift, that is, the random or stochastic process which results when a limited and therefore incomplete sample of genes from one generation is selected for transmission to the next. In other words, reproduction constitutes a random draw of some of the alleles in the population for perpetuation in the offspring; alleles which are not passed on to any of the offspring are lost to genetic drift.

Founder representation An animal transmits a copy of only one of each pair of genes to its offspring which thus receives half of its genes from its sire and half from its dam. Thus by constructing an individual's pedigree back to its founders it is possible to determine what founder genes the living individual

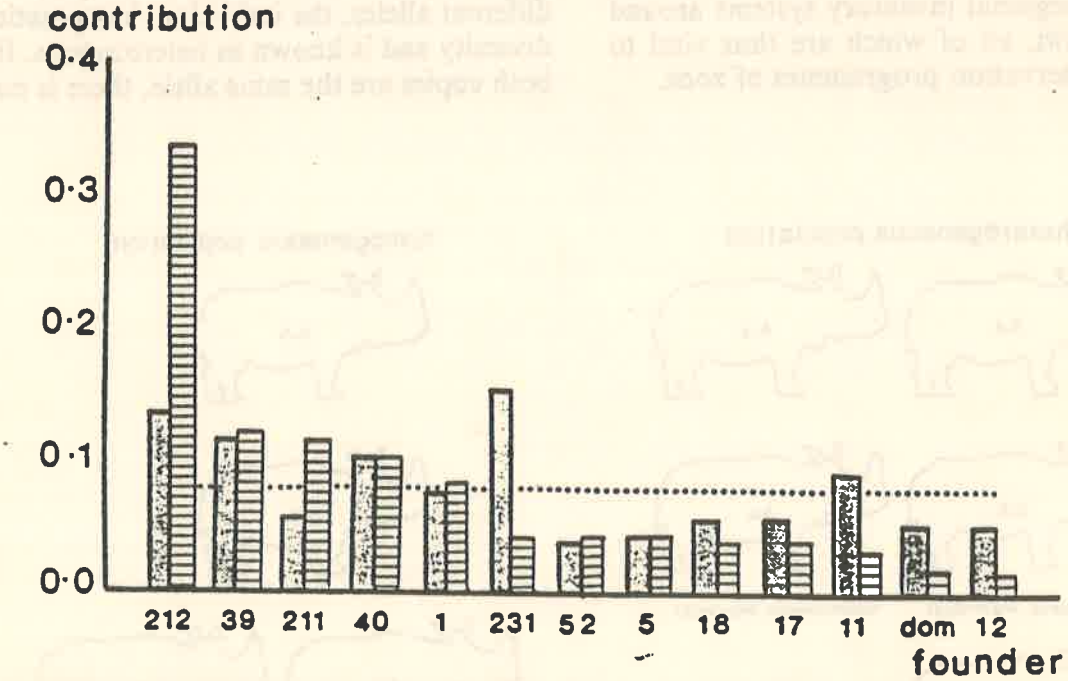


Fig. 3. Founder representation in the Przewalski horse *Equus przewalskii* SSP population at 1 March 1988. Cross-hatching = founder contribution; shading = target founder contribution; dotted line = parity.

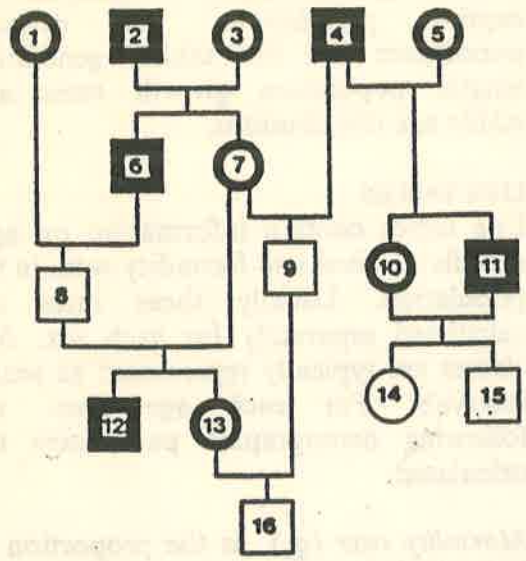


Fig. 4. An example of bottlenecks in a pedigree. Circles represent ♀♀, squares ♂♂; the black borders show deceased individuals; numbers 1–5 represent wild-bred founders; numbers 6–16 are their captive-bred descendants.

actually carries, that is, its founder representation. Summation of the founder representation over all founders provides the distribution of each founder genome in the population (Fig. 3). If the situation were this simple, maximising representation of genetic diversity over time would be a matter of equalising founder representation but the process is more complicated. Simple founder representation does not acknowledge that each founder actually carried two copies of each gene, that is, the founders are diploid not haploid. (For simplicity these two copies are referred to as two alleles in each founder even though they may not be different forms of the gene. Thus it is conventional to discuss founder alleles, not founder copies of genes.)

Because only one copy of each gene is transmitted from parent to offspring and because which of the two alleles carried by a parent is actually transmitted is a matter of chance, problems can be caused when a line of descent passes through very few individuals or perhaps even one animal. Such bottlenecks may prevent the perpetuation of one of the two founder alleles from that point onwards. As a result only part of the genome of some founders may actually survive in the living population while all the alleles of other founders may still be present. In the extreme example of a bottleneck of one in the F_1 generation from a founder, at most only one allele from each locus, or 50% of the genome if all loci are considered, could actually be transmitted to future generations (Fig. 4).

Genetic management In order to maximise preservation of genetic diversity (i.e. maintain as many of the founder's alleles as possible), genetic management should attempt to develop representation of founder lineages that are proportional to the percentage of the founder genomes that still survive. Again, referring to the extreme example of a bottleneck of one, the ideal representation of this founder in the population should be half as much as for founders whose entire genomes survive.

It is possible from computer algorithms, such as gene drop programs, to estimate the probable loss of founder genes through pedigrees (MacCluer *et al.*, 1986). For any living population it is then possible to compute a distribution of the proportion of each founder's genome that survives. Based on this distribution, a target or desired distribution of founder representations can be calculated. Matings that will adjust the existing founder lineage representations towards the target distribution can then be recommended.

Loss of alleles will obviously reduce the diversity of the population as a whole. Once the alleles have disappeared they can be recovered only through the very slow process of mutation or through migration from another population (if there is one). Attempts to prevent the loss of alleles from the population therefore have priority in genetic management.

The way in which alleles are arranged in individuals is also important. In small populations, animals are more likely to breed with relatives resulting in increased

levels of inbreeding. Inbred organisms have higher levels of homozygosity since there is a greater probability that an identical copy of an allele at any one locus will be received from its sire and dam. These alleles are said to be identical by descent. Thus the alleles that do remain in the population tend to become organised into more homozygous than heterozygous individuals. Since increased levels of homozygosity can expose potentially deleterious recessive alleles, the result can be so-called inbreeding depression, that is, a reduction in the animal's ability to survive and reproduce compared with that of the non-inbred animal. Inbreeding depression has been demonstrated in many captive populations (Ralls & Ballou, 1983) and management must therefore consider inbreeding levels in captive populations. Preventing loss of alleles and avoiding inbreeding are not always equivalent; they may even be in conflict. In most such cases the prevention of loss of alleles will have the higher priority unless inbreeding depression is severe enough to endanger the continued survival of the population (Templeton & Read, 1984).

Recommended reading on genetic management and conservation biology includes Soulé & Wilcox, 1980; Frankel & Soulé, 1981; Foose, 1983; Allendorf & Leary, 1986; Ralls & Ballou, 1986.

APPENDIX 2

BASIC DEMOGRAPHY FOR CAPTIVE MANAGEMENT

Population management requires the integration of both genetic and demographic analyses. For example, generation length, which is calculated from survival and fecundity rates, is essential for estimates of MVP size requirements while age distribution, sex ratios and reproductive rates are used to calculate effective population sizes.

This Appendix is essentially a basic demographic primer covering some of the fundamental concepts necessary for conducting demographic analyses of captive populations. Of primary importance are life tables, generation length, population growth rates and stable age distributions.

LIFE TABLES

Life tables contain information on age-specific survival and fecundity rates in the population. Usually these rates are calculated separately for each sex. Age classes are typically represented as yearly intervals. For each age class, the following demographic parameters are calculated:

Mortality rate (q_x) is the proportion of individuals that die during age class x. It is calculated from the number of animals that die during an age class divided by the number of animals that were alive at the beginning of the age class (i.e. the number of animals 'at risk' during the age class) (Table 3). Individuals still alive in the age class are not included in the q_x calculation because they have not yet lived through the entire age class.

Age-specific survival rate (p_x) is the proportion of individuals surviving from the beginning of the age class (x) to the beginning of the next age class ($x+1$). It is simply $1-q_x$ (Table 3).

Age-specific survivorship (l_x) is the proportion of individuals surviving from birth to the beginning of the age class. The l_x for the first age class (denoted age class 0 because it includes animals aged 0–1 years) is 1·00; 100% of the individuals survive to the beginning of age class 0. The l_x values are most simply calculated from the p_x values and are the product of all p_x values from age class 0 up to, but not including, the age class for which the l_x is being calculated (Table 3).

Fecundity rate (m_x) is the average number of same-sexed young born to (or sired by) animals in that age class. For example, in a life table for ♀♀ the m_x

CLASS	NO. OF ♀♀	NO. OF DEATHS	NO. OF ♀ BIRTHS	MORTALITY RATE q_x	AGE-SPECIFIC SURVIVAL RATE p_x	AGE-SPECIFIC SURVIVORSHIP RATE l_x	FECUNDITY RATE m_x	$l_x m_x$
0–1	100	40	0	0·4	0·6	1·0	0	0
1–2	60	10	36	0·17	0·83	0·6	0·6	0·36
2–3	50	30	75	0·6	0·4	0·5	1·5	0·75
3–4	20	20	10	1·0	0	0·2	0·5	0·1
4–5	0	0	0			0	0	0

Net Reproductive Rate (R_o) = 1·21

Table 3. Calculation of ♀ life-table data from information on ♀ births and deaths in a hypothetical population.

would refer to the average number of ♀ offspring born to ♀♀ in age class x. The m_x values are calculated by dividing the number of ♀ (or ♂) births by the number of ♀♀ (or ♂♂) alive at the beginning of an age class (Table 3). The fecundity rates provide information on the age of first and last reproduction, and ages of maximum reproduction.

Table 3 shows a simple life table for a population consisting of five age classes, aged 0–5. Values of q_x, p_x, l_x and m_x are calculated for each age class from data on numbers of deaths and births in a hypothetical population. Values of l_x and m_x can be plotted to illustrate graphically the survivorship and reproductive rates in the population (Figs 5 and 6).

These data are used to calculate values of generation time, population growth rate and stable age structure for the population.

GENERATION LENGTH

Conceptually, the generation length is the age at which an individual 'replaces' itself in the population. Technically, it is defined as the average age at which a ♀ (or ♂) produces offspring (Caughley. 1977). If data were available on the age of each parent when it produced young, the average of these ages would be the generation length of the population. Generation length is often incorrectly thought of as the age of first reproduction; however, this underestimates generation lengths in most cases. Males and ♀♀ often have different generation lengths.

The generation length can also be calculated direct from the l_x and m_x life table data. The l_x values provide the proportion of animals surviving to each age class while the m_x values provide how many young these survivors produce. The product of $l_x m_x$ is therefore the

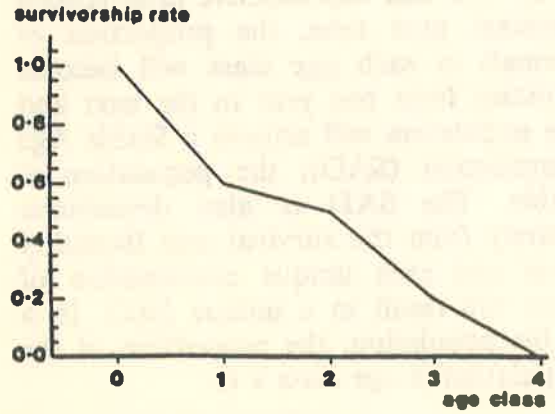


Fig. 5. Age-specific survivorship, l_x.

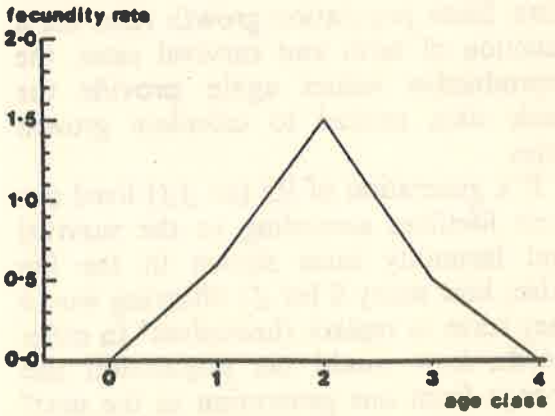


Fig. 6. Age-specific fecundity, m_x.

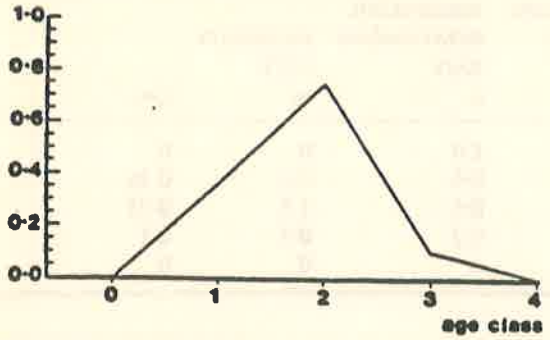


Fig. 7. The product of $l_x m_x$.

contribution of age class x to total reproduction. Figure 7 shows the distribution of $l_x m_x$ values for the hypothetical population in Table 3. The generation length is the mean age of this distribution and is calculated by:

$$\text{Generation Length (T)} = \frac{\sum x l_x m_x}{\sum l_x m_x}$$

Therefore, the generation length is the weighted average of reproductive age classes where the weights are the $l_x m_x$ values.

$$\text{For the example: } T = \frac{2{\cdot}16}{1{\cdot}21} = 1{\cdot}79$$

which corresponds with the apparent average in Fig. 6.

POPULATION GROWTH RATES

The life table data provide accurate estimates of the future growth rate of the population only if survival and reproductive rates remain constant over time. Since population growth rates are a function of birth and survival rates, the reproductive values again provide the basic data needed to calculate growth rates.

If a generation of ♀♀ (or ♂♂) lived out their lifetimes according to the survival and fecundity rates shown in the life table, how many ♀ (or ♂) offspring would they leave to replace themselves? In other words, how would the population size change from one generation to the next? The sum of $l_x m_x$ values over all ages is the average number of ♀ (or ♂) offspring surviving to replace each individual and therefore provides an estimate of the population's growth rate per generation. The sum of $l_x m_x$ values in the example is 1·21 (Table 3). Therefore, on the average, each ♀ leaves 1·21 ♀ young in the next generation and the population grows at 21% per generation. This sum is called the net reproductive rate (R_o).

If each ♀ were only to replace herself each generation, the net reproductive rate would be 1·00 and the population would remain the same size. The R_o in growing populations is greater than one; declining populations have an R_o of less than one.

A more useful measure is the growth rate per year (λ). This is calculated direct from the growth rate per generation:

$$\lambda = R_o^{(1/T)}$$

For the example, with R_o at 1·21 and T at 1·8, the yearly growth rate is :

$$\lambda = 1{\cdot}21^{(1/1{\cdot}8)} 1{\cdot}11, \text{ or } 11\% \text{ per year.}$$

Another commonly used measure of yearly growth rate is the intrinsic rate of increase, or r. It is the exponential yearly growth rate of the population and is related to λ in the following way:

$$r = \log_e(\lambda).$$

For the example:

$$r = \log_e(1{\cdot}11) = 0{\cdot}10.$$

STABLE AGE DISTRIBUTION

If survival and reproductive rates remain constant over time, the proportion of animals in each age class will become constant from one year to the next and the population will achieve a Stable Age Distribution (SAD); the population is stable. The SAD is also determined entirely from the survival and fecundity rates and each unique combination of rates will result in a unique SAD. In a stable population, the proportion of the population in age class x is:

$$C_x = \frac{\lambda^{-x} l_x}{\sum \lambda^{-i} l_i}$$

AGE CLASS	$l_x m_x$	C_x
0–1	1·0	48%
1–2	0·54	26%
2–3	0·41	20%
3–4	0·15	7%
4–5	0	0%
Total =	2·1	101%

Table 4. ♀ stable age distribution (C_x) calculated from life table values shown in Table 3 and a yearly growth rate of 11%.

SAD for the example is shown in Table 4. Since survival and reproductive rates can differ between sexes, so can their SAD.

OTHER APPLICATIONS AND POPULATION MANAGEMENT

Several other demographic parameters can be calculated from life-table data. One demographic question of particular interest to population management is how to manage reproductive and survival rates to achieve zero population growth (i.e. λ = 1·00) for a population at carrying capacity.

One simple option is to examine the effect of delaying age of first reproduction to a later age class. In the example, if age of first reproduction were delayed to age class 2, the R_o would be reduced to 0·85 (the sum of the $l_x m_x$ values for ages 2, 3 and 4). This would reduce the generation growth rate to below self-sustainment; the population would be reduced by 15% per generation; the generation length would be extended to 2·1 years and the per year growth rate would be 0·92 (8% reduction per year). Clearly this is too much of a reduction in reproduction. However, if the age of first reproduction were delayed for only 50% of the individuals, the m_x value in the example for age class 1 would be 0·3, the R_o reduced to 1·03, the generation length increased to 1·9 years, and the yearly growth rate reduced to 1·02 (a 2% increase per year). This is obviously a more appropriate alternative than delaying age of first reproduction for all individuals.

Similar types of manipulation are possible for estimating survival rates necessary for zero population growth. Rather than guess what levels of reproduction or survival rates will achieve λ of 1·00, however, methods for calculating exact m_x and l_x values are available (Goodman 1980).

These basic demographic concepts summarise some of the primary calculations used for demographic management. When analysing real populations, however, modifications of basic methods are often necessary. For example, calculations of generation length need to be modified for populations with largely overlapping generations and life tables might be calculated differently for seasonal versus aseasonal breeders (Caughley, 1977). Several computer programs are currently available for calculating and modeling demographic data for captive populations (Ballou & Bingaman, 1986; Bingaman & Ballou, 1986; Rockwell & Teare, 1986; Flesness & Scobie, 1987). Most of these require studbook data in pre-specified format (ISIS ARKS format or Omaha Studbook format). Methods for statistical analyses of life-table data are included and discussed in Lee (1980).

Further useful references include: Caughley, 1977; Keyfitz, 1968; Goodman, 1980; Mertz, 1970; Beddington & Taylor, 1973; Foose, 1983.

APPENDIX 3

THE AAZPA SSP PROTOCOL FOR DEVELOPMENT OF A POPULATION MANAGEMENT MASTERPLAN

DATA COMPILATION

The first step in the development of an SSP Masterplan is to compile the basic data required for population analysis. This compilation will often be in the form of a studbook. However, ISIS should be involved in the compilation process: initially as a source of some of the data for studbook development; ultimately as

a repository of the assembled data. An important part of the compilation process is a 'clean up' of the ISIS data.

The basic data required on each animal for population analysis and management are: (a) individual identification (a simple numeric lifetime identity); (b) sex; (c) birth date; (d) death date; (e) parentage (if captive born); (f) place of capture (if wild caught); (g) institutions/facilities where it has been held, with dates; (To achieve this identification, it may be necessary to link a series of different ID numbers the animal has had as it moved from one institution to another in its captive history, e.g. the local ISIS specimen ID numbers.) (h) available information on circumstances of death.

With these data, genetic and demographic analyses can be performed.

GENETIC ANALYSES

(a) construct the pedigree for each animal in the population; (This process may be the construction of a pedigree chart; more often it will be an inherent part of various algorithms and computer programs, e.g. the additive relationship matrix or various 'gene drop' computations.) (b) identify all the founders of the population; (A founder is an animal which is from outside the population, usually the wild, and which has no known relationship to any other individual at its time of entry into the population and has descendants in the living population.) (c) compute the representation of founders ('bloodlines'), or preferably the probable distribution of founder alleles, in living individuals and the present population as a whole; (d) locate any extreme bottlenecks in the history of particular founder lineages or bloodlines and compute the proportion of each founder's genome that has survived to the living population (see Appendix 1); (This step may be an inherent part of more sophisticated algorithms that calculate probable distributions of founder alleles rather than just crude founder representation.) (e) calculate the founder representation or founder allele distribution in offspring of the possible matings of living members of the population; (f) determine the number and sex ratio of animals that actually reproduce in the population; (g) calculate the number of offspring of each living individual in the population and hence the mean and variance of lifetime family sizes; (h) estimate the genetically effective population size (N_e) of the population and then the N_e/N ratio, where N is the total number of animals in the population; (i) calculate the inbreeding coefficients of existing individuals in the population and of the potential offspring of possible matings between these animals; (j) conduct various biochemical analyses as needed that measure genetic variability, genetic distance and identity (e.g. electrophoretic, DNA and karyotypic studies).

DEMOGRAPHIC ANALYSES

(a) determine the size of the current population and the number of institutions over which it is distributed; (Usually it will also be necessary to obtain the same kind of information for other taxa with similar 'captive ecologies', that is, space and resource requirements, but in less detail, for example the Siberian tiger SSP needs to be cognisant of the other tiger and large felid populations.) (b) determine the age and sex structure of the population; (c) compute the age-specific survivorships and fertilities of the population, that is, construct a life table (see Appendix 2); (d) establish a carrying capacity that is a compromise between a minimum viable population (MVP) for genetic and demographic viability and a maximum number that will not preclude other taxa from the zoo ark; (This carrying capacity should be based on the programme's goals as well as the biological characteristics of the population and should specify the number not only of animals but of the facilities over which they should best be distributed. In the absence of more

refined or species-specific recommendations on the long-term genetic objectives, the guideline of maintaining 90% of the founders' heterozygosity for 200 years may be used as a crude starting point.) (e) using the survival and reproductive rates from (c), calculate: the rate of change, that is, the growth or decline, of the population; the capacity of the population for self-sustainment; whether the population is at, or when it will be at, the carrying capacity; how the fertilities and survivorships can be managed by 'removals' of animals and regulation of reproduction (birth control) to stabilise the population at the desired carrying capacity; (This process may entail much 'what if..?' analysis to determine how management's modifications to the patterns of survivorship and fertilities will affect population size, growth rate, age distribution, etc.) (f) if survivorships and fertilities are not adequate for the population to be self-sustaining, devise appropriate research and husbandry programmes to resolve the problems.

POPULATION MANAGEMENT

Once genetic and demographic analyses are performed, an SSP Masterplan for propagation and management of the population can be formulated. The SSP Masterplan should provide institution-by-institution and animal-by-animal recommendations for every individual in the population maintained by SSP participants. Specifically, the Masterplan should: (a) designate which animals are surplus because they are: from over-represented bloodlines or lineages, too old to reproduce, have already produced their share of offspring and have attained the oldest age class necessary or allowable for a stable age distribution in the SSP population; (b) state explicitly that surplus animals should not be allowed to reproduce again; (Further recommendations on disposal of surplus will vary from programme to programme, time to time and institution to institution. In this connection the issue of euthanasia will have to be confronted.) (c) recommend which animals should reproduce, when (a schedule over at least the next one to five years is needed) and with which mate (identify specific individuals and any recommended shipments of animals); (d) explain the genetic and demographic analyses and objectives on which the surplus and reproduction recommendations are based; (There should also be an explanation of how the Masterplan arrived at the particular carrying capacity established.)

Normally, these genetic and demographic guidelines will include: an attempt to expand rapidly and stabilise the population at its established carrying capacity and a strategy to maximise preservation of genetic diversity Currently, the best methods to achieve these objectives seem to be to: (1) adjust representation of founder lineages to be proportional to the probable distribution of alleles surviving from founders at the initiation of the programme; (2) equalise lifetime family sizes; (This process will become fully operative only when the past inequalities in founder representation have been corrected.) (3) manage inbreeding coefficients; (4) perhaps subdivide the population into several parts or demes between which gene flow (i.e. usually exchange of animals but also increasingly of gametes or embryos) is regulated.

HUSBANDRY STANDARDS

Husbandry standards for the taxon should be developed, culminating in a *Handbook* which can be kept current as new advances occur.

REVIEW AND RATIFICATION

Once the SSP Masterplan is formulated, it should be reviewed, revised if appropriate and ratified by the AAZPA Propagation Group. The Masterplan should then be submitted to the SSP Subcommittee for their evaluation, recommendations and endorsement.

IMPLEMENTATION

Once approved, the Masterplan should be distributed to each of the participating institutions through its institutional representatives. The Species Co-ordinators and Propagation Group should provide follow-up to encourage and facilitate implementation.

REFERENCES

ALLENDORF, F. (1986): Genetic drift and the loss of alleles versus heterozygosity. *Zoo Biol.* 5: 181–190.

ALLENDORF, F. W. & LEARY, R. F. (1986): Heterozygosity and fitness in natural populations of animals. In *Conservation biology: the science of scarcity and diversity*: 57–76 Soulé, M. E. (Ed.). Sunderland, MA: Sinauer Associates.

BALLOU, J.D. & BINGAMAN, L. (1986): *PEDDEMO: dBase III life-table generator*. Washington, DC: National Zoological Park.

BEDDINGTON, J. R. & TAYLOR, D. B. (1973): Optimum age specific harvesting of a population. *Biometrics* 29: 801–809.

BINGAMAN, L. & BALLOU, J. D. (1986): *DEMOGPHY: Lotus 1-2-3 spreadsheet demographic model*. Washington, D.C.: National Zoological Park.

CAUGHLEY, G. (1977): *Analysis of vertebrate populations*. New York: John Wiley & Sons.

DOBSON, A. P. & MAY, R. M. (1986): Disease and conservation. In *Conservation biology: the science of scarcity and diversity*. 345–365. Soulé, M. E. (Ed.). Sunderland, MA: Sinauer Associates.

FLESNESS, N. R. & SCOBIE, P. N. (1987): *User manual for ARKS Animal Records Keeping System*. Apple Valley, MN: International Species Inventory System (ISIS).

FOOSE, T. J. (1983): The relevance of captive propagation to the conservation of biotic diversity. In *Genetics and conservation*: 374–401. Schonewald-Cox, C. M., Chambers, S. M., MacBryde, B. & Thomas, L. (Eds). Menlo Park, CA: Benjamin/Cummings.

FOOSE, T. J. & SEAL, U. S. (1986): Species Survival Plans for large cats in North American zoos. In *Cats of the world*: 173–198. Miller, S. D. & Everett, D. D. (Eds). Washington, DC: National Wildlife Federation.

FRANKEL, O. H. & SOULÉ, M. E. (1981): *Conservation and evolution*. Cambridge: Cambridge University Press.

GILPIN, M. E. & SOULÉ, M. E. (1986): Minimum viable populations: processes of species extinction. In *Conservation biology: the science of scarcity and diversity*: 19–34. Soulé, M. E. (Ed.). Sunderland, MA: Sinauer Associates.

GOODMAN, D. (1980): Demographic intervention for closely managed populations. In *Conservation biology*: 171–195. Soulé, M. E. & Wilcox, B. A. (Eds). Sunderland, MA: Sinauer Associates.

HEDRICK, P. W., BRUSSARD, P. F., ALLENDORF, F. W., BEARDMORE, J. A. & ORZACK, S. (1986): Protein variation, fitness and captive propagation. *Zoo Biol.* 5: 91–99.

KEYFITZ, N. (1968): *Introduction to the mathematics of populations*. Reading, MA: Addison-Wesley.

LANDE, R. & BARROWCLOUGH, G. (1987): Effective population size, genetic variation, and their use in population management. In *Viable populations for conservation*: 87–124. Soulé, M. E. (Ed.). Cambridge: Cambridge University Press.

LEE, E. T. (1980): *Statistical methods for survival data analysis*. Belmont, CA: Lifetime Learning Publications.

MACCLUER, J. W., VANDEBERG, J. L., READ, B. & RYDER, O. A. (1986): Pedigree analysis by computer simulation. *Zoo Biol.* 5: 147–160.

MERTZ, D. B. (1970): Notes on methods used in life-history studies. In *Ecology and ecological genetics*: 4–17. Connell, J. H., Mertz, D. B. & Murdoch, W. W. (Eds). New York: Harper & Row.

OATES, J. F. (1985): *Action plan for African primate conservation: 1986–1990*. Washington, DC: WWF Primate Program and IUCN/SSC Primate Specialist Group.

RALLS, K. & BALLOU, J. D. (1983): Extinction: lessons from zoos. In *Genetics and conservation*: 164–184. Schonewald-Cox, C. M., Chambers, S. M. MacBryde, B. & Thomas, L. (Eds). Menlo Park, CA: Benjamin/Cummings.

RALLS, K. & BALLOU, J. D. (1986): Preface to the proceedings of the workshop on genetic management of captive populations. *Zoo Biol.* 5: 81–86.

ROCKWELL, R. & TEARE, A. (1986): *Studbook management system*. Omaha, NE: Henry Doorly Zoo.

SEAL, U. S. (In press). *Proceedings of workshop on reproductive biology of black-footed ferrets and small population biology as they relate to conservation*. New Haven, CN: Yale University Press.

SHAFFER, M. (1987): Minimum viable populations: coping with uncertainty. In *Viable populations for conservation*: 69–86. Soulé, M. E. (Ed.). Cambridge: Cambridge University Press.

SOULÉ, M. E. (Ed.) (1987): *Viable populations for conservation*. Cambridge: Cambridge University Press.

SOULÉ, M., GILPIN, M., CONWAY, W. & FOOSE, T. (1986): The millenium ark: how long a voyage, how many staterooms, how many passengers? *Zoo Biol.* 5: 101–113.

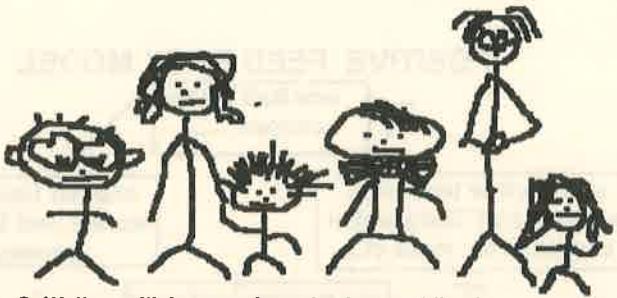
SOULÉ, M. E. & WILCOX, B. A. (1980): *Conservation biology*. Sunderland, MA: Sinauer Associates.

TEMPLETON, A. R. & READ, B. (1984): Factors eliminating inbreeding depression in a captive herd of Speke's gazelle. *Zoo Biol.* 3: 177–199.

Manuscript submitted 15 April 1988

A VERY, VERY BASIC INTRODUCTION TO SMALL POPULATIONS &

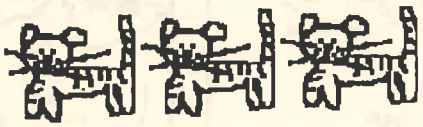
META-POPULATION MANAGEMENT for non-scientists



Written by S. Walker utilising a variety of written publications, lectures, and personal communications with Tom Foose, Ulie Seal, Colin Tudge and Malcolm Whitehead. (They should not be blamed, however.)

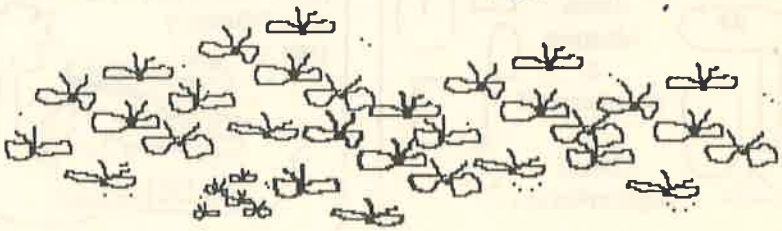
WHAT IS A "SMALL POPULATION" ?????

IS IT THIS ?
A small number of BIG things?



OR THIS?
A small number of SMALL things?

OR THIS?
A large number of SMALL things?



OR
None of the above
Um, No. Well, yes. Maybe. Some of those . . .

A
"small POPULATION"

IS A GROUP OF CREATURES
(OF THE SAME SPECIES)

THAT CANNOT

RETAIN
ENOUGH GENETIC DIVERSITY

OR

SUSTAIN
ENOUGH DUMB LUCK

TO SURVIVE
(and we GOT TO survive!)*

TO SURVIVE
INBREEDING
AND
RANDOM CATASTROPHES
(BAD THINGS)
OVER A

SHORT

SPAN
OF TIME

OR, TO

EVOLVE ADAPTATIONS
TO INSURE EVOLUTION
AND SURVIVAL
(e.g., ADJUST)

OVER A LOOOOOONG SPAN
OF TIME

A SMALL POPULATION

IS ONE THAT IS
VULNERABLE.

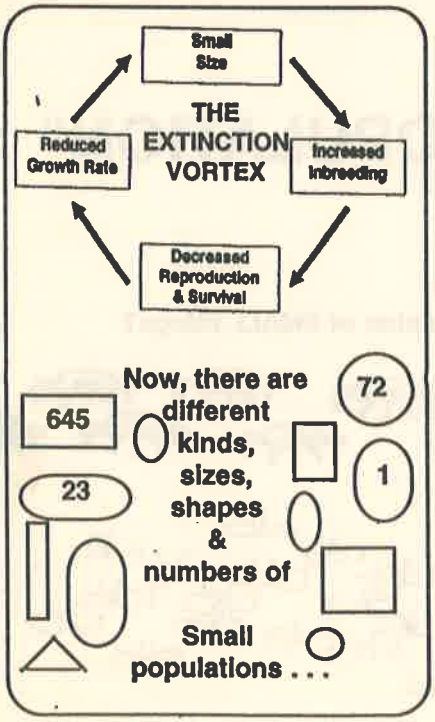
VULNERABLE TO ***RISK***

THAT IS, RISK OF *EXTINCTION*
(e.g., NO MORE
animals...forever)

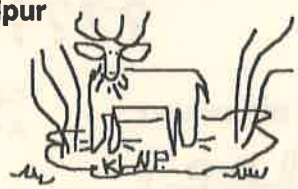
BY A SORT OF ATTRITION.
ATTRITION =
NEGATIVE FEEDBACK =
"ONE BAD THING
LEADS TO ANOTHER
AND MAKES IT EVEN WORSE!"
THE
EXTINCTION VORTEX

* Bob Marley, Kingston, 1987

POSITIVE FEED BACK MODEL



A SMALL POPULATION MAY
BE A SINGLE (SMALL)
POPULATION...e.g. Sangai
(76) IN ONE AREA ONLY
e.g. Keibul Lamjao N.P.,
Manipur



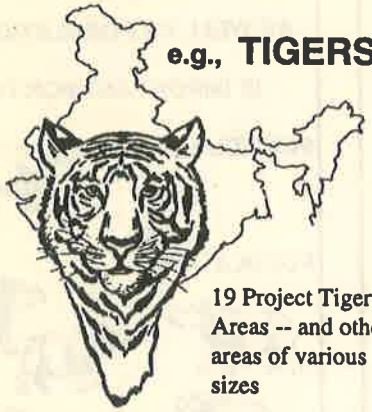
OR
ASIATIC LIONS --
(300 ONLY) IN THE
GIR FOREST
ONLY

OR A FEW (SMALL)
POPULATIONS IN A FEW
FRAGMENTED AREAS
e.g., LION-TAILED MACAQUES

IN
THREE
SOUTH
INDIAN
STATES

OR MANY
(SMALL)
POPULATIONS IN
MANY AREAS
WITH NO
NATURAL
CORRIDORS

e.g., TIGERS



19 Project Tiger Areas -- and other areas of various sizes

NOW, *USUALLY*

a "SMALL POPULATION"
consists of

a few 10's

a few 100's

or even a few 1000's

of individuals

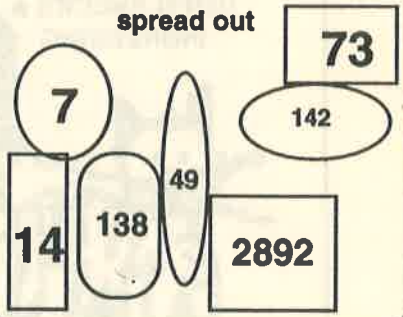
50 RHINOS
may be as
EFFECTIVE A
POPULATION AS
100 L.T.M.'S
OR 1000 FLAMINGOS
OR 1000000000 FROGS

because . . .The meaning of

"SMALL"
(or large, for that matter)

depends in part on

HOW
numbers in the population
are
spread out



...how they are spread out ... in small numbers,
in isolated, fragmented and otherwise
stressed-out habitats

WHY ?

Because of

Walker's Law

... huh?

A very small population could survive in the wild

IF

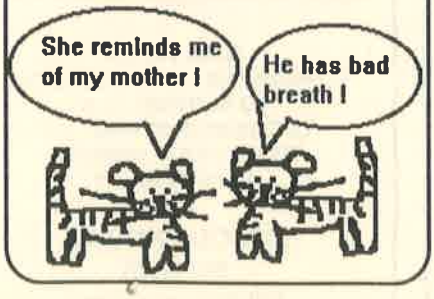
NOTHING WENT WRONK,

BUT according to

Walker's Law

IN LIFE
(AND IN NATURE)

THINGS GO WRONk



And what things can go wrong in Nature, pray tell?
WELL,

1. DEMOGRAPHIC THINGS
2. GENETIC THINGS and
3. ENVIRONMENTAL THINGS

1. DEMOGRAPHIC THINGS :

a. DISTORTED SEX RATIO
(i.e., a run of all male births ...

b. UNSTABLE AGE STRUCTURE
(i.e., too many kids and too many grandma's

c. REPRODUCTIVE FAILURES
(i.e., low romance factor?

2. GENETIC THINGS

a. Drift
b. Migration
c. Selection
d. Mutation

WHICH CAUSE . . .
LOSS OF GENETIC VARIATION
DUE TO
FOUNDER EFFECT &
BOTTLENECKS &
INBREEDING

3. ENVIRONMENTAL THINGS

Natural disasters
STORMS
FLOODS
FIRE
DROUGHT
PLAGUE
EARTHQUAKE
VOLCANIC ERUPTION

Human-caused disasters
DISEASE (domestic livestock)
SOCIAL (unrest)
POLITICAL (discontent)
ECONOMIC (instability)
EXOTIC (species introduction)*

. . . just to name a few

**** Plants and animals, e.g. Red fox, snails, trees, etc.***

Any one of these could wipe out a small population in short order

TO MEET SUCH CHALLENGES

GENETIC VARIATION,

AS WELL AS POPULATION SIZE

IS IMPORTANT FOR BOTH

INDIVIDUALS

AND
POPULATIONS

INDIVIDUALS

need <u>Genetic Variation</u> to be fit, .. healthy .. vigorous so that they can *<u>reproduce</u>*...

-- for survival

POPULATIONS

need <u>Genetic Variation</u> to be fit .. healthy .. vigorous so that they can *<u>adapt</u>* to the challenges of our changing world, or ...

-- to evolve.

Then, what to do?

How to save species and populations

In the circumstances of our modern world...

Population Explosion
Human pressure on land
Shrinking habitat
Fragmented habitat
Isolated populations

BE SURE YOU HAVE

ENOUGH

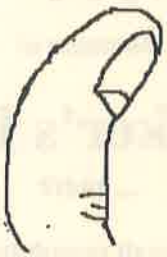
So that these problems DON'T MATTER

HOW MANY ARE ENOUGH?

TO BE FIT. . .?
TO INCREASE THE POPULATION?
TO SURVIVE . . . .?
TO TRANSCEND RISK . . . ?
TO PRESERVE GENETIC VARIATION . . .?

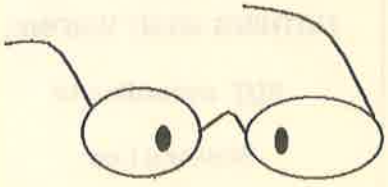
NOBODY KNOWS
(for sure)
but here are some

RULES OF THUMB



OR

VERY EDUCATED GUESSES



SURVIVAL IN CAPTIVITY	SURVIVAL IN THE WILD
Maintenance of sufficient genetic variation necessary to *MINIMISE INBREEDING.*	Maintenance of sufficient genetic variation necessary for *ADAPTIVE EVOLUTION*
Some population biologists suggest	Some population biologists suggest
50* *breeding adults*	500* *breeding adults*
or a census population of	or a census population of
250	2500

*** THESE NUMBERS ARE "RULES OF THUMB" NOT "HARD AND FAST" RULES**

SMALLER POPULATIONS HAVE SURVIVED, E.G.

Sangai -- from 14 to 87
Manipur
Asiatic lion -- 20 to 300
Gir Forest
Indian Rhino -- 16 to 1100Kaziranga

EVEN LARGER POPULATIONS HAVE GONE EXTINCT

Passenger pigeon
200,000,000 and odd to 0
U.S.A.

WHY DO POPULATION BIOLOGISTS SAY YOU NEED LESS TO INSURE SURVIVAL 50/250 IN CAPTIVITY THAN 500/2500 IN THE WILD ?

BECAUSE -- IN CAPTIVITY -- YOU HAVE

CONTROL

There are many advantages to captivity for small, "sick" populations

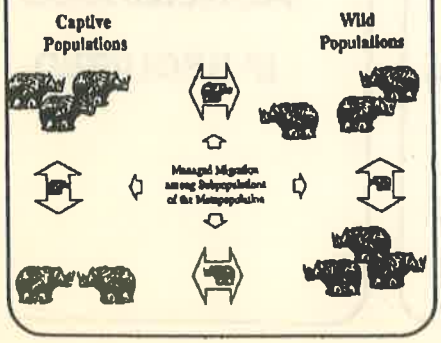
***WARNING ! These figures are RULES OF THUMB -- NOT the Golden Rule. They can vary greatly, depending on the life history of the species. When we say "50" or "500" breeding adults, we mean AT LEAST that many and possibly many,many more.**

HOW TO HAVE *ENOUGH?*

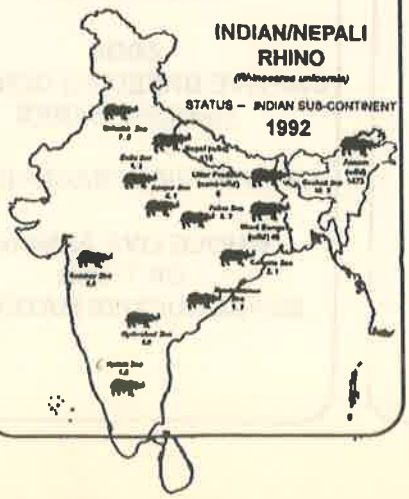
when so many things are going wrong in the wild and the world ?

METAPOPULATION MANAGEMENT IS ONE WAY

Metapopulation



WE MAKE USE OF THE TOTAL POPULATION CAPTIVE & WILD



USING A VARIETY OF THE "NEW ZOO SCIENCES" & MODERN MANAGEMENT TECHNIQUES IN WILD AND CAPTIVE POPULATIONS IN *COOPERATIVE MANAGEMENT PROGRAMMES* THROUGHOUT THE WORLD'S *ZOOS*

THAT'S WHY WE SAYS

ZOOS GIVE WILD ANIMALS WITH NO CHANCE

A *LAST* CHANCE

ZOOS ARE LIKE A

WHERE WE CAN KEEP A SMALL POPULATION SAFE AND INCREASE IT

IN CAPTIVITY You can:

...move animals more easily to readjust sex ratio and age distribution

...keep track of their identity-- age, sex, linage, etc.

...pair them according to genetic and demographic profile and the needs of the meta-population

...protect them from harm

...increase numbers faster

...promote them as Ambassadors for their Species

ADVANTAGES TO CAPTIVITY

There is
PROTECTION FROM POACHERS
LESS ENVIRONMENTAL PERTURBATIONS,
MORE GENETIC MANAGEMENT,
MORE DEMOGRAPHIC MANAGEMENT,
HEALTH/DISEASE MANAGEMENT,
SECURE EXPANSION OF POPULATION,
PUBLIC EDUCATION AND & SUPPORT,
RESEARCH USEFUL FOR CONSERVATION

ALL THIS, WITHOUT DISTURBING THE WILD ONES!

ZOOS ARE NOT A

SUBSTITUTE

FOR THE WILD

BUT THEY ARE

(-- OR CAN BE --)

A

SUPPORT

FOR THE WILD

SO FOR

INTERACTIVE MANAGEMENT

EX SITU <=>IN SITU
ZOO <=> WILD

ZOOS
CAPTIVE BREEDING CENTRES
GENOME BANKS

CAN PROVIDE "BACK-UP" OF

WHOLE LIVE ANIMALS
OR THEIR
REPRODUCTIVE MATERIAL

TO STRENGTHEN SMALL, WILD POPULATIONS

...

GENETICALLY
NUMERICALLY
DEMOGRAPHICALLY

IN SANCTUARIES,
NATIONAL PARKS,
RESERVE FORESTS, ...
PROTECTED AREAS

IF REQUIRED

ANOTHER METHOD IS INTENSIVE MANAGEMENT OF THE WILD POPULATION

* Marking/monitoring of individual animals

* Enhanced protection measures

* Habitat improvement

* Disease prevention

* Livestock control

* Relocation of human settlements

* Creating alternative populations in safer areas

* Translocation / reintroduction / introduction, etc. of live animals or their reproductive material

ACTIVE INTERVENTION can save species & populations

1. Adding animals or their reproductive material torestore

   a. numbers
   b. demographic stability
   c. genetic diversity

2. Translocating populations (or parts of populations) to establish new populations

3. Culling for habitat management

4. Initiating Alternative populations using stock from zoos or from other "too-small-to-save" populations in wild

5. Initiation of captive breeding programmes

WHAT DOES IT TAKE TO SAVE A small POPULATION)

COOPERATION, COORDINATION COMMUNICATION

e.g. , lots of people working together -- Action

RESOURCES RESEARCH RECORDS

e.g., lots of money, expertise and INFORMATION

High-tech reproductive techniques can put captive animals -- or their reproductive material -- ***BACK TO THE WILD***

Artificial insemination
Induction of estrus
Embryo transfer
In vitro fertilisation
Cryopreservation

COOPERATION, COORDINATION COMMUNICATION

from global to grass roots

SPECIES SURVIVAL COMMISSION

IUCN The World Conservation Union

IZDA INDIAN ZOO DIRECTORS' ASSOCIATION

EUROPÄISCHES EEP ERHALTUNGSZUCHTPROGRAMM

BRITISH AIRWAYS ASSISTING NATURE CONSERVATION

ZOO OUTREACH ORGANISATION

RESOURCES RESEARCH RECORDS

भारतीय वन्यजीव संस्थान
Wildlife Institute of India

CBSG INDIA

ASSOCIATION OF INDIAN ZOO AND WILDLIFE VETERINARIANS

BY COMBINING INFORMATION AND ACTION

IT IS POSSIBLE TO

RESCUE

A SMALL POPULATION

WITH A RECOVERY PROGRAMME

ONCE A
POPULATION
BECOMES A
SMALL **POPULATION,**
IT IS PROBABLY

HISTORY

...
UNLESS

WE INTERVENE

WITH A
RECOVERY
PROGRAMME

WHAT'S
SMALL POPULATIONS
GOT TO DO WITH
BIODIVERSITY

EVERYTHING!!!

MASS EXTINCTIONS OF
MANY, MINUTE
POPULATIONS IN
MULTIPLE AREAS OF
HIGH BIODIVERSITY
(SOME OF THEM WE DON'T
EVEN KNOW EXIST)
=
LOSS OF BIODIVERSITY

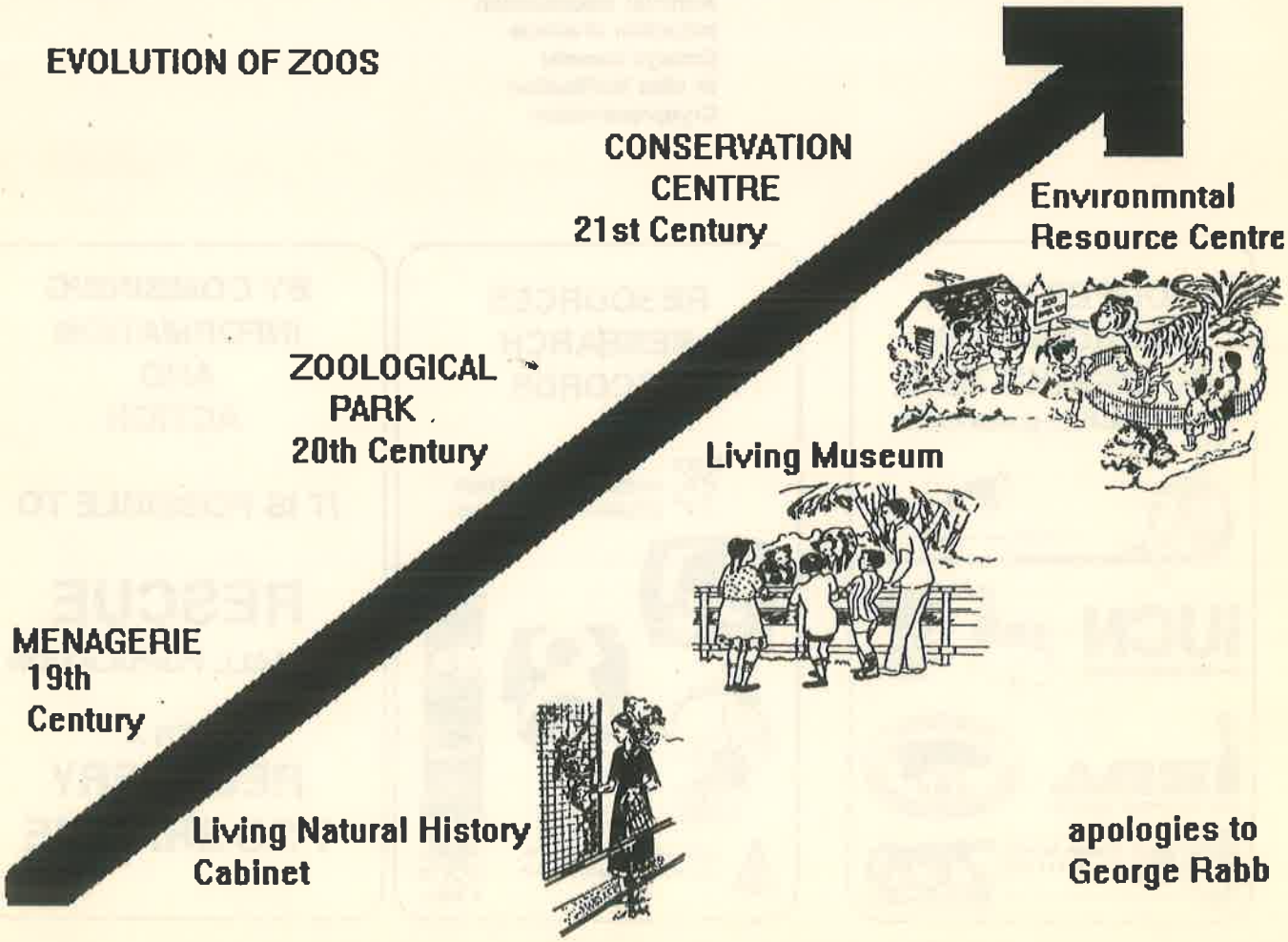
ZOOS AND
ZOO SCIENCES
WORKING WITH FIELD
MANAGERS IN AREAS
OF HIGH BIODIVERSITY
CAN REVERSE THE

EXTINCTION

VORTEX

FOR MANY
SPECIES

GOOD ZOOS CAN HELP CONSERVE BIODIVERSITY



IMPACTS OF INBREEDING IN NATURAL AND CAPTIVE POPULATIONS OF VERTEBRATES: IMPLICATIONS FOR CONSERVATION

*ROBERT C. LACY**

Many wildlife populations that were once large, widespread, and diverse have been reduced to small, isolated populations in a few remaining natural areas, nature preserves, and zoological parks. Once a population becomes very small and isolated from potential sources of immigrants, random demographic and genetic processes can lead the population rapidly toward extinction. Demographic problems faced by small, fragmented populations include difficulty in finding mates, random skewing of the sex ratio, and simply the misfortune of all animals dying or failing to breed in a given year due to chance, a disease outbreak, a temporary scarcity of food or an abundance of predators, or local weather conditions. These and other random demographic and environmental events can cause a small population to decline still further. Even if the causes of such a decline are temporary, the resulting very small population may be forced to inbreed. It has long been observed that inbreeding causes greater mortality and reduced fecundity in many species, the phenomenon called "inbreeding depression" [1–3]. Inbreeding and the loss of genetic diversity may lower fitness and reduce the potential for the population to adapt. This can lead to further decline, making the demographic problems worse, in turn making the genetic problems worse—a feedback that drives small populations ever faster toward extinction. This process has been termed the "extinction vortex" [4], and the size below which a population is likely to get drawn into the extinction vortex is one useful definition [e.g., 5] of the Minimum Viable Population size (or MVP) [6].

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Conservation of remnant wildlife populations requires knowledge of the minimum population size below which the combined effects of random genetic changes and demographic variation would likely result in extinction [4–11]. Estimating the MVP for a population will require considerable knowledge of the effects of inbreeding on endangered populations [12]. In particular, we need to know: (1) whether the inbreeding depression that is so prevalent among domesticated animal stocks is equally characteristic of most wild populations; (2) the severity of inbreeding depression (the function relating fitness traits to past and present inbreeding) in natural populations; (3) whether inbreeding depression is consistent and predictable; and (4) whether a depression in fitness traits can be avoided or reversed by combining inbreeding with selection to purge a population of the genes that cause inbreeding depression.

Causes of Inbreeding Depression

The common explanation for the hazards of inbreeding (inbreeding depression) rests on the presence of deleterious recessive genes, a "genetic load," in virtually all diploid organisms. Natural selection will reduce the frequencies of deleterious alleles in a population (or keep newly arisen harmful mutations from becoming common), but it is very inefficient at eliminating wholly a recessive allele because such alleles are almost always shielded from selection in heterozygotes. One consequence of inbreeding is that it makes it much more likely that an individual is homozygous for a rare gene because it is more likely that two related parents simultaneously possess a rare allele and transmit it to their inbred offspring than that two unrelated individuals independently transmit the same rare allele to a non-inbred offspring. Thus, inbreeding seems to reduce fitness because it reveals harmful genes in homozygotes.

If this explanation of inbreeding is correct, then we should be able to predict which populations would be most susceptible to inbreeding depression [12]. When inbreeding occurs, deleterious genes will be expressed in homozygotes and cause the death of individuals bearing them, but those genes are thereby eliminated from the population. Thus, populations with histories of inbreeding should now be largely purged of their genetic load of recessive deleterious genes. On the other hand, populations that have long been large and diverse may harbor many, individually rare, deleterious, recessive genes.

Thus, the black-footed ferret (*Mustela nigripes*), which probably existed in numbers no greater than 50 breeding adults during the past 50 years [13] and which was extinct in the wild [14] until reintroduction of captive-born ferrets began in 1991, may harbor few deleterious recessive

alleles and may be unaffected by the inbreeding that will necessarily occur as the small remnant population is propagated in captivity and restored to natural habitats. Similarly, endangered species that are endemic to small islands may suffer little depression in fitness when inbred. Species that declined to very low numbers only within the past generation or two, such as the California condor (*Gymnogyps californianus*) and black rhinoceros (*Diceros bicornis*), might still have large genetic loads and may be unable to survive even moderate inbreeding. The greater one-horned rhinoceros (*Rhinoceros unicornis*), which declined from thousands of animals prior to 1950 to fewer than 100 animals during the 1960s, retains high levels of genetic variation [15] and presumably still has a genetic load typical of nonendangered mammals.

An alternative explanation of inbreeding depression is that heterozygotes may be more adaptive than homozygotes for many or even most genetic loci. Lerner [16] proposed that such overdominance, or heterosis, may be a general phenomenon, with heterozygotes showing greater developmental stability and greater ability to tolerate environmental fluctuations and stresses. If such general heterosis is the primary cause of inbreeding depression, then we would expect fitness to decline monotonically as a population becomes inbred and loses heterozygosity. Little adaptation to inbreeding would occur because selection favoring heterozygotes maintains balanced polymorphisms and therefore retains, rather than removes, the genetic load. Only indirect selection on modifier loci to reduce the heterozygote advantage, perhaps by providing alternative biochemical pathways that make the heterotic locus superfluous, would effectively remove a genetic load resulting from heterosis. If general heterosis is the primary cause of inbreeding depression, then populations that have undergone prolonged bottlenecks might already have declined in fitness and might be the most vulnerable to further losses of genetic variation.

Clearly, whether inbreeding depression results primarily from deleterious recessive alleles or general heterosis is of considerable importance to conservation. The design of wildlife preserves, breeding programs, and recovery plans all require estimation of MVPs, which in turn depends upon estimates of the sensitivity of populations to inbreeding depression. We need to know which populations could survive the inbreeding that accompanies a severe bottleneck, and which would suffer irreversible decline in fitness if the population declined to numbers that forced matings between close relatives [12].

Inbreeding in Domesticated and Laboratory Animals

For hundreds of years, animal breeders have been aware of the problems associated with close inbreeding. Darwin [1] documented exten-

sively inbreeding depression in domesticated stocks, and Wright [2], Falconer [3], and others have reviewed data that have accumulated since. A very large body of data demonstrates that inbreeding depresses virtually every component of fitness that has been measured in each species of domesticated livestock. Fertility, birth weight, growth rate, survival, disease resistance, productivity (of meat, milk, eggs, wool, etc.) are all depressed when farm animals are inbred. Attempts to completely inbreed cattle, sheep, and other species have always ended in extinction of the stock, suggesting that it may not be possible to rid these populations entirely of their genetic loads. The failure to produce a viable, fully inbred stock of any domesticated livestock species is unexpected if inbreeding depression in livestock is due to fully recessive, deleterious genes. The lack of inbred strains of livestock suggests instead that heterozygote vigor at some loci or throughout much of the genome is essential to survival and/or reproduction.

Studies on inbreeding in laboratory rodents have yielded equivocal evidence on the nature of inbreeding depression. Although many inbred lines of mice and rats have been produced for biomedical research, few data were kept on the breeding records during the course of inbreeding [17]. What we know about the effects of inbreeding on those lines comes primarily from subsequent crosses between stocks to produce outbred mice [3]. More recently, researchers have inbred newly wild-caught house mice and documented the inbreeding depression that resulted [18, 19]. Many sublines went extinct during the production of the viable inbred lines, as would be expected if selection were removing deleterious alleles from the population during the course of inbreeding. None of the inbred lines of rodents, however, are as fit as are outbred stocks produced by crossing inbred lines or obtained from the wild. Thus, either some deleterious recessive alleles were fixed within each of the inbred lines, or fitness at heterotic loci was sacrificed during the production of the inbred lines. The data do not distinguish between causes of inbreeding depression.

Inbreeding in Captive Populations of Wild Animal Species

Domesticated animals and most laboratory stocks result from hundreds to thousands of years of intense artificial selection, and it is not clear that they are good models for natural populations. Zoological parks collectively breed thousands of vertebrate species, and either because of a lack of more suitable animals to pair or because of a lack of concern about the effects of inbreeding, zoos often inbreed their stocks. The following summary of the impacts of inbreeding on zoo populations is developed more fully by Lacy, Petric, and Warneke [20].

Scattered through the scientific literature are case studies of inbreed-

ing in species propagated by zoos and private breeders. For every species studied in detail, investigators report that inbreeding depresses at least some aspect of fitness in some populations, with infant mortality being the most commonly measured fitness component. However, different species and even different captive stocks of the same species often respond to inbreeding in divergent ways. For example, Shoemaker [21] found that inbreeding increased juvenile mortality in Persian leopards, but he reported no measurable effect on juvenile mortality in Chinese leopards or Amur leopards. Daniell and Murray [22] reported inbreeding to depress nestling survival in one colony of budgerigars, but not in another. Slatis [23] found higher mortality in inbred European bison, but only among the descendants of a male that was from a different subspecies than the rest of the remnant world population. Either this male harbored recessive lethal genes not found in the other bison, or the reported "inbreeding depression" was actually hybrid breakdown that occurred after hybridization between the subspecies.

Kathy Ralls and Jon Ballou of the National Zoological Park have compiled data on the effects of inbreeding on zoo populations (see [24] for a review). Examining 45 species of mammals bred in zoos, they showed that juvenile mortality was usually higher in inbred animals, but they did not quantify the severity of inbreeding depression in these zoo stocks. More recently, Ralls, Ballou, and Templeton [25] presented a more detailed study of the severity of inbreeding depression in 40 populations of mammals. They calculated the number of "lethal equivalents" per diploid individual, measured by regressing the logarithm of juvenile survival against the inbreeding coefficient. Lethal equivalents quantify the genetic load by estimating the number of recessive lethal alleles per individual if the observed inbreeding depression were due solely to lethal, fully recessive genes [26]. Ralls et al. [25] reported the effect of inbreeding depression on juvenile mortality to range from -1.36 lethal equivalents in maned wolves (*Chrysocyon brachyurus*) to 30.32 in Wied's red-nosed rat (*Wiedomys pyrrhorhinos*). (Although the negative value measured in maned wolves was not significantly less than zero, negative lethal equivalents would indicate greater survival in inbred litters than in noninbred offspring). The survey detected no clear trends among the mammalian orders. Nor did it demonstrate a difference between populations started from wild-caught animals and those started from stocks that had already been in captivity for one or more generations, which may have already had some of the genetic load removed by selection during previous episodes of inbreeding.

Recently, colleagues at the Chicago Zoological Park and I compiled and analyzed records for species of Artiodactyla with long-term breeding histories at the zoo [20]. When appropriate to the social system of the species, the Chicago Zoological Park maintains its hoofstock in herds.

Because of limited availability of unrelated stock, many herds were founded with only a few individuals, and often decades pass before unrelated animals are added to the herds. Thus, each population becomes progressively inbred through several generations, until a new breeding animal (usually a male) is introduced to outcross the herd. Like Ralls and Ballou, we assessed the impact of inbreeding on just one aspect of fitness, infant mortality prior to 30 days.

For two species, the banteng (*Bos javanicus*), a wild cattle species from southeast Asia, and the wisent or European bison (*Bison bonasus*), complete pedigree records allowed calculation of inbreeding coefficients for 33 and 63 descendant animals, respectively. In both species, infant mortality increased as the populations became inbred, although the trend was not statistically significant for the wisent. We estimated the number of lethal equivalents to be 2.33 for the banteng and 1.05 for the wisent. Although these estimates are based on herds established from very few animals (two and three, respectively), it is not surprising that the estimated genetic load of the wisent, a species which has been through a severe bottleneck and which was inbred prior to importation to the U.S.A. [23], is less than half the load of lethal equivalents estimated for the banteng, a species that has a much broader extant range and is not known to have been forced to inbreed during bottlenecks prior to its captive history. It cannot be determined, however, whether the genetic load of the wisent was reduced during the historical bottleneck, or whether its recovery from the bottleneck was facilitated by a smaller genetic load that existed even prior to the bottleneck.

For several other species of bovids bred at the Chicago Zoological Park, incompleteness of pedigrees prevented calculation of inbreeding coefficients. A large herd of Siberian ibex (*Capra ibex sibirica*) were begun from a single male and four females that were brought to the zoo 25 years ago. The herd has been maintained as a closed breeding stock since that time, and it could only have become progressively inbred (although at an unknown rate) during five to ten generations of captive breeding at the zoo. Infant mortality of the ibex was found to have increased significantly through the years, with a sudden increase in mortality coinciding with the replacement of a first-generation male by third and later generation (and therefore highly inbred) males as the dominant breeders in the herd.

For two large antelope species, sitatunga (*Tragelaphus spekei*) and addax (*Addax nasomaculatus*), herds were begun with a single pair more than 50 years ago, but breeding males were supplemented or replaced by unrelated animals every few generations. Thus, there was an alternation between periods of inbreeding and outbreeding within the herd. For both of these species, infant mortality was low in the first few years after each introduction of a new, unrelated breeding male, followed by

significant increases in infant mortality after the herd had been kept closed long enough for the breeding males to be mating with daughters and granddaughters. Because of hunting, addax are very rare and possibly almost extinct in the wild. The few remaining addax in Niger (no addax have been seen outside of Niger in recent years) likely live in family groups even smaller than the herd of 10 to 20 animals kept at Brookfield, and may now be even more inbred than are the zoo animals.

Inbreeding Depression in *Callimico*

Ideally, the effects of inbreeding on captive wildlife should be measured on a single captive population in which it can be documented that inbred and non-inbred animals were given the same care. Except for some ungulate species, like those cases described above, few species are kept in sufficient numbers in any one zoo to permit statistical analysis of the effect of inbreeding on mortality or other measures of fitness. For many species, however, international studbooks document the breeding records from many zoos. If incidences of inbreeding are widely distributed across a number of zoos, then it may be reasonable to assume that differences in mortality of inbred and noninbred animals are not solely due to differences in management among zoos.

One species with a particularly complete and probably accurate studbook is the Goeldi's monkey (*Callimico goeldii*). This monotypic genus is evolutionarily between the Callithricidae and the Cebidae, and often is accorded its own family, the Callimiconidae [27]. Pedigrees of most captive-born *Callimico* can be traced back to wild-caught ancestors from a few importations; paternity is rarely uncertain (because they are maintained as monogamous pairs), and most aborted fetuses, stillbirths, and neonatal deaths have been recorded. The international studbook [28, and unpublished updates] for captive *Callimico* now contains more than 1,000 animals. After eliminating all animals for which there is question as to one or more of the captive ancestors, complete pedigree data are available on 111 inbred and 679 non-inbred captive *Callimico* (including stillbirths and abortions). Working with the studbook keeper, I examined the effect of inbreeding on infant mortality before 30 days of age [20].

Callimico showed the most severe inbreeding depression reported in any vertebrate species for which large sample sizes are available for analysis. Each 10% increase in inbreeding resulted in a 33% decrease in survival. The genetic load was estimated as 7.90 lethal equivalents per diploid genome. Separate analysis of abortions, stillbirths, and infant deaths revealed twofold greater mortality among inbred than non-inbred progeny during each developmental stage. The effects of inbreeding on female infants were found to be much more severe than

on male infants (10.9 vs. 3.2 lethal equivalents), with female progeny from first-cousin matings showing less than half the viability of non-inbred offspring.

Zoo animals are sheltered from many of the causes of mortality that afflict natural populations (e.g., predation, food stress, extremes of weather, epidemic disease), and reduced nongenetic mortality relative to natural populations could lead to greater statistical sensitivity in detecting inbreeding depression. Yet, the benevolent captive environment, while removing many nongenetic causes of mortality, might also minimize the impact of the genotype on survival. Many deleterious alleles would affect fitness only during times of stress, and genetically handicapped animals often live much longer in captivity than they could in the wild. For example, weak infants and those rejected by their mothers are often hand-raised by zoos. Thus, while zoo breeding records provide an opportunity to gather data on much larger pedigrees than could be obtained from studies of wild populations, it should be recognized that they probably provide minimum estimates of the effects that inbreeding would have on populations subjected to the stresses of a more natural environment.

In summary of the cases presented thus far: Extensive data are available to document the deleterious effects of inbreeding on the survival and reproduction of domesticated animals. Studies utilizing laboratory strains of rodents demonstrate that viable, fully inbred strains can be developed, but they have reduced fitness relative to more outbred stocks. Data from zoos demonstrate that the phenomenon of inbreeding depression is widespread among nondomesticated species as well, causing increased infant mortality even in the nurturing environment of zoos. Data on zoo populations suggest also that the severity of inbreeding depression can vary widely among species, populations, and even sexes, but no trends have been identified that would allow prediction, *a priori*, of the quantitative impact of inbreeding on populations that have not yet been studied.

Inbreeding in Insular and Central Populations

To examine the causes of inbreeding depression and to develop quantifiable predictors of the severity of inbreeding depression in populations, Bruce Brewer, I, and colleagues collected mice of the genus *Peromyscus* from island populations and from otherwise isolated populations that had undergone severe declines in past decades, and also from large and stable mainland populations. Brewer established laboratory colonies from eight of the populations and began an extensive study of the effects of inbreeding on each population [29]. The results of that study, summarized briefly below, are presented in detail by Brewer et al. [30].

Several predictions were made regarding the effects of inbreeding on the mouse populations. First, because of the small size and long isolation of the insular and peripheral populations, they were expected to have less genetic variability, when assayed by gel electrophoresis of protein (allozyme) variation, than would the larger, contiguous, mainland populations. Second, the more isolated populations were expected to have smaller genetic loads and therefore to show less depression of fitness traits when forced to inbreed in the laboratory. If these predictions could be confirmed (not only with these mouse populations, but also with a number of species), then conservation programs may be able to use knowledge of the size and history of natural populations to predict the severity of inbreeding depression. Moreover, even if historical data on population size and isolation were lacking, assays of extant genetic variation would be indicative of the size of the genetic load.

Laboratory populations were established from six populations of five subspecies of *Peromyscus polionotus,* and two populations of distinct subspecies of *P. leucopus. P. p. leucocephalus,* a subspecies endemic to Santa Rosa Island along the Gulf coast of northwestern Florida, has been isolated from the mainland for several thousand years and is well differentiated morphologically, behaviorally, and genetically from inland subspecies. Hurricanes periodically decimate the coastal and island populations, and three other subspecies that are endemic to nearby islands and coastal areas are now listed as Endangered by the U.S. Fish and Wildlife Service [31]. *P. p. phasma* formerly inhabited the Atlantic coastal area of northeastern Florida, but has been restricted just to sections of Anastasia Island for the last few decades. This subspecies is now listed as Endangered. *P. p. niveiventris* once inhabited much of the eastern coast of Florida, from Miami to Daytona, but is now restricted to the federally protected habitat on Cape Canaveral. *P. p. rhoadsi* inhabits an inland region of south-central Florida, at the southern end of the present range of the species. Although still widespread, most of the habitat on which it relies (deep, well-drained, sandy soil) has been converted to citrus groves, and mouse densities appear to have declined over the past few decades. Mice from two populations of the subspecies *P. p. subgriseus* were collected from north-central Florida, in areas of extensive habitat. This subspecies is still widespread and abundant. Two subspecies of *P. leucopus, P. l. noveboracensis* from the deciduous forests of the northeastern U.S. and *P. l. tornillo* from Texas, were collected. Both populations inhabit expansive areas of habitat.

Genetic variability within each population was assayed by gel electrophoresis of 30 enzymes in blood, liver, kidney, and muscle samples. The wild-caught mice that were used to initiate lab stocks were analyzed and were found to accord with our predictions: Those populations that are large and abundant showed relatively high levels of genetic variability

(ranging from 8% to 13% heterozygosity), while the island populations had only one-half to one-third as much allozyme variation. Presumably, the island populations have lost variability while inbreeding during previous population crashes.

Each generation in the lab, some mice were paired with relatives to produce inbred offspring. Others were paired with nonrelated mice (of the same population) to produce outbred control litters. To assess the effects of inbreeding, we determined the relationship between several fitness components and the level of inbreeding of litters. We monitored the number of offspring born per litter, the survival of young until weaning at 20 days of age, and the mass of young at weaning. The severity of inbreeding depression was measured by the slope of each of these variables regressed against the inbreeding coefficient of the litters, after the effects of parity of the dam, age of the dam, inbreeding of the dam, and inbreeding of the sire were factored out. Logarithmic transformations were applied to each fitness measure prior to the regression analysis, and this measure of inbreeding depression for juvenile survival estimates the number of lethal equivalents per haploid genome [25, 26].

Unlike the clear trend relating genetic variation to the degree of isolation of populations, the responses of the populations to inbreeding did not follow predictions. First, different components of fitness were affected by inbreeding in the different populations. Some populations produced fewer offspring per litter when inbred, others had reduced growth of inbred litters, and yet a different set of populations suffered greater mortality. Thus, independent genetic factors must be controlling the response of these components of fitness to inbreeding. The ecological, demographic, selective, or historical determinants of the genetic loads of these populations must have acted differently on the various components of fitness because the relative rankings of inbreeding depression depended critically on which components of fitness were examined. Moreover, for none of the fitness measures was there a significant association between the measures of extant genetic variation and the severity of inbreeding depression. Thus, the island populations, which are depauperate in genetic variability and probably have had a history of inbreeding during past population declines, had no lower genetic loads than did mainland populations with more allozyme diversity. Moreover, within each population, inbred offspring of inbred parents fared no better than did inbred offspring of outbred parents, as would have been expected if the genetic load consisted primarily of recessive deleterious alleles that would be removed by selection during repeated generations of inbreeding.

Overall, the effects of inbreeding, both which aspects of life history are affected and the severity of the inbreeding depression, were not

predictable from knowledge of the genetics of the natural populations. This suggests that the effects of inbreeding on any given trait are controlled by a small number of genes and that the presence or absence of those genes in a population is more or less a chance phenomenon. Some populations may be unlucky and harbor a large genetic load, either due to recessive deleterious alleles or due to heterozygote vigor. Other populations may not have genes that would be problematic under inbreeding, but the difference is probably determined by historical accident, not easily identified aspects of population history. The establishment of our lab stocks was similar to the establishment of many reintroduced populations, zoo stocks, and natural recolonizations. In each case, a small number of founders (perhaps 5 to 10) from one population become the nucleus for a new population. Our findings suggest that we are not yet able to predict the severity of inbreeding depression likely to impact recovery programs for endangered species, nor even what traits are likely to be most affected by inbreeding. We must develop and confirm theories adequate to predict accurately the diversity of responses to inbreeding by wild populations. Until we do so, we will have to determine empirically the severity of inbreeding depression and the likely role of genetic processes in extinction or efforts at recovery for each endangered population.

Inbreeding depression is an almost universal phenomenon, and it can impact wild animal species as well as domesticated stocks, but its severity is not yet predictable. There is not yet convincing evidence that selection will commonly adapt populations to inbreeding by removing the genetic load. Yet all of the data that I have presented above have been based on nonnatural populations, either in laboratories or in zoological parks. Thus, an important question rèmains: Does inbreeding depression contribute to the decline and extinction of wild populations?

The Effect of Inbreeding on Natural Populations

It has been argued by many, and at times even by me, that while inbreeding depression is a serious concern for captive breeding programs, in which the goal would be to maintain a small, healthy population in captivity for many generations, habitat loss and demographic problems, not inbreeding, cause the demise of populations in the wild. Captive populations can be protected from so many of the risks that endanger wild populations that they can be propagated for many generations at numbers so low as to lead to severe inbreeding. Wild populations are unstable demographically when they decrease to numbers that would force inbreeding. Very small wild populations would become extinct before many generations pass, and therefore before there is an opportunity for inbreeding depression to exacerbate the decline.

While no natural population is known to have gone extinct because of inbreeding depression, there is increasing evidence that inbreeding depression is contributing to the decline, or impeding recovery, of some wild populations. Certainly, some wildlife populations are now so small that inbreeding is common or even inevitable. For example, the Florida panther (*Felis concolor coryi*) is the only remaining subspecies of puma or mountain lion in the eastern U.S.A. Only about thirty adults remain, and those are divided into two isolated subpopulations [5]. Of the five litters of kittens known to have been born in 1989, one was the product of a father-daughter mating, another was from a mother-son mating, and the other three were of uncertain paternity (two were likely sired by the male that mated to his daughter, and it is likely that he is also the sire of at least one of those two dams). While inbreeding depression (as opposed to just inbreeding) within this tiny population cannot be proven without extensive studies, it is notable that most of the male Florida panthers are cryptorchid (having just one descended testicle), some have congenital heart defects, and several of the traits considered to be diagnostic of the subspecies (a whorl of fur on the back and a kink in one of the tail vertebrae) are probably nonadaptive genetic abnormalities that have become fixed by chance in the small population. The sensitivity of black-footed ferrets, bighorn sheep, and other endangered species to viral diseases and parasites may also be related to a lack of genetic variability resulting from years of inbreeding in remnant populations [32, 33].

There is a great need for studies of the effects of inbreeding on remnant wild populations, but it is difficult to demonstrate the effects of inbreeding in the wild. For example, in the case of the Florida panther, although it is likely that cryptorchism and other developmental anomalies result from the prevalence or fixation of deleterious alleles in that small population, no studies have been conducted to show definitively that these traits have a genetic basis in that population. Lacking field data on inbreeding effects with which to help design a recovery program for the population, I investigated the *likely* impact of inbreeding on the Florida panther population as part of simulation modeling done in conjunction with a Population Viability Analysis workshop convened by the Captive Breeding Specialist Group of the Species Survival Commission (IUCN—The World Conservation Union) and state and federal agencies [5]. A computer program was used to simulate demographic, environmental, and genetic stochasticity. Birth and death rates and the present population structure were estimated from field data and entered into the program. Other examples of the use of this program are given by Lacy et al. [10] and Lacy and Clark [11].

To examine the possible effects of inbreeding on the persistence of the remnant population of Florida panthers, I simulated the population

10,000 times, monitoring changing population sizes and times to extinction. The simulations were repeated under several different scenarios regarding the genetic load of the population. Even in the absence of inbreeding depression, the simulated populations went extinct, on average, in about the year 2030 with a distribution of extinction dates ranging from 2000 to 2100. Three levels of inbreeding depression were modeled: genetic loads of 1.0, 1.7, and 3.4 lethal equivalents. The last case corresponds to the median level of inbreeding depression observed by Ralls, Ballou, and Templeton in their study of 40 populations of mammals in zoos [25]. We presently have no data on the genetic load in Florida panthers, or in any wild felid population. With increasing severity of inbreeding depression, the mean time to extinction of the simulated populations decreased only slightly: the Florida panther population will probably go extinct within 30 to 50 years whether or not fitness declines as the population becomes inbred. The incorporation of inbreeding in the models, however, did remove the right-hand tail of the distribution of times to extinction. If inbreeding affects Florida panthers as much as it does the typical mammal, there is virtually no probability that the population will survive more than 40 years. Inbreeding depression removes the possibility that the panthers will get lucky and happen to survive random demographic and environmental fluctuations into the second half of the next century. This occurs because inbreeding depression reduces fitness as soon as the population becomes very small, rapidly driving it to extinction (the extinction vortex). In the absence of inbreeding depression, some of the simulated populations recover from population crashes and survive for decades beyond an initial decline.

Further insight is gained by examination of the decline in genetic variation in the simulated populations. Selection for heterozygotes, which occurs under the mode of inbreeding depression (general heterosis) modeled in the simulation, was ineffective at retaining genetic variability. At very small population sizes, genetic drift overwhelms selection, and deleterious genes are as likely to be fixed as are advantageous alleles [34]. Simulated populations became extinct after losing 40% to 70% of the initial genetic variation. These simulations are consistent with much experimental work, which suggests that it is very difficult to retain a vertebrate population even for a few generations that has inbreeding coefficients as high as $F = .50$.

With respect to management of the Florida panther, these results suggest that if we wait until the population is very small (on the order of 10 animals) before very aggressive management actions are taken to reverse the decline, it will likely be too late. Similarly, and tragically, it is entirely possible that the California condor, the black-footed ferret, and other species receiving much attention and millions of dollars in recovery efforts may have been so damaged genetically prior to the

recent management efforts that recovery of healthy populations in the wild may be impossible.

Hybrid Vigor: Indirect Evidence of Inbreeding Depression

Recently, my colleagues and I at the Chicago Zoological Park have been examining the role that genetic variation may play in maintaining the health of natural populations, not by looking at the effects of forced inbreeding, but rather by examining the response of populations to increases in genetic variation resulting from hybridization among divergent populations of the same species. We have made 20 to 30 crosses between each pair of the six populations of *Peromyscus polionotus* used in the inbreeding experiments [29, 30] described above. (Only noninbred control stocks were used for this study, not the artificially inbred mice produced during the inbreeding experiments.) These populations include two local samples of one subspecies, several closely related subspecies, and also some subspecies (e.g., the island forms) that are sufficiently distinctive that some biologists have suggested that they merit consideration as full species. Hybrid progeny produced by these crosses were subsequently bred to produce second-generation (F2) between-population hybrids.

As in the inbreeding experiments, we monitored the number of offspring per litter, the survival of young to weaning, and the mass of offspring at weaning. In addition, we controlled the length of time that each pair was left together (63 days) and examined the proportion of pairs producing litters during that time. We expected that the matings between closely related subspecies would produce litters as large and healthy as those of the within-population control matings, but we thought that crosses between strongly divergent subspecies (e.g., those involving the Santa Rosa Island endemic population) might show reduced fitness due to incompatibility of hybrid genotypes. Often, first-generation hybrids between divergent populations or even biological species display hybrid vigor, while hybrid breakdown occurs when these hybrids are mated to produce second-generation hybrids [35, 36].

Our preliminary results (not yet published) have not supported our predictions. For every fitness component measured, there was strong hybrid vigor. This effect was even stronger in the F2 generation, perhaps due to good parental care given by F1 hybrid dams. Moreover, the isolated populations that are the most divergent genetically, and that have the least genetic variation, were the ones that showed the strongest hybrid vigor. Crosses between similar, and highly variable, mainland populations showed little or no hybrid vigor. This suggests that the isolated populations are already experiencing depression of fitness due

to the loss of genetic variation; inbreeding depression is suppressing fitness in the existing natural populations.

One of the two island populations included in this study is declining steadily and is now listed as Endangered, and the other is being reviewed for possible listing as Threatened. Of five other isolated subspecies of beach mice, a peninsular population (also used in this study) is listed as Threatened, an island population and two coastal populations are Endangered, and one coastal form is already extinct. Conversion of the habitat into residential areas and public beaches has limited the mouse populations to very narrow strips of coastal dunes, often in areas that are highly susceptible to damage by storms (and therefore unsuitable for residential development by humans). Hurricanes and house cats have decimated these remnant, local populations of beach mice. It now seems that losses of genetic variation have reduced the viability and reproduction of the more endangered populations, and that recovery of the populations may not be possible even if they are afforded protection from further habitat damage.

Conclusion

Inbreeding depression is likely to be one of the causal factors in the extinction of wildlife populations. Inbreeding will only become important after other factors have already reduced the populations to critically low numbers, but many wildlife populations are now down to so few breeding animals that genetic problems are likely to compound the more obvious causes of decline. While some theorists state that genetic considerations are unimportant in the conservation of wildlife populations because inbreeding depression only begins when the size of isolated populations are below 50, many wildlife agencies are failing to take aggressive action to reverse population declines until numbers drop as low as 10 or 20. Inbreeding effects will make the recovery of some endangered species even more difficult than has been presumed heretofore.

REFERENCES

1. Darwin, C. *The Variation of Animals and Plants under Domestication.* London: John Murray, 1868.
2. Wright, S. *Evolution and the genetics of populations. Vol. 3. Experimental Results and Evolutionary Deductions.* Chicago: University of Chicago Press, 1977.
3. Falconer, D. S. *Introduction to Quantitative Genetics.* 2d ed. New York: Longman, 1981.
4. Gilpin, M. E., and Soulé, M. E. Minimum viable populations; processes of species extinction. Pages 19–34 in *Conservation Biology: The Science of Scarcity and Diversity,* edited by M. E. Soulé. Sunderland, Mass.: Sinauer, 1986.

5. Seal, U. S., and Lacy, R. C. Florida panther population viability analysis. *Report to the U.S. Fish and Wildlife Service.* Apple Valley, Minn.: Captive Breeding Specialist Group, Species Survival Commission, IUCN, 1989.
6. Shaffer, M. Minimum population sizes for species conservation. *BioScience* 31:131–134, 1981.
7. Shaffer, M. L. Minimum viable populations: coping with uncertainty. Pages 69–86 in *Viable Populations for Conservation,* edited by M. E. Soulé. Cambridge: Cambridge Univ. Press, 1987.
8. Brussard, P. F. Minimum viable populations: how many are too few? *Restoration and Management Notes* 3:21–25, 1985.
9. Gilpin, M. E. Population viability analysis. *Endangered Species Update* 6: 15–18, 1989.
10. Lacy, R. C.; Flesness, N. R.; and Seal, U. S. Puerto Rican parrot population viability analysis. *Report to the U.S. Fish and Wildlife Service.* Apple Valley, Minn.: Captive Breeding Specialist Group, Species Survival Commission, IUCN, 1989.
11. Lacy, R. C., and Clark, T. W. Population viability assessment of the eastern barred bandicoot in Victoria. Pages 131–145 in *The Management and Conservation of Small Populations,* edited by T. W. Clark and J. H. Seebeck. Brookfield, Ill.: Chicago Zoological Society, 1990.
12. Lacy, R. C. The effects of inbreeding on isolated populations: are minimum viable population sizes predictable? Pages 278–296 in *Conservation Biology: The Theory and Practice of Nature Conservation, Preservation and Management,* edited by P. L. Fiedler and S. K. Jain. New York: Chapman and Hall, 1992.
13. Lacy, R. C., and Clark, T. W. Genetic variability in black-footed ferret populations: past, present, and future. Pages 83–103 in *Conservation Biology and the Black-Footed Ferret,* edited by U. S. Seal, E. T. Thorne, M. A. Bogan, and S. H. Anderson. New Haven: Yale Univ. Press, 1989.
14. Seal, U. S.; Thorne, E. T.; Bogan, M. A.; and Anderson, S. H., eds. *Conservation Biology and the Black-Footed Ferret.* New Haven: Yale Univ. Press, 1989.
15. Dinerstein, E., and McCracken, G. F. Endangered greater one-horned rhinoceros carry high levels of genetic variation. *Conserv. Biol.* 4:417–422, 1990.
16. Lerner, I. M. *Genetic Homeostasis.* New York: John Wiley and Sons, 1954.
17. Strong, L. C. Inbred mice in science. Pages 45–67 in *Origins of Inbred Mice,* edited by H. C. Morse. New York: Academic Press, 1978.
18. Lynch, C. B. Inbreeding effects upon animals derived from a wild population of *Mus musculus. Evolution* 31:526–537, 1977.
19. Connor, J. L., and Bellucci, M. J. Natural selection resisting inbreeding depression in captive wild house mice (*Mus musculus*). *Evolution* 33:929–940, 1979.
20. Lacy, R. C.; Petric, A. M.; and Warneke, M. Inbreeding and outbreeding depression in captive populations of wild species. In *The Natural History of Inbreeding and Outbreeding,* edited by N. W. Thornhill. Chicago: University of Chicago Press. (In press.)
21. Shoemaker, A. H. The effect of inbreeding and management on propagation of pedigree leopards. *Internat. Zoo Yearb.* 22:198–206, 1982.
22. Daniell, A., and Murray, N. D. Effects of inbreeding in the budgerigar *Melopsittacus undulatus* (Aves: Psittacidae). *Zoo Biol.* 5:233–238, 1986.
23. Slatis, H. M. An analysis of inbreeding in the European bison. *Genetics* 45:275–287, 1960.

24. RALLS, K., and BALLOU, J. Extinction: lessons from zoos. Pages 164–184 in *Genetics and Conservation: A Reference for Managing Wild Animal and Plant Populations,* edited by C. M. Schonewald-Cox, S. M. Chambers, B. MacBryde, and W. L. Thomas. Menlo Park, Calif.: Benjamin/Cummings, 1983.
25. RALLS, K.; BALLOU, J. D.; and TEMPLETON, A. Estimates of lethal equivalents and the cost of inbreeding in mammals. *Conserv. Biol.* 2:185–193, 1988.
26. MORTON, N. E.; CROW, J. F.; and MULLER, H. J. An estimate of the mutational damage in man from data on consanguineous marriages. *Proc. Natl. Acad. Sci. USA.* 42:855–863, 1956.
27. HERSHKOVITZ, P. *Living New World Monkeys, Vol. 1.* Chicago: University of Chicago Press, 1977.
28. WARNEKE, M. *Callimico goeldii 1988 international studbook.* Brookfield, Ill.: Chicago Zoological Society, 1988.
29. BREWER, B. A. *An Investigation of Protein Electrophoresis as a Predictor of Inbreeding Depression in Captive Populations of Peromyscus.* Ph.D. Thesis. Cornell University, Ithaca, New York, 1988.
30. BREWER, B. A.; LACY, R. C.; FOSTER, M. L.; and ALAKS, G. Inbreeding depression in insular and central populations of *Peromyscus* mice. *J. Heredity* 81:257–266, 1990.
31. U.S. FISH AND WILDLIFE SERVICE. *Recovery Plan for the Choctawhatchee, Perdido Key and Alabama Beach Mouse.* Atlanta, Ga.: US Fish and Wildlife Service, 1987.
32. O'BRIEN, S. J., and EVERMANN, J. F. Interactive influence of infectious disease and genetic diversity in natural populations. *Trends in Ecology and Evolution* 3:254–259, 1988.
33. BERGER, J. Persistence of different-sized populations: An empirical assessment of rapid extinctions in bighorn sheep. *Conserv. Biol.* 4:91–98, 1990.
34. ROBERTSON, A. Selection for heterozygotes in small populations. *Genetics* 47:1291–1300, 1962.
35. TEMPLETON, A. R. Coadaptation and outbreeding depression. Pages 105–116 in *Conservation Biology: The Science of Scarcity and Diversity,* edited by M. E. Soulé. Sunderland, Mass.: Sinauer, 1986.
36. TEMPLETON, A. R.; HEMMER, H.; MACE, G.; SEAL, U. S.; SHIELDS, W. M.; and WOODRUFF, D. S. Local adaptation, coadaptation, and population boundaries. *Zoo Biol.* 5:115–125, 1986.

The importance of genetic variation to the viability of mammalian populations

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Abstract -- Small populations lose genetic variability because of genetic drift, and inbreeding within populations can further decrease individual variability. Lower variation depresses individual fitness, resistance to disease and parasites, and flexibility in coping with environmental challenges. Lower variation decreases population mean fitness (population growth rates), resilience, and long-term adaptability. Genetic drift can threaten population viability not just by depleting variation, but also by replacing natural selection as the predominant force driving evolutionary change. Although most genetic studies use laboratory or domesticated populations, evidence is accumulating that the effects of inbreeding are at least as severe on wild animals in natural habitats. Natural selection is expected to reduce the frequency of deleterious alleles in populations that persist through bottlenecks, but as yet there is little evidence for such purging of the genetic load in mammalian populations. No species of mammal has been shown to be unaffected by inbreeding. Genetic problems are contributing to the decline and vulnerability of at least several mammalian taxa. Genetic threats to population viability will be expressed through their effects on and interactions with demographic and ecological processes. Theoretical analyses, experimental tests, field studies, and conservation actions should recognize the fundamental interdependency of genetic and non-genetic processes affecting population viability.

Biodiversity, both in terms of the numbers of varieties of living organisms and the variety of processes that support and interlink forms of life, is being depleted at an unparalleled rate. The loss of biodiversity is occurring at all levels: damaged ecosystems, destroyed ecological communities, extinction of species, loss of genetically and ecologically distinctive populations, and loss of genetic variation among and within individuals in local populations. While most biologists bemoan losses of biodiversity at all of its levels, and all levels are clearly dependent upon the health of the others, there has been considerable debate regarding which levels are fundamental to sustaining biodiversity. In this essay, I address the importance of genetic variation within and among individuals to the viability of populations, and therefore also to the higher levels of biodiversity of which those populations are functional components. Although I will argue that individual variation is critical to population viability and deserves more attention in conservation efforts, I do not presume to make a case for the urgency or importance of such attention compared to actions focussed on other levels of biodiversity.

ROLES OF GENETIC VARIATION IN POPULATION VIABILITY: THEORETICAL FOUNDATIONS

Genetic variation is both a trait of individuals and a trait of populations. Variation within individuals of diploid species is most commonly characterized by the percent of loci at which an individual is heterozygous. Variation within populations includes also inter-individual variation, and is often quantified by the "gene diversity" (the heterozygosity expected under Hardy-Weinberg equilibrium), by the number of distinct alleles per locus, or by the percent of loci which are polymorphic (Nei, 1973). Mean within-individual variation is usually highly correlated with populational (between-individual) variation, and all measures of populational variation encompass also the within-individual variation in the population. Thus, the distinction between within- and between-individual variation and the distinct roles of variation at these different levels are easily confused.

Effects on individual fitness. -- Heterozygosity is depleted by inbreeding (mating between relatives), which leads to a greater probability of the two alleles at a locus being identical by descent from an ancestor common to both

sides of the pedigree, and by genetic drift (random fluctuations in allele frequencies). Inbreeding has been observed to cause higher mortality, lower fecundity, reduced mating ability, slower growth, developmental instability, more frequent developmental defects, greater susceptibility to disease, lowered ability to withstand stress, and reduced intra- and inter-specific competitive ability (Darwin, 1868, 1876; Lerner, 1954; Wright, 1977; Allendorf and Leary, 1986; Ledig, 1986; Ralls et al., 1988; Falconer, 1989). The variety of impacts on fitness is collectively termed "inbreeding depression". Inbreeding depression could result from the increased exposure in homozygotes of deleterious recessive alleles, or from an advantage of heterozygotes over each homozygous type (heterozygote advantage), or both (Crow, 1948).

Effects on population fitness. -- Loss of genetic variation can impact population persistence in several ways. First, the lower fecundity and survival of inbred individuals within a population will depress population growth rate, which in turn can increase the probability of extinction from stochastic fluctuations (Goodman, 1987). Lower capacity for population growth will also reduce ability to rebound from population declines, especially as the impacts of inbreeding on individuals are accentuated in stressful environments (Lerner, 1954; Schmitt and Ehrhardt, 1990; Miller, 1994; Keller et al., 1994). Prolonged population bottlenecks will lead to yet greater loss of variation through genetic drift.

Even if lowered variation has little impact on a population's fitness in its present environment, or if the reduction in population growth and resilience is not sufficient to threaten short-term persistence, a decrease in variation will reduce the population's ability to adapt to changing environments. First, the rate of evolutionary response to selection on a trait is proportional to the additive genetic variation (heritability) of that trait (Fisher, 1958), which is in turn proportional to expected heterozygosity or gene diversity of loci influencing the trait (Falconer, 1989). Second, the scope for evolutionary adaptation is delimited by the existence of alternate alleles within the population (Robertson, 1960; James, 1971). Thus, a population with low heritability, low heterozygosity, and few polymorphic loci will adapt more slowly and attain lesser adaptation before reaching the limits of response to selection than will a more diverse population. Selfing and parthenogenetic species, with little variation within individuals and between individuals, respectively, tend to have short evolutionary histories (Selander, 1983; Vrijenhoek, 1989). Bürger and Lynch (1995) found that fluctuations in genetic variance in small populations can reduce the rate of adaptation sufficiently to cause small populations to go extinct in the face of environmental change to which large populations would be able to adapt. We cannot know what adaptations will be required for persistence in future environments, but we do know that the rate of environmental change is much more rapid presently than perhaps at any time in past evolutionary history.

Franklin (1980) first suggested that populations would need effective sizes of about 500 in order for mutation to offset the loss of variation through genetic drift, thereby preserving long-term adaptive potential. Further analyses led to similar conclusions, even when the effects of stabilizing selection on quantitative traits were considered (Lande and Barrowclough, 1987). As a result of more recent work, however, Lande (1995a) has become even less optimistic about the potential for small populations to retain adaptive potential. Because most mutations with large effects are highly detrimental, the rate of incorporation of new genetic variation into populations is much slower than the overall mutation rate. Considering the rate of mutations that are not strongly detrimental, Lande concluded that effective breeding population sizes will need to be on the order of 5,000, rather than 500, to ensure long-term viability. Given that total population sizes of mammals are usually several-fold larger than effective population sizes (Frankham, in press a), the long-term genetic risks to small, isolated populations have probably been substantially underestimated in most conservation programs.

Recent theoretical models (Lynch and Gabriel, 1990; Lande, 1994) demonstrate yet another process by which the long-term viability of small populations might be threatened by genetic drift. In small populations (on the order of hundreds of breeding individuals), changes in allele frequencies are more strongly determined by random genetic drift than by natural selection, except when selection is strong (Kimura, 1983; Lacy, 1987). Therefore, deleterious mutations occasionally become fixed in a small population, due to chance drift, replacing more adaptive alleles. As deleterious mutations accumulate, population size may decrease, causing genetic drift to become even more rapid. This feedback has been termed "mutational meltdown." The time course of mutational meltdown is on the order of hundreds of generations, however, so it would not be a significant contributor to recent and rapid population declines.

EVIDENCE FOR EFFECTS OF GENETIC VARIATION ON THE VIABILITY OF POPULATIONS OF MAMMALS

The possibility that decreased genetic variation can impact individual and population fitness does not necessarily mean that lack of genetic variation is a causal factor in population declines and extirpation. First, the inbreeding depression observed in laboratory animals and domesticated livestock might not be generalizable to natural populations. Second, populations that remain small may become adapted to low levels of genetic variation. Third, because of habitat destruction, direct killing by humans, replacement by exotic competitors, and the demographic instability inherent in small numbers, natural populations may rarely persist at small numbers long enough to be threatened by genetic processes (Lande, 1988; Caughley, 1994). Finally, mechanisms maintaining and restoring adaptive variation may be sufficient to preserve adequate variation for adaptive evolution even in rather small populations (Lande, 1975, 1995b). Each of these points will be addressed below.

Does inbreeding depression affect mammals in natural habitats? -- Inbreeding depression has been assessed mostly in domesticated livestock and laboratory organisms, with relatively few studies on inbreeding in wildlife populations or recently established captive populations of wild species (Lacy, 1993a; Lacy et al., 1993). Domesticated and laboratory populations have had their genomes considerably modified by centuries of artificial selection, most have been specifically selected for viability under inbreeding, and they are studied in highly modified and benign habitats. Therefore, it is possible that effects of inbreeding on such populations might be different from effects on wild populations in natural habitats. House mice (*Mus domesticus*) are thought to inbreed frequently in the wild (Selander, 1970; Smith, 1993), and extensive inbred lines have been developed for biomedical research, so it might be expected that they would be less affected by inbreeding than are most mammals. Yet, much of what is known about the deleterious effects of inbreeding in mammals comes from studies on house mice (Falconer, 1989). Production of inbred mice entails losses of many of the lines (Bowman and Falconer, 1960; Lynch, 1977), established inbred lines still show strong increases in fitness when outcrossed (Wright, 1977; Falconer, 1989), and renewed inbreeding of heterogeneous stocks created by crossing previously inbred lines results again in depression of a wide variety of fitness traits (Deckard et al., 1989).

Experiments on plants (Dudash, 1990) and animals (Jiménez et al., 1994) indicate that the deleterious impacts of inbreeding are more severe in more natural environments than in controlled laboratory or agricultural settings. However, Shields (1982, 1993) argues that natural populations might be much less impacted by inbreeding than would be suggested by laboratory studies. With respect to wild populations of vertebrates, Shields (1993, p. 169) states "in every case with sufficient evidence there is either no inbreeding depression or in a few cases even significant inbreeding enhancement." Indeed, a few studies have purported to show (or have been claimed by others to show) that a natural population of mammals was unaffected by inbreeding (e.g., Rood, 1987; Bulger and Hamilton, 1988; Hoogland, 1992). However, in each case, the sample size examined was inadequate to allow detection of inbreeding depression, even if it were more severe than is typically reported in experimental populations.

It is important to recognize, especially when sample sizes are small and confounding factors are difficult to control, that the lack of statistical evidence for inbreeding depression is not equivalent to evidence for the lack of inbreeding depression. For example, Bulger and Hamilton (1988) reported that the mortality among infants born in baboon troops in which the dominant male had remained in his natal troop was similar to the mortality of infants born in troops into which the dominant male had immigrated (5/20 inbred vs. 4/27 non-inbred deaths). Yet, guessing that non-dispersing males were on average breeding with half-siblings, the 12% lower survival of their progeny reported in the study, while not approaching statistical significance, would be comparable to the typical depression in survival of progeny from half-sibling matings in domesticated and experimental populations of mammals (Falconer, 1989). Another study on a natural population of baboons indicated significant damaging effects of inbreeding, albeit also with small sample size (Alberts and Altmann, 1995). Hoogland (1992) analyzed a much larger data set, but because prairie dogs avoid close inbreeding most of his "inbred" matings were between relatives more distant than first-cousins (inbreeding coefficient $F < 0.0625$). There were too few matings between closer relatives to allow statistical detection of any impacts of inbreeding. To achieve a 50% probability of detecting a difference in survival (D) between inbred and non-inbred animals at a significance level of $P < 0.05$ requires sample sizes as large as $2/D^2$ for each of the two groups. Thus, detecting a 1% decline of survival for each 1% increase in inbreeding requires samples of more than 128 half-sib matings, 500 first-cousin matings, or 8,000 second-cousin matings, as well as comparable numbers of non-inbred matings. These numbers make clear that the demonstration of effects of inbreeding on wild populations will continue to be difficult. With sample sizes attainable in wild populations, we will not normally be able to detect fitness differentials of a few percent. Yet a few percent decline in demographic rates is sufficient to turn

many healthy populations of wildlife into declining ones.

Contrary to suggestions that the existence of some inbreeding in natural populations indicates that inbreeding depression may not be a real phenomenon in the wild (Shields, 1993), measuring the degree of inbreeding in a natural population is not the same as testing for inbreeding depression. There are costs to dispersal which might make absolute avoidance of inbreeding counterproductive, and harmful inbreeding could result from the inability of individuals to distinguish kin or the inability to disperse randomly (Smith, 1993). Because many habitats have been severely reduced and fragmented, some species may have dispersal behaviors that are no longer optimal for the landscapes in which they find themselves, or they may simply be unable to avoid inbreeding. The naked mole rat (*Heterocephalus glaber*) is a particularly interesting mammalian species with regard to inbreeding. DNA fingerprinting data indicate that colonies of mole rats are highly inbred (Reeve et al., 1990), presumably as a result of the difficulty in dispersing and entering or establishing new colonies. Clearly, this high level of inbreeding has not been frequently fatal to colonies, and the species may have evolved a genome that is relatively unaffected by inbreeding. It would be of considerable interest to know whether more heterozygous colonies (e.g., those more recently established) have greater fecundity and survival than those colonies which have accumulated higher levels of inbreeding.

In spite of the difficulty of demonstrating inbreeding depression in the wild, several recent studies have demonstrated effects of inbreeding on mammals in natural habitats. Stockley et al. (1993) studied a population of common shrews (*Sorex araneus*) in England, using multilocus DNA fingerprinting to identify pairs that were closely related. They found that more inbred shrews tended to be smaller at weaning and less likely to survive to maturity than more outbred individuals. Jiménez et al. (1994) released into a woodland habitat 367 *Peromyscus leucopus* produced by brother-sister matings ($F = 0.25$), and 419 mice produced by matings between non-relatives. Re-trapping showed a more rapid loss of inbred than non-inbred mice.

Although the correlation between heterozygosity at a few sampled allozyme loci and overall heterozygosity across the genome is expected to be weak, a few studies of mammals have found significant associations between allozyme variation and fitness components. Cothran et al. (1983) reported a greater rate of twinning, higher maternal weight, and faster fetal growth in white-tailed deer (*Odocoileus virginianus*) that were heterozygous at more allozyme loci. Horn growth is faster in more heterozygous bighorn sheep (*Ovis canadensis*), presumably giving them an advantage in competition for mates (Fitzsimmons et al., 1995). It is possible that the allozyme loci themselves were responsible for these fitness differences, but it is more likely that allozyme heterozygosity was serving as a marker for important variation at linked loci, or that allozyme variation was correlated with inbreeding and that the effects of inbreeding were manifest through actions of genes elsewhere in the genome. Many non-mammalian examples of effects of allozyme heterozygosity on fitness components are known, including some in which heterozygosity of the enzyme variants themselves has been shown to be responsible for the fitness benefits (reviewed by Mitton, 1993).

Do natural populations of mammals become adapted to inbreeding? -- If the impacts of inbreeding are primarily due to the expression of a "genetic load" of deleterious recessive alleles in the more homozygous inbred individuals, then a population that weathers several generations of inbreeding may become purged of this genetic load (Charlesworth and Charlesworth, 1987; Hedrick and Miller, 1992). Thus, some inbreeding may actually be beneficial to future population viability by improving the efficiency with which natural selection removes deleterious recessive mutations from the gene pool.

There are data suggesting that selfing plants become partly purged of their genetic loads (Lande and Schemske, 1985; Barrett and Charlesworth, 1991; but see Barrett and Kohn, 1991), and some data on human populations is suggestive that purging takes place (Rao and Inbaraj, 1980; but see Bittles et al., 1991). Overall, however, there is as yet little evidence for purging of the genetic load through bouts of inbreeding in mammalian populations. For example, mammalian taxa that are endangered or known to have gone through bottlenecks (e.g., parma wallaby, *Macropus parma*; golden lion tamarins, *Leontopithecus rosalia*; Pere David's deer, *Elaphurus davidianus*; Eld's deer, *Cervus eldi thamin*; scimitar-horned oryx, *Oryx dammah*; Speke's gazelle, *Gazella spekei*) have genetic loads as great as the common species listed in a survey of 40 mammalian populations by Ralls et al. (1988). Templeton and Read (1983, 1984) reported a reduction in genetic load in a captive population of Speke's gazelle when measured in the progeny of inbred parents, but their statistical approach was flawed. The small sample correction they applied to survival at each level of inbreeding causes a bias toward lower estimates of genetic load. Because the sample sizes for animals with inbred parents were smaller than the sample sizes for animals with non-inbred parents, this bias could have created the appearance of reduced inbreeding depression. In addition, they forced the regression lines of viability against inbreeding to have a y-intercept equal to the weighted average of the intercepts

estimated for inbred and non-inbred parents. This exaggerated the difference in slopes and greatly reduced the estimated error variances of the slopes. Their analysis would be appropriate only if it could be assumed that the survival of non-inbred progeny did not change over time and was unaffected by the inbreeding level of the dam, and if the intercept were known without error. These assumptions are not warranted. When the lines are not forced to have a common fixed intercept, the difference between the genetic loads measured in progeny of inbred vs. non-inbred parents becomes small and non-significant, even with the bias of the small sample correction. More appropriate analyses of the now larger Speke's gazelle pedigree do not support the claim for purging (Ballou, 1995; Willis and Wiese, in press). Ballou (1995) found evidence for only slight amelioration of the effects of inbreeding through generations of captive breeding in zoo populations. While most species showed a shift in the expected direction of lower genetic loads, significant ($P < 0.05$) purging was seen in just one of 25 species.

The recovery of European bison (*Bison bonasus*) from a bottleneck of 17 animals earlier this century is often cited as evidence for the ability of natural selection to purge populations of their genetic load and allow recovery in spite of low genetic variation (Templeton and Read, 1983; Simberloff, 1988). Although Slatis (1960) reported low inbreeding depression in the post-bottleneck herd of bison, more recent analyses show that significant inbreeding depression still occurs (Lacy et al., 1993), and that there has been no significant purging of the genetic load in the pedigree of the captive population (Ballou, 1995).

In a controlled breeding experiment designed to test the hypothesis that small natural populations have become partly adapted to inbreeding, Brewer et al. (1990) found that insular populations of *Peromyscus* mice had genetic loads at least as great as the larger central populations. Ribble and Millar (1992) compared the effect of full-sib matings in a stock of *Peromyscus maniculatus* that had become moderately inbred over 20 generations of laboratory breeding to the effect of inbreeding in a recently established stock. The recently established stock showed significant inbreeding depression; the previously inbred stock did not. Their data are suggestive of prior purging in the long-established laboratory stock. However, small sample sizes precluded statistical significance of even sizable inbreeding depression, as the full-sib matings in the older stock produced 21% fewer progeny than the control matings in this stock. Moreover, the recently established stock cannot be considered to be a control group against which to compare inbreeding depression in the older stock, because they were derived from different source populations. Inbreeding depression in the base population from which the older stock had been derived may have been no different than the inbreeding depression observed after 20 generations of selection for productivity.

Unless the genetic load consists of a few highly deleterious recessive alleles, selection is inefficient at purging the genetic load, and extinction is a more likely consequence of inbreeding than is purging of the genetic load (Hedrick, 1994). If the impacts of inbreeding are due to numerous weakly deleterious alleles, alleles that are damaging only in some environments, or alleles maintained by heterozygote advantage, then genetic drift will often lead to fixation of deleterious alleles during population bottlenecks. Technically, if deleterious alleles are fixed in a population, reducing its average fitness but perhaps not causing extinction, then the genetic load has been "removed" because no genetic variance in fitness remains. Further inbreeding may not cause further harm, because there is no scope for getting worse!

Does variation affect the viability of natural populations? -- The recovery of northern elephant seals (*Mirounga angustirostrus*) to large numbers even after they apparently lost much of their genetic variation during a population bottleneck (Bonnell and Selander, 1974) and the persistence of cheetahs (*Acinonyx jubatus*) with very low levels of variation (O'Brien et al., 1983, 1985; O'Brien, 1994a) are sometimes cited as evidence that losses of genetic variation might be unimportant to population viability (e.g., Simberloff, 1988; Caro and Laurenson, 1994). However, it is not known how many comparable populations went extinct following such bottlenecks.

In questioning the importance of genetics to conservation, Caughley (1994, p. 239) stated "no instance of extinction by genetic malfunction has been reported". Similar claims have been made by others (e.g., Lande, 1988; Caro and Laurenson, 1994; Harcourt, 1995). Unfortunately, evidence is accumulating that some small and isolated populations of mammals have been depleted of genetic variation and as a result are suffering decreased fitness. The population of lions isolated in the Ngorongoro Crater in Tanzania was reduced to just 10 animals in 1962, with 7 subsequent immigrants. The descendant population has been found to have less genetic variation, a higher rate of sperm abnormalities, and lower sperm motility than the nearby population in the Serengeti (Packer et al., 1991). Similarly, the remnant population of Asian lions in the Gir Forest has relatively little genetic variation, very low sperm counts, and a high rate of deformed spermatozoa (O'Brien et al., 1987a; Wildt et al., 1987a). Florida panthers (*Felis concolor coryi*) have been reduced to about 30 individuals in the remnant population in southern Florida, and parent-offspring breeding has been documented. Compared to the larger populations of the species in the western

U.S.A., Florida panthers have low genetic variation, poor sperm quality, frequent cryptorchidism, and high susceptibility to microbial parasites (Roelke et al., 1993; O'Brien, 1994a). Cheetahs may have recovered from one or more ancient bottlenecks (O'Brien et al., 1987b) and may suffer presently from numerous non-genetic threats (Caro and Laurenson, 1994; Merola, 1994), but they also have a high rate of defective sperm (Wildt et al., 1987b), appear unusually susceptible to diseases (O'Brien et al., 1985; O'Brien, 1994a), have high fluctuating asymmetry (Wayne et al., 1986; but see Willig and Owen, 1987, and the rebuttal by Modi et al., 1987), and suffer increased juvenile mortality when inbred further in captivity (Caughley, 1994). See Merola (1994) and O'Brien (1994b) for continuing debate on the cheetah controversy.

The concentration of examples of damaging effects of loss of genetic variation among the large cats might be because large solitary carnivores are likely to be more quickly impacted by fragmentation and loss of habitat than are species with greater population densities, or it might simply be a consequence of the focus on the conservation genetics of felids by O'Brien and his collaborators versus the lack of such attention to other taxa. Similarly, the frequency of reports of impacts on male fertility might indicate a particular vulnerability to inbreeding, or it might simply reflect that semen quality is more easily monitored in natural populations than female fecundity, disease resistance, feeding efficiency, social dominance, or other components of fitness.

Caughley (1994) questioned the relevance of measures of genetic variation in natural populations, pointing out that there are not data to show a correlation between equilibrium heterozygosity and individual or population fitness. Equilibrium levels of genetic variation result from the balance between the forces of mutation, genetic drift, migration, and various types of selection (Wright, 1969); low equilibrium heterozygosity can occur for any of a number of reasons; and the amount of genetic variation observed in apparently healthy natural populations varies widely (Nevo, 1978). Thus, it would not be justified to assume that populations with lower equilibrium levels of genetic variation are less viable.

Caughley apparently over-looked, however, that low genetic variation resulting from a bottleneck is not in evolutionary equilibrium. Concern about genetic variation is appropriate when there is evidence that variation has been reduced below historic equilibrium levels. The evidence that variation is depressed is admittedly indirect, but comes from comparisons to similar taxa (cheetahs and lions [O'Brien, 1994a], Florida panthers [Roelke et al., 1993], elephant seals [Hoelzel et al., 1993; Hedrick, 1995]), from analytical calculations (elephant seals [Hedrick, 1995], black-footed ferrets, *Mustela nigripes*, [Lacy and Clark, 1989]), from simulation models (Florida panthers [Seal and Lacy, 1989], elephant seals [Hoelzel et al., 1993]), or from documentation of matings between close relatives (Florida panthers [Roelke et al., 1993]). Unfortunately, in none of these cases do we have a direct measure of the genetic variation that was present before the observed or hypothesized bottleneck. Similarly, although we have some cross-populational comparative data for the big cats, we do not have measures of fitness in these populations prior to the bottlenecks.

While none of the above taxa has yet gone extinct, and all suffer more from non-genetic threats than genetic ones, Berger (1990) found that populations smaller than 50 bighorn sheep went extinct within 50 years, whereas populations of more than 100 persisted for more than 70 years. He speculated that loss of genetic variation in smaller populations contributed to the more rapid population extinctions. Outside of the Mammalia, there is evidence that inbreeding depression was a contributing proximate cause of the extinction of the Swedish population of middle spotted woodpeckers (*Dendrocopos medius*) and the heath hen (*Tympanuchus cupido*) (Pettersson, 1985; Simberloff, 1988). While the evidence of genetic problems in these cases is circumstantial, it is not true that "no instance of extinction by genetic malfunction has been reported".

Assessing the impacts of inbreeding on population viability can be difficult because usually only one or a few components of fitness are monitored (Hedrick and Miller, 1992; Shields, 1993). English great tits (*Parus major*) showed reduced nesting success when inbred (Greenwood et al., 1978). An island population of the same species in the Netherlands had a reduced hatching rate when inbred, but also greater nestling survival and consequently no difference in recruitment into the breeding population (van Noordwijk and Scharloo, 1981). A series of studies of natural populations of desert topminnows (*Poeciliopsis* spp.) demonstrated multiple impacts of genetic variation on population viability. Loss of genetic variation during population bottlenecks caused slower growth, lower fecundity, greater fluctuating asymmetry, high frequency of developmental abnormalities, poorer survival under stressful conditions (hypoxia), higher parasite loads, and lower interspecific competitive ability -- and these multiple impacts were reversed when genetic variation was restored via outcrossing (Quattro and Vrijenhoek, 1989; Vrijenhoek, 1994). Contrary to claims that "it has yet to be shown that inbreeding depression has caused any wild population to decline" (Caro and Laurenson, 1994), population numbers of the topminnows responded as expected when genetic

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variation was lost and then restored.

It will be difficult to obtain data on sufficient numbers of populations of any species in the wild to allow determination of the functional relationship between inbreeding and population extinction. To examine this relationship, Frankham (1995) analyzed the effect of inbreeding on population survival in four studies using 60 to 120 of experimental populations of *Mus* and *Drosophila*. In each case, he found that population extinction rates were strongly elevated by progressive inbreeding within the populations. Moreover, he found that the relationship was a threshold effect. Extinction rates remained low through early generations of inbreeding, but increased sharply after several generations of inbreeding. This was especially true of the *Mus* populations, in which only three of 60 populations survived. Frankham (1995, p. 797) appropriately cautions: "There may be little warning of impending extinction due to inbreeding in wildlife, especially with species that are not intensively monitored."

Is evolutionary potential constrained by extant genetic variation? -- The limitation of adaptive potential by reduced levels of genetic variation has been demonstrated with respect to the response by experimental populations to selection for specific traits (Wright, 1977), and is a fundamental principle of modern agricultural genetics. Response to selection is one means of measuring the heritable variation in a population (Falconer, 1989). One of the arguments made for the preservation of natural biodiversity is that the genetic variability contained in wild populations of domesticated species is an essential resource needed to bolster the resilience of domesticated strains to disease and other stresses and to allow rapid development of new, advantageous traits.

While the dependency of response to selection on genetic variation has been demonstrated theoretically and experimentally, it is much more difficult to document that natural populations have gone extinct or been locally extirpated because of a lack of adaptive potential. Yet, every extinction is an example of a population not adapting sufficiently rapidly to a changing environment -- whether that change is the presence of new predators or disease, the disappearance of preferred food resources, or any other ecological threat. Thus, debates about whether genetic variation is relevant to population viability are moot. From a conservation perspective, however, the more pressing question is whether recent reductions in levels of genetic variability in natural populations, due to human-caused destruction and fragmentation of habitats, has contributed to accelerated rates of extinction.

Although mutational meltdown is unlikely to be an imminent threat to any population, it is also unlikely that we would be able to detect the process until it had considerably and irreversibly degraded population fitness. It is not clear to me how we could demonstrate that a population decline in a natural environment was due to the accumulation of deleterious alleles and loss of previous adaptations, rather than degradation of habitat or introduction of threats to which the population had never been adapted.

POPULATION VIABILITY ANALYSIS AND THE INTERACTION BETWEEN GENETIC AND NON-GENETIC THREATS

Recently, analytical and simulation models have been used to estimate the probability of extinction and the likely time to extinction. Such "population viability analyses" (PVAs) commonly examine both deterministic impacts which depress mean population growth (the "driven declines" of Caughley, 1994) and stochastic or probabilistic processes which increase variation in population numbers (Shaffer, 1981; Boyce, 1992; Lacy, in press). The deterministic factors include those which are the familiar and all-too-common threats to biodiversity, such as over-harvest, habitat destruction, and ecological replacement by introduced competitors. The contribution of stochastic processes to destabilization of populations has only more recently been recognized (Simberloff, 1988). These stochastic processes include genetic drift, random demographic fluctuations due to the uncertainty of mating and surviving in small populations, spatial and temporal variation in environmental conditions, and local catastrophes such as disease epidemics and severe weather.

The loss of genetic variation should not be viewed in as an independent threat to population viability, but in the context of interactions with non-genetic threats (Gilpin and Soulé, 1986). Genetic instability and decline can cause demographic instability and decline, and greater susceptibility to environmental fluctuations and catastrophes. Demographic fluctuations and catastrophe-caused bottlenecks can in turn cause more genetic instability and depletion of genetic variation. Using Leslie matrix projection models that incorporated inbreeding depression, Mills and Smouse (1994) demonstrated the importance of considering the joint and interacting impacts of genetic and other factors on population viability. Thus, a distinction between genetically minimum viable populations and demographically minimum viable populations (Shaffer, 1981; Reed et al., 1993) is inappropriate.

The interactions among destabilizing processes is easier to model than to study in the field. The cumulative effects of feedbacks between genetic variation, demographic rates, and environmental effects can be simulated by

computer (Lacy, 1993b). It is much more difficult to determine, for example, whether high disease mortality is due to increased environmental stresses, lowered variability of the immune system genes, or both. The greater losses of inbred *Peromyscus leucopus* in the release-recapture study by Jiménez et al. (1994) were not directly "genetic deaths." The inbred mice had good viability in the lab, but suffered losses in the woodland habitat due to perhaps greater predation, disease, energy stress, or lower social dominance in competition for territories -- all "non-genetic" processes. The practice of excluding deaths due to accident, disease (Slatis, 1960), predation, infanticide (Bulger and Hamilton, 1988), or abandonment by parents (Caro and Laurenson, 1994) from determinations of the effects of inbreeding mistakenly assumes that inbreeding depression is manifest only in obvious congenital defects of development. The effects of genetic variation on the viability of individuals and populations is through demographic and ecological processes. Accordingly, demographic rates assessed by field biologists and the response of populations to environmental change are each modified by genetic variation both within individuals and within populations, as was illustrated by the lower survival of inbred song sparrows during a severe winter (Keller et al., 1994).

CONTINUING RESEARCH NEEDS

In spite of the abundance of data on the importance of genetic variation to the viability of domesticated and laboratory populations of mammals -- or perhaps *because* the experimental data are so compelling -- relatively few data have been collected on the effects of inbreeding on wild populations in natural habitats. To confirm that lab models are appropriate models for wildlife populations, and to determine how the effects of changes in genetic variation interact with the multiple stresses faced by animals in the wild, there is a need for more experimental studies examining the role of genetic variation on multiple aspects of fitness of individuals in natural habitats. Although the experiments would be even more difficult, there is a need also for direct tests of the effects of genetic variation on demographic performance and stability, and on rates of extinction, of populations in their natural environments. Anecdotes about populations that survived or did not survive depletion of genetic variation are not adequate; we need controlled experimental tests that can be analyzed statistically.

We need to broaden and deepen our examinations of the effects of inbreeding. Higher infant mortality has been demonstrated in many taxa of mammals, but we have few data on mate acquisition, fecundity, disease resistance, physiological response to stress, social dominance, longevity, or other components of fitness. Data on *Drosophila* and lab mice suggest that some of these components might be more influenced by genetic variability than is survival (Wright, 1977; Falconer, 1989; Hedrick and Miller, 1992; Miller and Hedrick, 1993). The mechanisms of inbreeding depression need to be explored at a more proximate level. Is poor reproduction of inbred animals due to inability to acquire mates, infertility, embryonic or fetal death, poor development of neonates, or lack of adequate parental care? Is higher mortality due to metabolic disorders, developmental defects, disease and parasite susceptibility, inability to obtain prey or to avoid becoming prey, or social conflicts?

The underlying genetic mechanisms of inbreeding depression need to be elucidated. Experimental genetics has shown that inbreeding depression can have multiple bases, including expression of deleterious recessive alleles, loss of heterozygote advantage or flexibility, and disruption of the co-evolved genetic system. Theoretical work has shown that these different mechanisms have very different evolutionary consequences, but how much each of these contributes to the genetic loads of mammalian populations has not been determined. Why do different mammalian populations have different equilibrium levels of variation? What causes populations to respond differently to inbreeding? Is the genetic load of small, isolated populations purged in a self-correcting evolutionary process, or does further inbreeding beget further inbreeding depression?

Recognizing that genetic variation can impact individual fitness and population viability, there is a need for closer monitoring of genetic variation and its effects in a number of wildlife populations. Such data will allow us to build an understanding of the frequency and ways in which changes in genetic variation are contributing to losses of biodiversity at the higher levels of populations, species, interdependent communities, and ecosystem functions. Only through genetic monitoring of threatened populations can we determine when genetic intervention would be an important component of a conservation strategy, and when it is not. Although acute genetic changes that imminently threaten populations require immediate corrective actions, we also need to understand better and then manage the long-term, cumulative changes to and impacts of genetic variation. Often the consequences of losses of variation will be delayed considerably from when the genetic changes occurred.

In theoretical work, experimental studies, and field monitoring, there needs to be greater consideration and examination of the many interactions between genetic, demographic, and environmental processes. For example, PVA models presently in use never consider more than a few of the many possible effects of genetic variation on

population viability, and often do not consider genetic effects at all (Frankham, in press b).

CONCLUSIONS

An initial flurry of books and papers in conservation biology highlighting the potential importance of genetic variation to population viability was followed by a backlash of papers doubting the role of genetic variation in the persistence of natural populations. Recent theoretical analyses, experimental verifications of theory, and field studies on natural populations (Frankham, in press b) are now providing evidence to support even the more pessimistic conclusions of earlier authors.

I have been unable to find statistically defensible evidence showing that *any* mammalian species is unaffected by inbreeding. Moreover, endangered species seem no less impacted by inbreeding, on average, than are still common taxa. More research is needed to determine if and under what circumstances populations could be purged of their genetic loads, but data do not yet allow us to presume that any mammalian population will emerge from a bottleneck with constant or recovered fitness and a greater ability to withstand future inbreeding.

Other processes that involve genetic-demographic-environmental interactions have been hypothesized to threaten population viability. The depletion of genetic variability will slow adaptive evolution, and genetic drift in small populations can lead to accumulation of maladaptive traits. Both of these processes occur so slowly that their effects on population viability would be hard to recognize. Their impacts would be seen in a reduced ability to survive in present environments and to adapt to new environments, and thus would be intertwined with non-genetic threats. While the impacts of losses of genetic variation might be slow, they would also be insidious. Once the impacts are sufficiently large as to be easily recognized, they would also be very difficult to reverse. Exchange with other populations can restore variation, but only with the risk of losing genetic variants which had been unique to the local population. When a population is the only representative of its taxon, or exchange with other populations is not possible, then reversal of genetic depletion would come about only if the population can recover to large numbers and survive the 100s to 1000s of generations needed for new mutations to restore variation.

The effects of genetic variation on population viability have received extensive theoretical treatment and experimental verification. Yet, few conservation efforts for endangered mammalian taxa recognize that ecological and anthropological threats to persistence could be magnified by interactions with the effects of depleted genetic variation. One notable exception is the Florida panther recovery program. The evidence for inbreeding, depletion of genetic variation, and their consequences on fitness was too great to ignore, and led to actions to restore genetic variability through reestablishing gene flow with western puma populations by selective translocations (Seal, 1991, 1992).

Because the contribution of genetic variation to population viability is fundamentally an interaction with physiological, behavioral, and ecological processes, conservation efforts need not necessarily target directly the components of the system that have been disrupted. A population low in variation is likely to be less resilient to other threats and less adaptable. Thus, the most productive management options might involve better preservation of natural habitats, more aggressive control of introduced exotics, and lower limits on harvest, in addition to or instead of genetic management. For example, even if there is a genetic basis to species vulnerability in the cheetah (O'Brien et al., 1985), reducing predation, eliminating disturbance by humans, and controlling disease might be effective at reversing population decline. The decimation of biodiversity has a singular predominant cause -- the over-abundance of humans -- but it is a multi-faceted problem simultaneously impinging on many levels of biotic organization. Stemming losses of biodiversity will require a diversity of conservation actions applied at many levels.

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LITERATURE CITED

Alberts, S.C., and J. Altmann. 1995. Balancing costs and opportunities: dispersal in male baboons. American Naturalist, 145:279-306.

Allendorf, F.W., and R.F. Leary. 1986. Heterozygosity and fitness in natural populations of animals. Pp. 57-86, *in* Conservation biology: The science of scarcity and diversity (M.E. Soulé, ed.). Sinauer, Sunderland, Massachusetts, 584 pp.

Ballou, J.D. 1995. Genetic management, inbreeding depression, and outbreeding depression in captive

populations. Ph.D. dissert., University of Maryland, College Park, 172 pp.
Barrett, S.C.H., and D. Charlesworth. 1991. Effects of a change in the level of inbreeding on the genetic load. Nature, 352:522-524.
Barrett, S.C.H., and J.R. Kohn. 1991. Genetic and evolutionary consequences of small population size in plants: Implications for conservation. Pp. 3-30, *in* Genetics and conservation of rare plants (D.A. Falk and K.E. Holsinger, eds.). Oxford University Press, Oxford, United Kingdom, 283 pp.
Berger, J. 1990. Persistence of different-sized populations: an empirical assessment of rapid extinctions in bighorn sheep. Conservation Biology, 4:91-98.
Bittles, A.H., W.M. Mason, J. Greene, N.A. Rao. 1991. Reproductive behavior and health in consanguineous marriages. Science, 252:789-794.
Bonnell, M.L., and R.K. Selander. 1974. Elephant seals: genetic variation and near extinction. Science, 184:908-909.
Bowman, J.C., and D.S. Falconer. 1960. Inbreeding depression and heterosis of litter size in mice. Genetical Research, 1:262-274.
Boyce, M.S. 1992. Population viability analysis. Annual Review of Ecology and Systematics, 23:481-506.
Brewer, B. A., R. C. Lacy, M. L. Foster, and G. Alaks. 1990. Inbreeding depression in insular and central populations of *Peromyscus* mice. Journal of Heredity, 81:257-266.
Bulger, J., and W.J. Hamilton III. 1988. Inbreeding and reproductive success in a natural chacma baboon, *Papio cynocephalus ursinus*, population. Animal Behaviour, 36:574-578.
Bürger, R., and M. Lynch. 1995. Evolution and extinction in a changing environment: A quantitative-genetic analysis. Evolution, 49:151-163.
Caro, T. M., and M.K. Laurenson. 1994. Ecological and genetic factors in conservation: a cautionary tale. Science, 263:485-486.
Caughley, G. 1994. Directions in conservation biology. Journal of Animal Ecology, 63:215-244.
Charlesworth, D., and B. Charlesworth. 1987. Inbreeding depression and its evolutionary consequences. Annual Review of Ecology and Systematics, 18:237-268.
Cothran, E.G., R.K. Chesser, M.H. Smith, and P.E. Johns. 1983. Influences of genetic variability and maternal factors on fetal growth in white-tailed deer. Evolution, 37:282-291.
Crow, J.F. 1948. Alternative hypotheses of hybrid vigor. Genetics, 33:477-487.
Darwin, C.R. 1868. Variation of animals and plants under domestication. Vols. 1 and 2. John Murray, London, 473+495 pp.
Darwin, C.R. 1876. The effects of cross and self fertilization in the vegetable kingdom. John Murray, London, 482 pp.
Deckard, B.S., J.R. Wilson, K. Schlesinger. 1989. Behavioral and reproductive differences in mice as a function of inbreeding. Behavior Genetics, 19:433-445.
Dudash, M.R. 1990. Relative fitness of selfed and outcrossed progeny in a self-compatible, protandrous species, *Sabatia angularis* L. (Gentianaceae): A comparison in three environments. Evolution, 44:1129-1140.
Falconer, D.S. 1989. Introduction to quantitative genetics. 3rd Ed. Longman, New York, 438 pp.
Fisher, R.A. 1958. The genetical theory of natural selection. Dover, New York, 291 pp.
Fitzsimmons, N.N., S.W. Buskirk, and M.H. Smith. 1995. Population history, genetic variability, and horn growth in bighorn sheep. Conservation Biology, 9:314-323.
Frankham, R. 1995. Inbreeding and extinction: A threshold effect. Conservation Biology, 9:792-799.
Frankham, R. In press a. Effective population size/adult population size ratios in wildlife: a review. Genetical Research.
Frankham, R. In press b. Conservation genetics. Annual Review of Genetics.
Franklin, I.R. 1980. Evolutionary changes in small populations. Pp. 135-149, *in* Conservation biology: An evolutionary-ecological perspective (M.E. Soulé and B.A. Wilcox, eds.). Sinauer, Sunderland, Massachusetts, 395 pp.
Gilpin, M.E., and M.E. Soulé. 1986. Minimum viable populations: processes of extinction. Pp. 19-34, *in* Conservation biology: The science of scarcity and diversity (M.E. Soulé, ed.). Sinauer, Sunderland, Massachusetts, 584 pp.
Goodman, D. 1987. The demography of chance extinction. Pp. 11-34, *in* Viable populations for conservation (M.E. Soulé, ed.). Cambridge University Press, Cambridge, United Kingdom.
Greenwood, P.J., P.H. Harvey, and C.M. Perrins. 1978. Inbreeding and dispersal in the great tit. Nature, 271:52-54.

Harcourt, A.H. 1995. Population viability estimates: Theory and practice for a wild gorilla population. Conservation Biology, 9:134-142.
Hedrick, P.W. 1994. Purging inbreeding depression and the probability of extinction: full-sib mating. Heredity, 73:363-372.
Hedrick, P.W. 1995. Elephant seals and the estimation of a population bottleneck. Journal of Heredity, 86:232-235.
Hedrick, P.W., and P.S. Miller. 1992. Conservation genetics: techniques and fundamentals. Ecological Applications, 2:30-46.
Hoelzel, A.R., J. Halley, S.J. O'Brien, C. Campagna, T. Arnbom, B. LeBoeuf, K. Ralls, and G.A. Dover. 1993. Elephant seal genetic variation and the use of simulation models to investigate historical population bottlenecks. Journal of Heredity, 84:443-449.
Hoogland, J.L. 1992. Levels of inbreeding among prairie dogs. American Naturalist, 139:591-602.
James, J.W. 1971. The founder effect and response to artificial selection. Genetical Research, 12:249-266.
Jiménez, J.A., K.A. Hughes, G. Alaks, L. Graham, and R.C. Lacy. 1994. An experimental study of inbreeding depression in a natural habitat. Science, 266:271-273.
Keller, L.F., P. Arcese, J.N.M. Smith, W.M. Hochachka, and S.C. Stearns. 1994. Selection against inbred song sparrows during a natural population bottleneck. Nature, 372:356-357.
Kimura, M. 1983. The neutral theory of molecular evolution. Cambridge University Press, Cambridge, 367 pp.
Lacy, R.C. 1987. Loss of genetic diversity from managed populations: Interacting effects of drift, mutation, immigration, selection, and population subdivision. Conservation Biology, 1:143-158.
Lacy, R.C. 1993a. Impacts of inbreeding in natural and captive populations of vertebrates:Implications for conservation. Perspectives in Biology and Medicine, 36:480-496.
Lacy, R.C. 1993b. VORTEX: A computer simulation model for Population Viability Analysis. Wildlife Research, 20:45-65.
Lacy, R.C. In press. What is population (and habitat) viability analysis? Primate Conservation.
Lacy, R.C. and T.W. Clark. 1989. Genetic variability in black-footed ferret populations: Past, present, and future. Pp. 83-103, *in* Conservation biology and the black-footed ferret (U.S. Seal, E.T. Thorne, M.A. Bogan, and S.H. Anderson, eds.). Yale University Press, New Haven, 302 pp.
Lacy, R.C., A.M. Petric, and M. Warneke. 1993. Inbreeding and outbreeding depression in captive populations of wild animal species. Pp. 352-374, *in* The natural history of inbreeding and outbreeding (N.W. Thornhill, ed.). University of Chicago Press, Chicago, 575 pp.
Lande, R. 1975. The maintenance of genetic variation by mutation in a quantitative character with linked loci. Genetical Research, 26:221-235.
Lande, R. 1988. Genetics and demography in biological conservation. Science, 241:1455-60.
Lande, R. 1994. Risk of population extinction from fixation of new deleterious mutations. Evolution, 48:1460-1469.
Lande, R. 1995a. Mutation and conservation. Conservation Biology, 9:782-791.
Lande, R. 1995b. Breeding plans for small populations based on the dynamics of quantitative genetic variance. Pp. 318-340, *in* Population Management for Survival & Recovery. Analytical Methods and Strategies in Small Population Conservation (J.D. Ballou, M. Gilpin, and T.J. Foose, eds.). Columbia University Press, New York, 375 pp.
Lande, R., and G.F. Barrowclough. 1987. Effective population size, genetic variation, and their use in population management. Pp. 87-123, *in* Viable populations for conservation (M.E. Soulé, ed.). Cambridge University Press, Cambridge, United Kingdom.
Lande, R., and D.W. Schemske. 1985. The evolution of self-fertilization and inbreeding depression in plants. I. Genetic models. Evolution, 39:24-40.
Ledig, F.T. 1986. Heterozygosity, heterosis, and fitness in outbreeding plants. Pp. 77-104, *in* Conservation biology: The science of scarcity and diversity (M.E. Soulé, ed.). Sinauer, Sunderland, Massachusetts, 584 pp.
Lerner, I.M. 1954. Genetic homeostasis. Oliver and Boyd, Edinburgh, Scotland.
Lynch, C.B. 1977. Inbreeding effects upon animals derived from a wild population of *Mus musculus*. Evolution, 31:526-537.
Lynch, M., and W. Gabriel. 1990. Mutation load and survival of small populations. Evolution, 44:1725-1737.
Merola, M. 1994. A reassessment of homozygosity and the case for inbreeding depression in the cheetah, *Acinonyx jubatus*: implications for conservation. Conservation Biology, 8:961-971.
Miller, P.S., and P.W. Hedrick. 1993. Inbreeding and fitness in captive populations: Lessons from *Drosophila*.

Zoo Biology, 12:333-351.
Miller, P.S. 1994. Is inbreeding depression more severe in a stressful environment? Zoo Biology, 13:195-208.
Mills, L.S., and P.E. Smouse. 1994. Demographic consequences of inbreeding in remnant populations. American Naturalist, 144:412-431.
Mitton, J.B. 1993. Theory and data pertinent to the relationship between heterozygosity and fitness. Pp. 17-41, *in* The natural history of inbreeding and outbreeding (N.W. Thornhill, ed.). University of Chicago Press, Chicago.
Modi, W.S., R.K. Wayne, and S.J. O'Brien. 1987. Analysis of fluctuating asymmetry in cheetahs. Evolution, 41:227-228.
Nei, M. 1973. Analysis of gene diversity in subdivided populations. Proceedings of the National Academy of Sciences U.S.A., 70:3321-3323.
Nevo, E. 1978. Genetic variation in natural populations: patterns and theory. Theoretical Population Biology, 13:121-177.
O'Brien, S.J. 1994a. A role for molecular genetics in biological conservation. Proceedings of the National Academy of Sciences U.S.A., 91:5748-5755.
O'Brien, S.J. 1994b. The cheetah's conservation controversy. Conservation Biology, 8:1153-1155.
O'Brien, S.J., D.E. Wildt, D. Goldman, C.R. Merril, and M. Bush. 1983. The cheetah is depauperate in genetic variation. Science, 221:459-462.
O'Brien, S.J., M.E. Roelke, L. Marker, A. Newman, C.A. Winkler, D. Meltzer, L. Colly, J.F. Evermann, M. Bush, and D.E. Wildt. 1985. Genetic basis for species vulnerability in the cheetah. Science, 227:1428-1434.
O'Brien, S.J., J.S. Martenson, C. Packer, L. Herbst, V. devos, P. Joslin, J. Ott-Joslin, D.E. Wildt, and M. Bush. 1987a. Biochemical genetic variation in geographic isolates of African and Asian lions. National Geographic Research, 3:114-124.
O'Brien, S.J., D.E. Wildt, M. Bush, T.M. Caro, C. FitzGibbon, I. Aggundey, and R.E. Leakey. 1987b. East African cheetahs: evidence for two population bottlenecks? Proceedings of the National Academy of Sciences U.S.A., 84:507-511.
Packer, C., A.E. Pusey, H. Rowley, D.A. Gilbert, J. Martenson, and S.J. O'Brien. 1991. Case study of a population bottleneck: Lions of the Ngorongoro Crater. Conservation Biology, 5:219-230.
Pettersson, B. 1985. Extinction of an isolated population of the middle spotted woodpecker *Dendrocopos medius* (L.) in Sweden and its relation to general theories on extinction. Biological Conservation, 32:335-353.
Quattro, J.M., and R.C. Vrijenhoek. 1989. Fitness differences among remnant populations of the endangered Sonoran Topminnow. Science, 245:976-978.
Ralls, K., J.D. Ballou, and A. Templeton. 1988. Estimates of lethal equivalents and the cost of inbreeding in mammals. Conservation Biology, 2:185-193.
Rao, P.S.S., and S.G. Inbaraj. 1980. Inbreeding effects on fetal growth and development. Journal of Medical Genetics, 17:27-33.
Reed, J.M., J.R. Walters, T.E. Emigh, and D.E. Seaman. 1993. Effective population size in Red-Cockaded Woodpeckers: population and model differences. Conservation Biology, 7:302-8.
Reeve, H.K., D.F. Westneat, W.A. Noon, P.W. Sherman, and C.F. Aquadro. 1990. DNA "fingerprinting" reveals high levels of inbreeding in colonies of the eusocial naked mole-rat. Proceedings of the National Academy of Sciences U.S.A., 87:2496-2500.
Ribble, D.O., and J.S. Millar. 1992. Inbreeding effects among inbred and outbred colonies of *Peromyscus maniculatus*. Canadian Journal of Zoology, 70:820-824.
Robertson, A. 1960. A theory of limits in artificial selection. Proceedings of the Royal Society (London), B, 153:234-249.
Roelke, M.E., J.S. Martenson, and S.J. O'Brien. 1993. The consequences of demographic reduction and genetic depletion in the endangered Florida panther. Current Biology, 3:340-350.
Rood, J.P. 1987. Migration and dispersal among dwarf mongooses. Pp. 85-103, *in* Mammalian dispersal patterns: The effects of social structure on population genetics (B.D. Chepko-Sade and Z.T. Halpin, eds.). University of Chicago Press, Chicago, 342 pp.
Schmitt, J., and D.W. Ehrhardt. 1990. Enhancement of inbreeding depression by dominance and suppression in *Impatiens capensis*. Evolution, 44:269-278.
Seal, U.S., and R.C. Lacy. 1989. Florida panther population viability analysis. Report to the U.S. Fish and

Wildlife Service. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.
Seal, U.S. (ed.) 1991. Genetic management considerations for threatened species with a detailed analysis of the Florida panther (*Felis concolor coryi*). Report to the U.S. Fish and Wildlife Service. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.
Seal, U.S. (ed.) 1992. Genetic management strategies and population viability of the Florida panther (*Felis concolor coryi*). Report to the U.S. Fish and Wildlife Service. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.
Selander, R.K. 1970. Behavior and genetic variation in natural populations. American Zoologist, 10:53-66.
Selander, R.K. 1983. Evolutionary consequences of inbreeding. Pp. 201-215, *in* Genetics and conservation: A reference for managing wild animal and plant populations (C.M. Schonewald-Cox, S.M. Chambers, B. MacBryde, and W.L. Thomas, eds.). Benjamin/Cummings, Menlo Park, California.
Shaffer, M.L. 1981. Minimum population sizes for species conservation. BioScience, 31:131-134.
Shields, W.M. 1982. Philopatry, inbreeding, and the evolution of sex. State University of New York Press, Albany, New York.
Shields, W.M. 1993. The natural and unnatural history of inbreeding and outbreeding. Pp. 143-169, *in* The natural history of inbreeding and outbreeding (N.W. Thornhill, ed.). University of Chicago Press, Chicago, 575 pp.
Simberloff, D.S. 1988. The contribution of population and community biology to conservation science. Annual Review of Ecology and Systematics, 19:473-511.
Slatis, H.M. 1960. An analysis of inbreeding in the European Bison. Genetics, 45:273-287.
Smith, A.T. 1993. The natural history of inbreeding and outbreeding in small mammals. Pp. 329-351, *in* The natural history of inbreeding and outbreeding (N.W. Thornhill, ed.). University of Chicago Press, Chicago, 575 pp.
Stockley, P., J.B. Searle, D.W. Macdonald, and C.S. Jones. 1993. Female multiple mating behaviour in the common shrew as a strategy to reduce inbreeding. Proceedings of the Royal Society of London, B, 254:173-179.
Templeton, A.R., and B. Read. 1983. The elimination of inbreeding depression in a captive herd of Speke's gazelle. Pp. 241-261, *in* Genetics and conservation: A reference for managing wild animal and plant populations (C.M. Schonewald-Cox, S.M. Chambers, B. MacBryde, and W.L. Thomas, eds.). Benjamin/Cummings, Menlo Park, California.
Templeton, A.R., and B. Read. 1984. Factors eliminating inbreeding depression in a captive herd of Speke's gazelle (*Gazella spekei*). Zoo Biology, 3:177-199.
van Noordwijk, A.J., and W. Scharloo. 1981. Inbreeding in an island population of the great tit. Evolution, 35:674-688.
Vrijenhoek, R.C. 1989. Genotypic diversity and coexistence among sexual and clonal lineages of *Poeciliopsis*. Pp. 386-400, *in* Speciation and its consequences (D. Otte and J.A. Endler, eds.). Sinauer, Sunderland, Massachusetts, 679 pp.
Vrijenhoek, R.C. 1994. Genetic diversity and fitness in small populations. Pp. 37-53, *in* Conservation genetics (V. Loeschcke, J. Tomiuk, and S.K. Jain, eds.). Birkhäuser Verlag, Basel, Switzerland, 440 pp.
Wayne, R.K., W.S. Modi, and S.J. O'Brien. 1986. Morphological variability and asymmetry in the cheetah (*Acinonyx jubatus*), a genetically uniform species. Evolution, 40:78-85.
Wildt, D.E., M. Bush. K.L. Goodrowe, C. Packer, A.E. Pusey, J.L. Brown, P. Joslin, and S.J. O'Brien. 1987a. Reproductive and genetic consequences of founding isolated lion populations. Nature, 329:328-331.
Wildt, D.E., S.J. O'Brien, J.G. Howard, T.M. Caro, M.E. Roelke, J.L. Brown, and M. Bush. 1987b. Similarity in ejaculate-endocrine characteristics in captive versus free-ranging cheetahs of two subspecies. Biology of Reproduction, 36:351-360.
Willig, M.R., and R.D. Owen. 1987. Fluctuating asymmetry in the cheetah: Methodological and interpretive concerns. Evolution, 41:225-227.
Willis, K., and R. Wiese. In press. Elimination of inbreeding depression from captive populations: Speke's gazelle revisited. Zoo Biology.
Wright, S. 1969. Evolution and the genetics of populations, Vol. 2, The theory of gene frequencies. University of Chicago Press, Chicago, 511 pp.
Wright, S. 1977. Evolution and the genetics of populations. Vol. 3. Experimental results and evolutionary deductions. University of Chicago Press, Chicago, 611 pp.

Clarification of Genetic Terms and their Use in the Management of Captive Populations

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Abstract

Many of the concepts, terms, and methods used in the genetic management of captive populations have been poorly defined and consequently misunderstood. The definitions and interrelationships among gene diversity, effective population size, founder genome equivalents, inbreeding, allelic diversity, mean kinship, and kinship value are presented here. It is important to understand what populations and generations are used as the baselines against which losses of genetic diversity are measured. Gene diversity and founder genome equivalents are defined relative to a source population from which founders of the captive population were randomly sampled. Inbreeding and allelic diversity are assessed relative to the founders. The "potential" gene diversity that would result from an equalization of frequencies of founder alleles retained in the population can never be achieved because, among other limitations, the random process of gene transmission will prevent equalization of allele frequencies even if animals are bred optimally. The gene diversity achievable with the population can be determined by iterative production of hypothetical offspring from the pairs with lowest mean kinship. The long-term objective for offspring production from each animal needed to achieve that potential is also thereby generated. Mean kinships should be re-calculated with each real or hypothetical birth and death, because offspring objectives based on current mean kinships might correlate poorly with the optimal long-term offspring objectives.

INTRODUCTION

The methods of pedigree analysis used to help guide the genetic management of captive populations have been evolving rapidly. While various papers have described some of the methodologies used [e.g., Ballou, 1983, 1991; Ballou and Foose, 1995; Ballou and Lacy, 1995; Boyce, 1983; DeBoer, 1988; Foose and Ballou, 1988; Lacy, 1988, 1989, 1993, 1994; Lacy et al., 1995; MacCluer et al., 1986; Thomas, 1990; Willis and Wiese, 1993], there remain aspects of pedigree analysis that have been increasingly used by AZA Species Survival Plans (SSP) but which have not been described adequately in the professional literature. In particular, due to the lack of formal definition and description of some terms and concepts, there has been confusion regarding the use of the genetic metrics calculated by the GENES computer program [Lacy, 1993]. The purpose of this note is to clarify some terms that were only poorly defined previously, and to discuss appropriate uses of some genetic metrics that have been applied to SSP management.

TERMINOLOGY AND CONCEPTS

Gene diversity

"Gene diversity" (GD), also termed "expected heterozygosity", is a common and useful measure of genetic variation within a population [Wright, 1969; Nei, 1973; Willis and Wiese, 1993]. GD is the variance in allele frequencies at a genetic locus, and is equal to the heterozygosity expected in a population with random union of gametes (i.e., in Hardy-Weinberg equilibrium). For a single genetic locus, GD is defined as:

$$GD = 1 - \Sigma(p_i^2), \qquad \text{Eq. 1}$$

in which p_i is the frequency of allele i, and the summation is over all alleles at that locus. GD reflects both the number of alleles and the evenness of their frequencies. GD can be applied to describe variation at a single locus, as above, or it can be averaged over loci to provide a genome-wide metric of genetic variation. In the absence of new variation introduced to a population by mutation or immigration, in the absence of natural selection, and in a randomly mating population of constant size, GD decays due to genetic drift according to:

$$GD_t = GD_0 \times [1 - 1/(2N)]^t, \qquad \text{Eq. 2}$$

in which the subscripts denote generations, and N is the number of individuals in each generation. The decay of

GD due to genetic drift is a result of the sampling of the gene pool that occurs when each generation is produced from the previous generation.

The rate of decay of GD is independent of initial level of gene diversity and of the allele frequencies at the loci. Therefore, it is often useful to represent the genetic diversity of a population by the proportional gene diversity, relative to some baseline generation. The proportional gene diversity (GD_t / GD_0) is a widely used metric in the management of captive breeding programs, although frequently it is called "gene diversity", with the unstated implication that it is proportional to a baseline population. For example, Willis and Wiese [1993] assumed $GD_0 = 1$, so that absolute and proportional gene diversity were the same and were symbolized interchangeably as GD in their equations. Usually, GD_0 of the baseline population is not known. To obtain GD_0 for a single locus would require random sampling of individuals from that baseline population; to obtain GD_0 averaged across the genome would require random sampling of genetic loci as well.

Although GD_0 is not needed to determine GD_t/GD_0, it is essential that the baseline used in each analysis be explicitly understood. For example, the GENES program calculates GD of a captive population *relative to GD_0 of the possibly hypothetical population from which the founders were randomly sampled.* This is not the same as the GD of the founders themselves, because the founders are a (usually small) sample of a larger population (for example, in the wild). The founders will therefore contain only a subset of the GD of the population from which they were sampled. The baseline GD_0 is also not necessarily the same as GD of the entire wild population, because often the founders of a captive stock were sampled non-randomly from a subset of the range of the taxon. Thus, GD_0 of the true source population might be less than GD of the entire wild population. The sampling of founders from the wild is analogous to the production of an offspring generation, and the GD of the founders is therefore reduced relative to the population from which they were sampled according to Eq. 2, with N being the number of founders and $t = 1$, and with the assumption that the founders were a random sample of the source population [Lacy, 1988, 1994; Willis and Wiese, 1993].

Effective population size

The above discussion assumed that the genes transmitted to each generation are a random sample of the genes of the previous generation. If individuals do not breed randomly, then the rate of loss of GD might be slower or (much more commonly) faster than predicted from Eq. 2. The genetically "effective population size" (N_e) has multiple related meanings [Crow and Kimura, 1970; Crow and Denniston, 1988], but the form most commonly used in the design of captive breeding programs is the "variance effective size." The variance N_e is defined as the size of an idealized population, with random union of gametes each generation, which would have the same inter-generational variance in allele frequencies as does the studied population. Thus, the variance effective population size is the number N that must be used in Eq. 2 to yield the observed rate of loss of GD. It is because captive breeding programs are often directed toward minimizing the loss of GD that the variance effective size is appropriate. (For example, it is the one that should be used in the CAPACITY computer program [Ballou, 1992] for estimating the required population size to achieve a desired low rate of loss of GD.) The effective population size is reduced relative to the census population if there is greater variance among individuals in reproduction than expected by chance, as would happen if some individuals were particularly successful breeders, or if there are not equal numbers of male and female breeders [Crow and Kimura, 1970; Lande and Barrowclough, 1987]. Moreover, most species have overlapping generations, so that a portion of the existing population consists of the present generation breeders, a portion consists of the future breeders of the next generation, and a portion may consist of post-reproductive individuals from the previous generation. Only the present-generation breeders contribute to the effective population size (of their generation).

Founder genome equivalents

The concept of founder genome equivalent (FGE) was introduced by Lacy [1989] as "that number of equally contributing founders with no random loss of founder alleles in descendants that would be expected to produce the same genetic diversity as in the population under study". FGE provides another metric of the amount of genetic variation in a captive population, expressed in units of randomly sampled founders. Like GD_t/GD_0, FGE is relative to a baseline, in that it is the number of founders drawn at random from a defined source population which would contain the observed amount of gene diversity. Two populations with the same FGE may have very different gene diversities, if the source populations differed in genetic variation. I originally [Lacy, 1989] defined FGE by the equation:

$$FGE \equiv 1 / \Sigma(p_i^2/r_i) \quad \text{Eq. 3}$$

in which p_i is the proportion of genes in the descendant population that are contributed by founder i, and r_i is the proportion of founder i's alleles that have been retained within the descendants. In that paper, I pointed out that FGE, defined in this way, provides a means of approximating the loss of GD, in that

$$GD_t/GD_0 \approx 1 - 1/(2\ FGE). \quad \text{Eq. 4}$$

This approximation is quite accurate for large pedigrees, but can deviate by about 1% to 5% for pedigrees of fewer than about 10 individuals (for example, the hypothetical pedigrees in Lacy [1989]). However, the pedigree analysis method of mean kinship, developed subsequently, provides a method of calculating exactly the proportional loss of GD expected in a pedigree [Ballou, 1991; Lacy, 1994; Ballou and Lacy, 1995]. Therefore, it is logical to use this precise method to quantify losses of GD, and to determine and define FGE by:

$$FGE \equiv 0.5 / [1 - (GD_t / GD_0)], \quad \text{Eq. 5}$$

so that the relationship between GD and FGE in Eq. 4 holds exactly. With this modified definition of FGE, more precisely representing the original concept, Eq. 3 is now only approximate. The GENES program calculates FGE by first calculating GD_t/GD_0, by either the mean kinship method or by a "gene drop" simulation [MacCluer et al., 1986], and then applying Eq. 5. GENES does not use the approximate method described in Lacy [1989].

The GENES program normally calculates metrics summarizing the genetic variation present in a captive population based only on the genetic variation present *within the captive born descendants of the founders*. (An undocumented option of GENES version 11.30 and later versions is that the program will produce metrics summarizing the genetic variation present in all living animals within the population -- including living founders -- if the flag 'F' is appended to the command line: e.g., GENES STUDBOOK F). It is important to exclude founders themselves from tallies of genetic variation within a captive population [Lacy, 1989, 1994] in order to ensure that the genetic metrics reflect the progress of the captive propagation program and not simply the number of (possibly non-breeding) animals collected from the wild. Thus, adding a new wild-caught animal to a captive population does not change the GD, the FGE, or the number of founders reported by GENES. That animal remains a "potential founder" until it contributes genes (in the form of offspring) to the captive-born population. When it breeds in captivity, the number of founders increases by one, and GD and FGE will also increase.

FGE and N_e share some similarities as methods of quantifying the loss of genetic diversity from a population. FGE represents the *cumulative* loss of GD since the baseline generation as the number of founders that would contain that amount of gene diversity (Eq. 5). The variance N_e represents the *per generation rate* of loss of GD as the number of randomly breeding animals that would lose GD at the observed rate (Eq. 2). If N_e is moderately large (on the order of 10 or more), then Eq. 2 becomes approximately

$$GD_t/GD_0 \approx 1 - t/(2N_e), \quad \text{Eq. 6}$$

so that, combining with Eq. 4,

$$FGE \approx N_e/t. \quad \text{Eq. 7}$$

For example, if a randomly breeding population of 20 founders produces 20 offspring, which in turn produce 20 grand-offspring ($N_e = N = 20$ each generation), then the percent of GD retained would be (by Eq. 2) GD_t/GD_0 = 97.5%, 95.1%, 92.7%, and 90.4% in the founders, first, second, and third generation descendants, and FGE = 20, 10.1, 6.8, and 5.2, respectively (by Eq. 5).

Inbreeding

Inbreeding is the mating between animals that are related by descent from a common ancestor, *subsequent to a defined baseline generation*. The baseline generation for inbreeding calculations is normally taken as the *founders* of the population, which are defined arbitrarily to be non-inbred and unrelated to each other. If more distant ancestors are known (for example, kinships among animals from the wild can be determined by molecular genetic methods -- Geyer, et al. [1993]), then an earlier baseline can be defined and inbreeding coefficients might be greater. Thus, whether an animal is inbred is dependent upon the baseline chosen, but earlier baselines can only increase the quantified level of inbreeding, by revealing additional shared ancestors in the pedigree. The first descendant generation of sexually reproducing, non-hermaphroditic species can contain animals that are inter-related, but cannot contain inbred animals, because (by definition) their founder parents were unrelated. Thus, the second generation descendants are the first animals that can be considered to be inbred. The inbreeding coefficient (or coefficient of consanguinity), F, in a pedigree is

commonly taken to be the probability that the two alleles at homologous loci in the individual are identical by descent from a common ancestor of the parents [Wright, 1922, 1969; Jacquard 1975]. Inbreeding reduces average heterozygosity, therefore, by a proportion F. (Because random genetic drift also reduces heterozygosity, the rate of loss of GD -- "expected heterozygosity" -- due to drift is at times quantified by an "inbreeding" coefficient, with $F = 1 - GD_t/GD_0$, even though there may have been no inbreeding in the sense of matings between relatives [Templeton and Read, 1994]).

Gene diversity is the heterozygosity expected if breeding is at random, so it might seem problematic that GD in the founders is less than GD_0, and GD decreases with each generation, but it is not until the second generation of captive-born animals that there can be any inbreeding or individuals homozygous for founder alleles. This conflict is resolved, however, because the baseline generation used in inbreeding calculations is the founders while the baseline for GD calculations is the (wild) parental population of the founders. Moreover, the "random breeding" assumed in equating GD with heterozygosity includes the possibility that an animal will mate with itself. Hermaphroditic animals can be inbred in the first generation, if one founder was both their sire and dam. In non-hermaphroditic animals, the requirement of bisexual reproduction causes irregular accumulation of inbreeding in early generations of random breeding, while GD declines regularly according to Eq. 2.

Inbreeding is relative in another sense: Inbreeding is a scalar quantity, not a categorical quality. It makes little sense to consider (as is often done) animals with inbreeding coefficients of, say, F = 0.0156 (from second-cousin matings), F = 0.0625 (from first-cousin matings), and F = 0.25 (from full-sib matings) to be a distinct class of "inbred" animals to be contrasted with "non-inbred" animals (F = 0.00). Animals with F = 0.0625 would, with respect to heterozygosity and various measures of fitness, be much more similar to animals with F = 0.00 than to animals with F = 0.25. The concern with low levels of inbreeding in managed pedigrees should not be with immediate loss of fitness (which would be undetectably small, especially in a captive environment), but with the cumulative aspect of inbreeding. Unless reversed by later outcrossing to less related animals, an incremental increase of, say, 0.03 per generation would cause an unobservable incremental loss of genetic variation and fitness which could within about four generations lead to a damaging cumulative loss of fitness in the population. Breeding programs designed to sustain a healthy population without further import of new founders for 100 or more years should keep incremental increases in inbreeding and losses of gene diversity to minimal levels.

Allelic diversity

The loss of allelic variants due to genetic drift is much more difficult to predict, because the rate of loss is dependent upon the initial allele frequencies [Fuerst and Maruyama, 1986; Lacy, 1994]. Rarer alleles are more likely not to be transmitted to any offspring in the next generation than are more common alleles. To model the loss of alleles, a common practice in "gene drop" simulations is to assume that each founder carries two unique alleles at each genetic locus [MacCluer et al., 1986]. This is the limiting case, with each allele initially present in just one copy, and it will lead to the most rapid loss of allelic diversity. Thus, gene drop analyses, such as the one in the GENES program, assume a baseline of the *founder* generation, and assess the probability that an allele unique to a founder would be lost in subsequent generations. If an earlier generation were chosen as the baseline or, equivalently, the founder generation were assumed not to contain all unique alleles, then the rate of loss of allelic diversity would be less. This dependency on initial conditions can make interpretation of metrics of allelic diversity difficult.

The assumption of all unique founder alleles in a gene drop simulation facilitates calculation of several other metrics of gene diversity. First, the frequency, over many iterations of the simulation, of an individual being homozygous at the modeled locus will be equal to that animal's inbreeding coefficient, as both are measures of the probability of homozygosity by descent since the baseline, founder generation. (Homozygosity due to identical alleles descending from unrelated ancestors is excluded by the assumption that all founder alleles are unique.) Second, GD calculated from the allele frequencies of the founders ($GD = 1 - \Sigma[1/(2N)]^2 = 1 - [1/(2N)]$, in which N is the number of founders) gives a standard measure of the "founder effect", or loss of GD in the founders relative to the source population from which they came [Willis and Wiese, 1993; Lacy, 1994]. The GD of the source population must be considered to be 1.0 (i.e., it consisted of an infinite number of heterozygous and genetically unique individuals), in order for the founders to all have been (by assumption) heterozygous. With the assumption that the source (wild) population had $GD_0 = 1$, $GD_t/GD_0 = GD_t$, leading to common confusion regarding the distinction between absolute and relative GD.

Mean kinship and kinship value

Mean kinship (MK), the average coefficient of kinship of an animal to each living, non-founder animal in a pedigree (including itself, if it is not a founder), can be a useful metric for summarizing the genetic value of an animal to a breeding program [Ballou, 1991; Lacy, 1994; Ballou and Lacy, 1995]. The overall mean kinship of a population (the mean MK) equals 1 - GD_t/GD_0: both are the probability that two alleles sampled at random from homologous loci in the population will be homozygous by descent from a common ancestor. Breeding the animals with lowest MK will necessarily maximize GD in the next generation, as it ensures that the founder alleles with lowest frequency are preferentially propagated. Simulations have shown that a program of breeding animals based on choosing those with lowest MK is a better strategy for maximizing long-term preservation of gene diversity and allelic diversity, and avoiding future inbreeding, than is minimizing immediate inbreeding, equalizing founder contributions, or breeding animals most likely to contain unique alleles [Ballou and Lacy, 1995].

A complication with the use of MK to select breeders is that each offspring added to a pedigree (or dead animal removed from a pedigree) changes the matrix of kinships in the population and changes the MK of every animal related to that new (or departed) animal. Thus, for selecting breeders, MKs should be used and recalculated iteratively, with the kinship matrix [Ballou, 1983] updated with the addition of a hypothetical offspring each time a mating is selected. The GENES program allows users to do such iterative selection of breeders based on MKs.

Not all individuals in a population are equally likely to survive and produce future offspring. Therefore, the probable future distribution of allele frequencies in a population might be skewed relative to the present distribution. In the extreme case, alleles unique to post-reproductive or otherwise sterile animals are certain to be lost. Animals in the prime of their reproductive lifespans have high probabilities of contributing many more copies of their genes to the population. Therefore, better genetic management might be achieved by considering the shifts in the gene pool of the population that will likely occur due to the age structure of the population. Top priority for breeding should be given to those animals who have the least commonality of genes with the probable next generation.

A modification of mean kinship, kinship value (KV), has been proposed but to date rather little used, to adjust breeding priorities for the reproductive potential of each animal in the present generation [Ballou and Lacy, 1995]. The KV of an animal is the weighted mean kinship of the animal to each living, non-founder, with the weights being the reproductive value of each kin entered into the calculation. Reproductive value is age-specific and is defined as the expected future lifetime reproduction [Fisher, 1930]. Reproductive values are determined from analysis of age-specific fecundity and mortality rates. It is important to note that KV is not the MK multiplied by an animal's own reproductive value, but rather is a mean weighted by the reproductive values of all kin to that animal. Thus, the kinships of post-reproductive kin do not contribute to an animal's KV, and an animal with mostly aged kin would likely be a priority breeder. An animal with many kin of young breeding age would have its KV elevated relative to its MK, and would therefore not likely be a priority breeder.

Just as the 1 - mean MK of a population predicts the heterozygosity of the next generation (relative to the baseline generation) expected if breeding is at random, 1 - the mean KV predicts the heterozygosity of the next generation if all animals produce the number of future offspring expected based on the population fecundity and mortality rates. One minus the mean KV of a population has therefore been termed the "gene value" of the population in the GENES program, in reference to it being a modification of gene diversity to account for reproductive values.

MANAGEMENT ISSUES

How potential are potential founders, FGEs, and GD?

The gene drop analysis performed by the GENES program provides summary statistics not only on the living captive-born population as it exists when the analysis was run, but also on the "potential" population that could be derived from the existing captive population if there were perfect genetic management in the future. These metrics of the potential population can be useful in providing an indication of the scope for genetic improvement through wise genetic management, in the absence of any further import of newly wild-caught animals. This scope for genetic improvement provides a target for the future, an indication of the need or lack thereof for new wild-caught animals to achieve genetic objectives, and an upper ceiling to future genetic variation in the absence of further imports. It is important, therefore, to consider how closely and under what circumstances the "potential" can be reached.

Potential founders are already in the captive collection, and the probability of their ever contributing progeny should be assessed by consideration of their age, health, behaviors, responses to past breeding opportunities, and the biology and history of captive propagation for the species. For each potential founder that is lost, the potential FGE is decreased by 1. The resulting decrease in potential GD depends on the number of founders (and can be determined from Eq. 4), as each additional founder contributes proportionately less to the acquisition and preservation of GD [Lacy, 1994].

The potential FGE and GD that is actually achievable will be reduced further for several reasons. The potential values reported by the GENES program assume that all founder and potential founder alleles still existing in the captive population will be brought to equal frequencies through genetic management. (For a given set of alleles, GD is maximized if all allele frequencies are equal: Eq. 1.) As is well known, however, even if managers plan the optimal breeding program, rarely if ever do all animals that are paired produce exactly the number of offspring that are desired from them. Not only might some not breed, or not produce the numbers of progeny that are desired, but unequal numbers of males and females in the population will diminish the effectiveness of the breeding program in preserving GD. The "potential" FGE and GD given by the GENES program assumes that any animal can mate with any animal, regardless of sex. Moreover, even if all animals do produce exactly the desired number of offspring, we still cannot control which of the two homologous genes at each locus are transmitted to each offspring. Thus, the mechanisms of Mendelian genetics include a random sampling process that invariably adds variation to the allele frequencies in future generations. For example, even if contributions are kept equal among founders, the frequencies of the two alleles contributed by each founder will deviate randomly. Therefore, it should be recognized that the "potential" values given by the analyses in the GENES program can never, even in theory, be achieved.

It is possible to determine how close to the "potential" FGE and GD it may be possible to reach, if managers plan the perfect breeding program and animals all reproduce as desired, but allelic transmission is still random and animals are constrained to breed only with the opposite sex. GENES provides the option of testing the genetic effects of producing future offspring from any desired mating. By iterating this process to produce 100s or 1000s of offspring, always selecting the most genetically valuable pair (those with the lowest MK) as parents, the theoretically achievable FGE and GD can be generated. (GENES version 11.40 restricts the number of hypothetical offspring to 1000, but GD and FGE usually largely level off at a asymptotic functional maximum within fewer pairings. GENES does allow the breeding of same-sex pairs or even selfing to produce hypothetical offspring, but such breeding is not possible except in hermaphroditic organisms.)

Table 1 shows for two SSPs, okapi (*Okapia johnstoni*) and Goeldi's monkey (*Callimico goeldii*), the metrics summarizing the genetic variation in the existing SSP population as of late 1994, the "potential" reported by GENES if all founder alleles still retained within the population were brought to equal frequency, and the theoretically achievable levels of genetic variation as determined by the production of 1000 optimal offspring. Potential values of genetic variation were obtained from a gene drop simulation with 100,000 iterations. Matings to produce optimal offspring were constrained to be between a male and female, but no restrictions were placed on the numbers of progeny that could be produced from any pair. The okapi population has received recent imports of three new founders that are not yet well represented, so founder allele frequencies are presently quite unequal. Through good genetic management it might be possible to reach levels of GD and FGE that are close to those reported as "potential" by GENES. Goeldi's monkey have a large, complex pedigree, with no living founders, and presently more equal representation of founder alleles. Only about one-third of their "potential" increase in GD and FGE could be recovered even with optimal genetic management.

Lifetime offspring objectives for breeders

Planning of breeding programs often involves determination of the number of offspring to be desired from each breeder in the population. Such offspring objectives can be valuable aids in determining how to assign males to females as pairs for breeding, whether it is worth the moving an animal to a new social group or institution for future breeding, and how to space breeding attempts over time. (MKs and KVs assign priority to males and females for breeding, but do not necessarily identify which males to put with which females. However, pairing animals of approximately equal MK will minimize the need for future rearrangement of pairings and will also facilitate selection of valuable breeders in future generations [Lacy, 1994].) Various methods have been employed by SSPs in the past to try to determine desired lifetime offspring objectives. Most

attempts can be described as guesses about how far down a list of MKs (or some other prioritizing metric) we might desire fewer (or no) progeny.

MKs (or KVs, if one wishes to adjust for expected future reproduction by kin) can be used to assign offspring objectives to each animal, but not in the manner that has typically been employed. Because MKs are dynamic, changing with each birth or death in the population, it is not adequate to use a static list of MK to set lifetime offspring objectives. For example, a large cohort of siblings may all have high MK (because they have many kin) and therefore be assigned low or no breeding priority. However, after some of that cohort die, or non-kin produce many offspring, the MK of each of the remaining siblings in the cohort will decrease and their priority for future breeding will rise. Similarly, two siblings of an otherwise unrepresented founder pair may both have low MK and be given high priority for breeding. As soon as one produces progeny, however, the MK of both will increase (although the MK of the breeding sib will increase more), and the future priority for breeding either sib will decline.

Unfortunately, because of the complexities of kinship relationships in many pedigrees, it is not appropriate to use a static MK list to determine relative offspring objectives for optimal future breeding. Optimal long-term breeding programs (those that always select breeders with the lowest continuously updated MK) may produce more offspring from some animals of higher current MK than from other animals with greater present breeding priority. The GENES program (version 11.40) provides an option of automatically generating a list of up to 1,000 matings, and tallying the number and frequency of use of each breeder in those planned matings.

Table 2 shows the current MK, and the optimal number of progeny for okapis, with the assumptions that 10, 100, or 1000 progeny are desired. Although MK gives an approximate ranking of the number of offspring to be desired from each okapi, the rank order of present-day MK does not match the order of offspring objectives. Moreover, the first 10 optimal offspring do not give a good indication of the longer range offspring objectives, nor does use in the first 100 matings predict accurately the frequency of use over the first 1,000 matings. Animals chosen more often as breeders are not always chosen for the first time sooner than animals with lower long-term offspring objectives.

Much of the discordance between present MK rankings and long-term offspring objectives derived by iterative use of MK results from the low utilization as breeders animals who have living parents. Among the okapi, none of the animals with both parents still in the population were chosen in the next 1,000 matings. This is a common, but not inevitable, consequence of choosing matings based on iterative MKs. A male okapi (studbook # 259) with the highest present MK but neither parent living was chosen for 11% of the future matings, while some of his sons (studbook # 392 and 414), with much lower present MKs, were never chosen. This phenomenon, of animals with living parents rarely being chosen for breeding, results from progeny containing no genes not present in their parents (but containing only a subset of those genes). When the parents die, the reduced MKs of the progeny immediately give them higher priority for breeding. A secondary consequence is that generation time will be extended, as animals are kept as breeders until they die or become physiologically post-reproductive.

Although generating hypothetical matings provides a method for estimating long-term offspring objectives, it would be counter-productive to use such offspring objectives, rather than the dynamic ranked list of MK, to select breeders in the short term. Animals that are presently low on the MK ranking, but who are assigned large proportions of future offspring (e.g., okapi # 259), will become optimal future breeders only if and when animals with present priority for breeding have produced a number of offspring. Optimal retention of gene diversity is achieved by always selecting breeders with the lowest MK, while constantly updating the MK calculations to adjust for new progeny, deaths, or animals otherwise removed from the potential breeding population.

CONCLUSIONS

While training in the management of populations and increased use in SSPs has over the past decade produced considerable understanding and familiarity with genetic concepts among zoo animal managers, some of the details of genetic metrics and techniques in use have been unavailable, overlooked, or misused. SSPs and other breeding programs set goals of maintaining prescribed levels of original genetic diversity, so it is important to understand and specify the baseline population that defines the goals to which we aspire. The precise meaning of metrics of genetic variation must be clear if we are to use quantitative methods to plan breeding programs and to assess progress. Iterative procedures can be used to project the genetic improvement

possible through pedigree management, and can provide long-term breeding objectives for animals in the population. Intuitive guesses about the priority and timing of breeding of animals for optimizing genetic variation can yield poor results.

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REFERENCES

Ballou, J. Calculating inbreeding coefficients from pedigrees. Pp. 509-520 in GENETICS AND CONSERVATION: A REFERENCE FOR MANAGING WILD ANIMAL AND PLANT POPULATIONS. C.M. Schonewald-Cox; S.M. Chambers; B. MacBryde; W.L. Thomas, eds. Menlo Park, California, Benjamin/Cummings, 1983.

Ballou, J.D. Management of genetic variation in captive populations. Pp. 602-610 in THE UNITY OF EVOLUTIONARY BIOLOGY, FOURTH INTERNATIONAL CONGRESS OF SYSTEMATICS AND EVOLUTIONARY BIOLOGY. E.C. Dudley, ed. Portland, Oregon, Dioscorides Press, 1991.

Ballou, J.D. CAPACITY 3.0. Washington, DC, National Zoological Park, 1992.

Ballou, J.D.; T.J. Foose. Demographic and genetic management of captive populations. In WILD MAMMALS IN CAPTIVITY. D.G. Kleiman; S. Lumpkin; M. Allen; H. Harris; K. Thompson, eds. Chicago, University of Chicago Press, in press.

Ballou, J.D.; Lacy, R.C. Identifying genetically important individuals for management of genetic diversity in pedigreed populations. In POPULATION MANAGEMENT FOR SURVIVAL AND RECOVERY. J.D. Ballou; T. Foose; and M. Gilpin, eds. New York, Columbia University Press, in press.

Boyce, A.J. Computation of inbreeding and kinship coefficients on extended pedigrees. JOURNAL OF HEREDITY 74:400-494, 1983.

Crow, J.F.; Denniston, C. Inbreeding and variance effective population numbers. EVOLUTION 42:482-495, 1988.

Crow, J.F.; Kimura, M. AN INTRODUCTION TO POPULATION GENETICS THEORY. New York, Harper and Row, 1970.

DeBoer, L.E.M. Genetics and breeding programs. In EEP COORDINATORS MANUAL. Amsterdam, Netherlands, National Foundation for Research in Zoological Gardens. 1988.

Fisher, R.A. THE GENETICAL THEORY OF NATURAL SELECTION. London, Oxford University Press, 1930.

Foose, T.J.; Ballou, J.D. Population management: Theory and practice. INTERNATIONAL ZOO YEARBOOK 27: 26-41, 1988.

Fuerst, P.A.; Maruyama, T. Considerations on the conservation of alleles and of genic heterozygosity in small managed populations. ZOO BIOLOGY 5:171-180, 1986.

Geyer, C.J.; Ryder, O.A; Chemnick, L.G.; Thompson, E.A. Analysis of relatedness in California condors, from DNA fingerprints. MOLECULAR BIOLOGY AND EVOLUTION 10:571-589, 1993.

Jacquard, A. Inbreeding: One word, several meanings. THEORETICAL POPULATION BIOLOGY 7:338-363, 1975.

Lacy, R.C. Genetic variability in captive stocks: Assessing past loss, present status, and future outlook. AAZPA 1988 ANNUAL PROCEEDINGS 113-121, 1988.

Lacy, R.C. Analysis of founder representation in pedigrees:Founder equivalents and founder genome equivalents. ZOO BIOLOGY 8:111-124, 1989.

Lacy, R.C. GENES: A COMPUTER PROGRAM FOR THE ANALYSIS OF PEDIGREES AND GENETIC MANAGEMENT OF POPULATIONS. Brookfield, Illinois, Chicago Zoological Society, 1993.

Lacy, R.C. Managing genetic diversity in captive populations of animals. Pages 63-89 in RESTORATION AND RECOVERY OF ENDANGERED PLANTS AND ANIMALS. M.L. Bowles; C.J. Whelan, eds. Cambridge, UK, Cambridge University Press, 1994.

Lacy, R.C.; Ballou, J.D.; Princée, F.; Starfield, A.; Thompson, E. Pedigree analysis. In POPULATION MANAGEMENT FOR SURVIVAL AND RECOVERY. J.D. Ballou; T. Foose; and M. Gilpin, eds. New York, Columbia University Press, in press.

Lande, R.; G.F. Barrowclough. Effective population size, genetic variation, and their use in population management. Pages 87-123 in VIABLE POPULATIONS FOR CONSERVATION. M.E. Soulé, ed. Cambridge, Cambridge University Press, 1987.

MacCluer, J.W.; VandeBerg, J.L.; Read, B.; Ryder, O.A. Pedigree analysis by computer simulation. ZOO BIOLOGY 5:147-160, 1986.

Nei, M. Analysis of gene diversity in subdivided populations. PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES USA 70:3321-3323, 1973.

Templeton, A.R.; Read, B. Inbreeding: One word, several meanings, much confusion. Pages 91-105 in CONSERVATION GENETICS. V. Loeschcke; J. Tomiuk; and S.K. Jain, eds. Boston, Birkhäuser Verlag, 1994.

Thomas, A. A comparison of an exact and a simulation method for calculating gene extinction probabilities in pedigrees. ZOO BIOLOGY 9:259-274, 1990.

Willis, K; Wiese, R.J. Effect of new founders on retention of gene diversity in captive populations: A formalization of the nucleus population concept. ZOO BIOLOGY 12:535-548, 1993.

Wright, S. Coefficients of inbreeding and relationship. AMERICAN NATURALIST 56:330-338, 1922.

Wright, S. EVOLUTION AND THE GENETICS OF POPULATIONS, Vol. 2, THE THEORY OF GENE FREQUENCIES. Chicago, University of Chicago Press, 1969.

TABLE 1. Number of founders (N_f), founder genome equivalents (FGE), and percent of original gene diversity (%GD) in SSP populations of okapi and Goeldi's monkey at present, the potential if all founder alleles still retained in the populations were to be brought to equal frequencies, and achievable after 1000 hypothetical offspring are added to the population.

	Okapia johnstoni[a]			*Callimico goeldii*		
	Present	"Potential"	after 1000 matings	Present	"Potential"	after 1000 matings
N_f	25	25	25	22	22	22
FGE	6.0	11.4	10.4	10.0	16.0	11.7
%GD	91.7	95.6	95.2	95.0	96.9	95.7

[a] Metrics calculated after omitting genes descended from an unknown, non-founder ancestor.

TABLE 2. Present Mean Kinships; frequency of use in 10, 100, or 1000 matings; and first mating used for *Okapia johnstoni* in the SSP.

♂♂[a]	Sire[b]	Dam[b]	MK[c]	Matings out of 10	100	1000	1st use
389	358	372	.017	5	26	200	1
391	WILD	381	.017	4	26	200	2
299	219	273	.035	1	14	108	9
283	219	249	.057	0	10	106	30
933	331	315	.064	0	0	0	
931	391	397	.064	0	0	0	
392	259	348	.065	0	0	0	
414	259	348	.065	0	0	0	
369	283	317	.072	0	0	0	
247	58	139	.073	0	11	112	27
338	283	275	.082	0	2	70	72
325	214	253	.082	0	1	21	73
408	331	351	.098	0	0	0	
331	259	216	.099	0	5	73	48
416	338	386	.103	0	0	0	
411	338	333	.103	0	0	0	
377	338	333	.103	0	0	0	
936	338	342	.105	0	0	0	
412	259	272	.107	0	0	0	
934	259	272	.107	0	0	0	
259	172	153	.107	0	5	110	62

♀♀[a]	Sire[b]	Dam[b]	MK[c]	Matings out of 10	100	1000	1st use
348	WILD	WILD	.011	4	26	202	1
315	WILD	WILD	.017	4	24	201	2
292	257	248	.022	2	19	152	4
390	247	315	.051	0	0	0	
908	247	315	.051	0	0	0	
929	389	351	.057	0	0	0	
253	198	160	.061	0	16	144	24
317	247	275	.076	0	6	67	43
906	283	368	.082	0	0	0	
336	214	253	.084	0	0	22	133
932	283	396	.084	0	0	0	
351	214	253	.085	0	0	21	162
383	331	253	.086	0	0	0	
368	247	313	.094	0	0	0	
417	310	313	.095	0	0	6	270
272	214	196	.096	0	5	110	66
313	259	216	.098	0	4	69	56
396	310	313	.098	0	0	6	371
397	331	336	.100	0	0	0	
907	325	378	.101	0	0	0	
935	338	386	.103	0	0	0	
398	338	342	.105	0	0	0	
378	338	342	.108	0	0	0	
333	259	272	.113	0	0	0	
386	259	272	.113	0	0	0	
342	259	272	.117	0	0	0	

[a] Studbook numbers greater than 900 are temporary numbers for recent births.

[b] Dead parents underlined.

[c] MK calculated after omitting portions (up to 12.5%) of the genome descended from an unknown ancestor.

DEMOGRAPHY OF THE LION-TAILED MACAQUE (*MACACA SILENUS*) IN THE WILD

AJITH KUMAR[1] & G.U.KURUP[2]

INTRODUCTION

The objective of this paper is to give a comprehensive description of the demographic parameters of the lion-tailed macaque in the wild, especially those which are of relevance to Population and Habitat Viability Analysis using VORTEX. There has been several reports on the distribution and population status of the lion-tailed macaque (Green & Minkowski 1977; Kurup 1978; Ali 1985; Bhat this vol; Kumar in prep.), and two long term studies on its ecology and behaviour (Green & Minkowski 1977; Kumar 1987; Kurup & Kumar 1993). There has been, however, only one study on the demography (Kumar 1987). Most of the demographic data presented here, therefore, come from this study which was carried out in the Indira Gandhi Wildlife Sanctuary in Tamil Nadu (previously Anamalai Wildlife Sanctuary) during 1978-84. Some data on group composition is also taken from a survey of the lion-tailed macaque carried out in 1987-90 (Kumar in prep.). The first part of the study in the Indira Gandhi Wildlife Sanctuary (1978-80) was carried out with funding from the Zoological Survey of India, and the second part (1982-84) with funding to the first author from WWF-US, Wenner-Gren Foundation for Anthropological Research, and WWF-India. This part was carried out with Dr.David J. Chivers of the Cambridge University. The survey of the lion-tailed macaque in 1987-90 was carried out with funding to the first author from the Wildlife Conservation International, New York Zoological Society.

During 1978-84, 5 groups of lion-tailed macaque were censused at least once in a month to monitor births and disappearances (which were assumed to have died). Between April 1980 and March 1982, however, only one census was carried out, in April 1981. One of five the groups (AS) fissioned in 1981-82 into two groups which were considered as two groups (ASI & ASII) from 1982 onwards. All the groups were within 10km radius of the Varagaliyar Elephant Camp in the Indira Gandhi Wildlife Sanctuary (Figure 1). Varagaliyar was also the field camp site during 1978-84. Ecological and behavioral studies were carried out on the group closest to the camp (VSI), for 9 months in 1978-80 and for 15 months in 1982-84. Details of the study site and methodology are given elsewhere (Kumar 1987).

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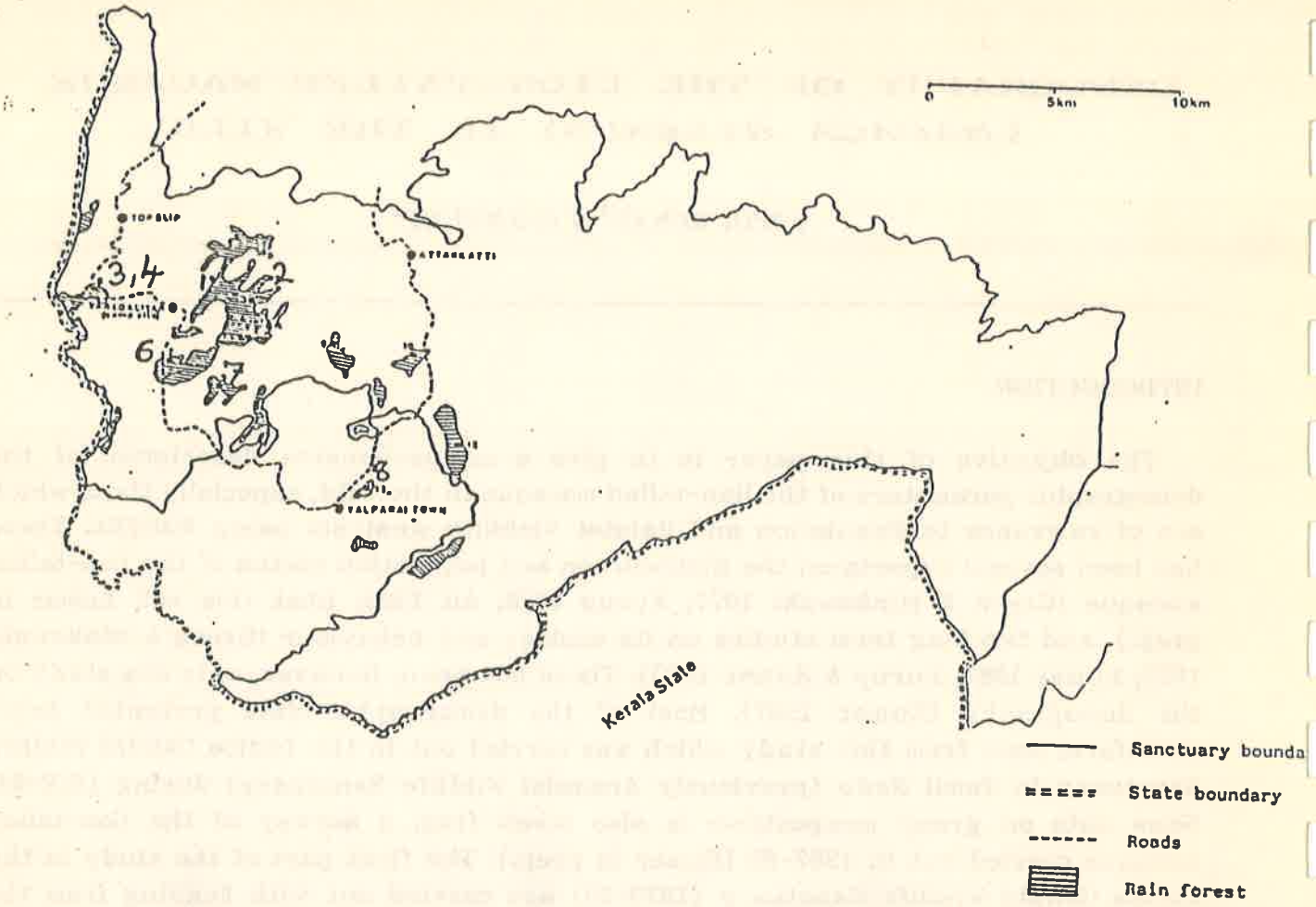

Figure 1: The Indira Gandhi Wildlife Sanctuary (previously Anamalai Wildlife Sanctuary) in Tamil Nadu State, showing the location of groups of lion-tailed macaques (numbered 1 to 8) from which demographic data were collected.

1. VSI: The main study group in Varagaliyar Shola
2. VSII: in Varagaliyar Shola
3. ASI: In Anakunthi Shola
4. ASII: In Anakunthi Shola
6. BS: in Banathiyar Shola
7. PH: in Power House Shola
8. EST: in Puthuthottam cardamom Estate

GROUP SIZE AND COMPOSITION

Data on group size and composition come from 20 groups. Of these, 10 groups were counted, six of them repeatedly at regular intervals, during the ecological study of the species in the Indira Gandhi Wildlife Sanctuary during 1978-84. Eight groups were counted during the survey of the lion-tailed macaque in 1987-89. For those groups which were censused repeatedly (at the Indira Gandhi WLS), the mean of group sizes on 1 April on each of the monitored year is taken as the typical group size. The frequency distribution of group size is shown in Figure 2 and group composition is shown in Table 1. The mean group size was 18.8 and the median 17.4, the smallest group size being 7 and the largest being 41. The number of adult males (more than about 8 years of age) varied from 1 to 3, with a mean of 1.5 and median of 1. Most of the groups had only one adult male. The number of males increased as the group size increased. The mean adult sex ratio was 4.9 females per male, and the median was 6.3. The percentages of adult and subadult males in the group appeared to vary more than that of the adult females, as indicated by the coefficient of variation. Even when the adult and subadult males were combined into one class, it showed considerable variation (43.5%). The percentage of immatures showed more variation than the females. Thus, the proportion of adult females is more or less constant in a group, when compared to that of adult and subadult males, and even immatures.

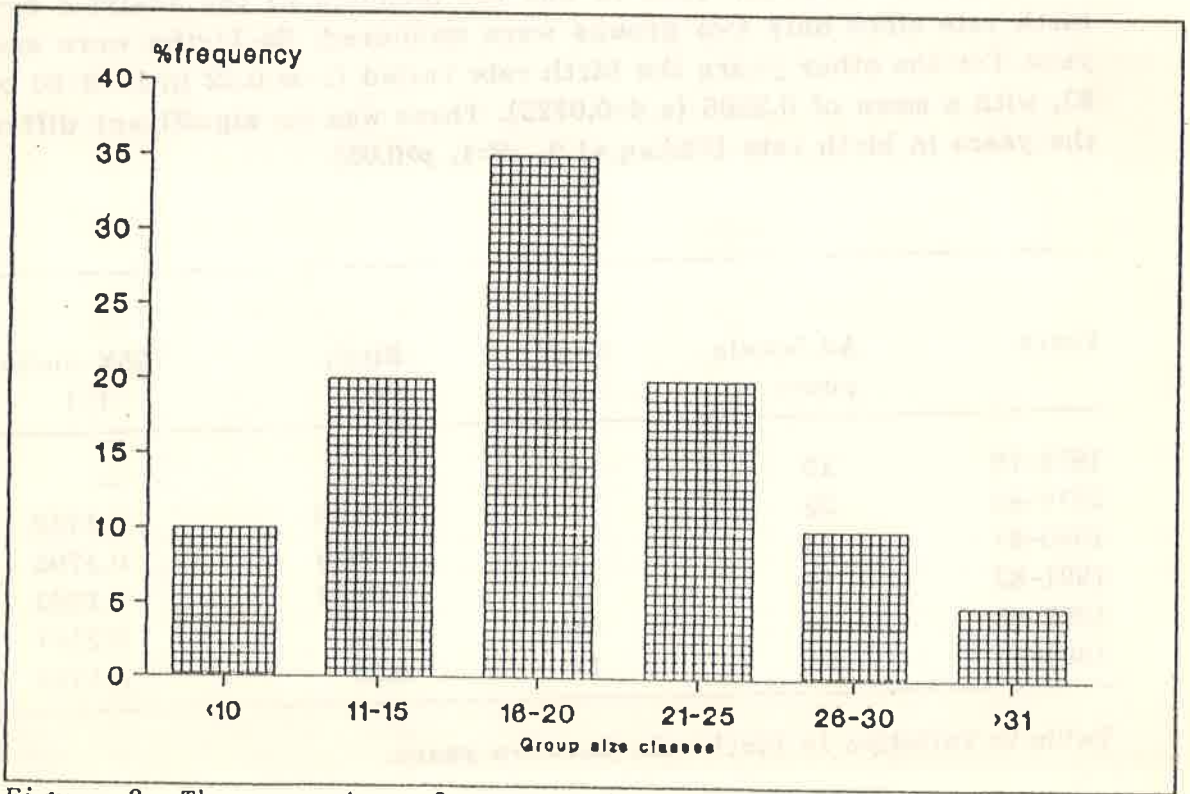

Figure 2: The percentage frequency distribution of group sizes in the lion-tailed macaque (n=20 groups).

	mean no.	s.e	median	range min-max	% comp	CV of % comp
Adult males	1.5	0.17	1.0	1-3	9.0	50.4
Subadult males	1.4	0.29	1.0	0-5	8.0	66.1
Adult females	7.3	0.74	6.3	3-14	41.5	16.5
Immatures	8.0	1.15	8.0	1-21	41.5	28.6
Total	18.8	1.93	17.4	7-41	---	

Table 1: The percentage age/sex composition of 20 groups of lion-tailed macaque.

BIRTH RATE

Birth rate is defined as the proportion of adult females which give birth in a year out of the total number of adult females under observation. Data for the estimation of birth rate come from 8 groups. Six of these were monitored regularly (at least once in a month) during 1978-80 and 1982-1984. Between 1980 and 1982 only one census of these groups was made. For two groups birth rate was estimated from the number of infants and adult females present in the group at the time of the census. A varying number of females were monitored each year from 1978 to 1984 (Table 2). The data for 1978-79 was not included in the analysis for estimation of birth rate since only two groups were monitored. No births were seen during this year. For the other years the birth rate varied from 0.22 in 1979-80 to 0.40 in 1982-83, with a mean of 0.2966 (s.d=0.0725). There was no significant difference between the years in birth rate (Chi.sq.=1.9, df=4, p>0.05).

Years	Ad.female years	No.of births	Birth rate	95% limits (±)
1978-79	10	0	--	--
1979-80	32	7	0.2188	0.1432
1980-81	23	6	0.2609	0.1795
1981-82	19	5	0.2632	0.1980
1982-83	20	8	0.40	0.2147
1983-84	50	17	0.34	0.1313

Table 2: Variation in birth rate between years.

Interbirth Interval

Direct observation of interbirth interval was made on 3 identifiable females in the main study group. These 3 females gave birth twice each during the six year study period, with interbirth intervals of 30, 28 and 31 months (mean=29.7 months or 2.47 years).

Age at First Birth

Data on this parameter is available from five females in the main study group. Their age could be reliably estimated since they were infants or juveniles at the start of the study in 1978. One gave birth at 6 years of age, three between 6 and 7 years, and one between 7 and 8 years of age. The mean age at first birth is thus estimated as 6.6 years (s.d=0.55), and the median as 6.5 years.

Age at Last Birth

The females in many primate species show a drastic drop in birth rate in late life, a stage called post-reproductive stage (Rowell 1969). This is probably due to increasing ovulatory irregularity (Graham et al 1979). Data on the duration of the post-reproductive stage is available from 2 females of the main study group. The oldest female of the group in 1978 gave birth in May 1977. Since then in spite of repeated sexual swelling and mating, it never gave birth. It disappeared from the group between March 1981 and April 1982, and was assumed to have died. Taking the midpoint between March 1981 and April 1982 as the time when it died, this female showed an infertile period of 51 months. The second female which gave birth in December 1979 did not give birth again until the end of the study period in 1984, but was still alive. It also did not show any swelling or mating. This also gave a post-reproductive period of at least 51 months.

MORTALITY RATE

Mortality rate, defined as the probability of an animal dying in a year, was estimated as the proportion of animals which disappeared out of the total number of animals which were monitored in a year. In total, 319.5 animals years (the sum of the number of animals which were monitored each year from each group, the fraction indicating infants which were born during the course of the year) were monitored from five groups in 1978-84 for the estimation of mortality (Table 3). Mortality had to be assumed in all cases upon the disappearance of an animal. In total 13 animals disappeared. The gross mortality rate for all the age/sex classes combined varied from 0.0904/year in 1981-82 to 0.0130 in 1979-80, with mean of 0.0450 (s.d=0.0342). The

data is not sufficient to examine variation between years for the various age/sex classes separately. The estimated mortality rate for each age/sex class is given in Table 4.

Year	Animal years	Deaths recorded	Mortality rate/year
1978-79	39.33	1	0.0254
1979-80	77.16	1	0.0130
1980-81	61.08	1	0.0164
1981-82	33.17	3	0.0904
1982-83	49.59	2	0.0403
1983-84	59.09	5	0.0846

Table 3: The mortality rate of all age/sex classes together in each year.

Male Mortality

Among the macaques emigration from groups is almost entirely confined to the subadult and adult males, and is indistinguishable in the field from mortality. Therefore, the mortality estimate for subadult and adult males represent emigration and mortality. Since the groups had only few males only 37 animal years of data could be collected on them. There were 3 cases of disappearance, giving a mortality/-emigration rate of 0.0810 (s.d=0.0448). This we consider as the minimum estimate since emigration or death of a male followed by immigration of another male into a group other than the main study group might not have been identified by us.

Adult Female Mortality

In total, 118 animals years of data were collected to estimate mortality rate of adult females. Only 4 females disappeared, giving a mortality rate of 0.0339/female/year (s.d=0.0167). The data is not sufficient to examine variation between years.

Immature Mortality

Immature or pre-reproductive mortality refers to mortality up to six years of age. As mentioned earlier the females give birth for the first time at about 6.6 years of

age, or conceive at 6 years of age. For males, however, there is a prolonged subadult hood from about 6 years of age to probably 8 years of age. During this stage mortality rates for the adult males, estimated above, is assumed to apply. For want of data, infants (0 to 1 year of age) are considered along with the juveniles, and no distinction is made between the sexes. In total, 164.4 animal years of data were collected to estimate mortality rate at the pre-reproductive stage. Eight animals disappeared, giving a mortality rate of 0.0487 (s.d=0.0168).

Age/sex class	Animals years	Deaths recorded	Mortality rate/year	s.d
adult male	37	3	0.0810	0.0448
adult female	118	4	0.0339	0.0167
immatures	164.4	8	0.0487	0.0168

Table 4: The mortality rate of the different age/sex classes

Male Immigration

During 17 group-years (the sum of the number of years for which each group was monitored), immigration into a group occurred at least 6 times. The immigration rate thus could be estimated as 0.35/group/year. All the six cases were by adult or subadult males.

DENSITY DEPENDENCE OF DEMOGRAPHIC PARAMETERS

Some form of density dependence of demographic parameters have been reported in many species. For example Tanner (1966) analyzed data from 71 species and found evidence of decreasing population growth rate with increasing population density in at least 47 species. Several factors could, however, confound this relationship and make it difficult to detect (Strong 1985). The density dependent variation in demographic parameters was examined in the lion-tailed macaque, taking group as the basic population unit for two reasons. Firstly, it was not possible to collect data on a single population, consisting of several groups, for sufficiently long periods of time. Secondly, and more importantly, as with most other macaques the lion-tailed macaque lives in distinct social groups. Demographic interactions between groups are infrequent and consist only of rare migration by males. Many of the ecological, behavioral and ultimately demographic responses to density might be expressed as

response to changes in group size. Many of the models on variation in demographic parameters in primates, therefore, have used group as the unit of population (Wittenberger 1980; Wrangham 1980; van Schaik 1983). For these reasons, in the analysis which follows, the variations in demographic parameters are correlated with changes in group size and the number of adult females in the group, the former always giving a better fit. Three demographic parameters are examined: the exponential rate of growth of group (r), to detect the existence of density dependent population growth rate: and birth rate and survival rate to examine the demographic mechanism by which the density dependence is brought about. The relationship is examined using linear and log-linear models, the latter giving better fits in all cases (Table 6).

Growth Rate

Data on growth rate of groups is available from 8 groups which were monitored from 1 to 6 years (Table 5). The mean annual growth rate of each group was estimated as the mean of the exponential growth rate of each year for which the group was monitored. The corresponding group size was estimated as the mean of the group size at the beginning of each observation year (1 April).

The exponential growth rate of group (r) in a year varied from -0.07 (negative growth) to 0.33. The mean growth rate of groups across the period for which they were monitored, however, were all positive and varied from 0.06 to 0.17 (Table 5). The mean r was negatively correlated to the mean group size (Pearson correlation R=-0.79, p<0.05, n=8, Figure 3), thus showing that the growth rate of groups is negatively density dependent. The regression equation of the relationship is given in Table 6.

Group	Years monitored	Group size start	end	mean	Mean females	Mean r	Birth rate
VSI	1979-84	12	24	16.40	6.8	0.12	0.3823
VSII	1979-84	13	17	15.20	6.6	0.06	0.2424
AS	1978-81	28	31	28.33	11.3	0.03	0.1470
ASI	1982-84	18	24	19.50	10.0	0.10	0.3000
ASII	1981-84	13	18	13.33	6.0	0.13	0.3333
BS	1979-80	21	23	22.00	9.0	0.09	0.2222
PH	1982-84	22	26	23.50	11.0	0.08	0.1818
EST	1982-84	12	17	12.00	5.0	0.17	0.5000

Table 5: The means of group size, number of adult females, growth rate and birth rate for 8 eight groups for the period for which they were monitored.

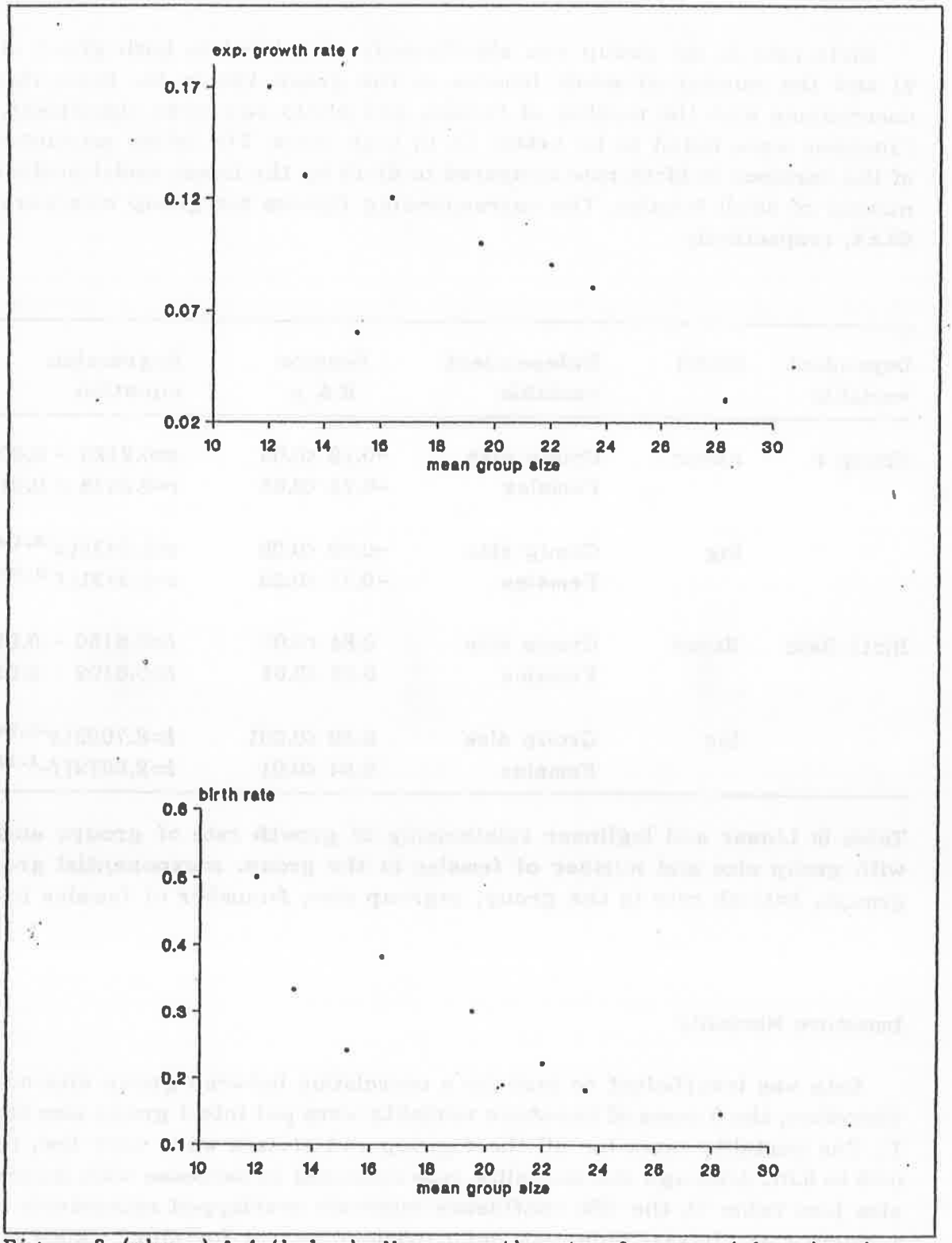

Figure 3 (above) & 4 (below): Mean growth rate of groups (above) and mean birth rate (below) plotted against mean group size for 8 groups during the period for which they were monitored.

Birth Rate

Birth rate in the group was significantly correlated to both group size (Figure 4) and the number of adult females in the group (Table 6). Even though linear correlations with the number of females and group size were significant, log-linear functions were found to be better fit in both cases. The latter accounted for 70.9% of the variance in birth rate compared to 67.1% by the linear model in the case of the number of adult females. The corresponding figures for group size were 78.6% and 69.8%, respectively.

Dependent variable	Model	Independent variable	Pearson R & p	Regression equation
Group r	linear	Group size	-0.79 <0.05	$r=0.2125 - 0.0061x$
		Females	-0.72 <0.05	$r=0.2048 - 0.0131f$
	log	Group size	-0.80 <0.05	$r=1.5432(x^{-0.1162})$
		Females	-0.75 <0.05	$r=1.3731(f^{-0.1063})$
Birth Rate	linear	Group size	0.84 <0.01	$b=0.6150 - 0.0174x$
		Females	0.82 <0.01	$b=0.6102 - 0.0392f$
	log	Group size	0.89 <0.001	$b=8.7093(x^{-1.2013})$
		Females	0.84 <0.01	$b=2.6679(f^{-1.1103})$

Table 6: Linear and loglinear relationship of growth rate of groups and birth rate with group size and number of females in the group. r=exponential growth rate of groups, b=birth rate in the group, x=group size, f=number of females in the group.

Immature Mortality

Data was insufficient to test for a correlation between group size and mortality. Therefore, the 8 cases of immature mortality were put into 4 group size classes (Table 7). The mortality rates for all the 4 group size classes were very low, ranging from 0.03 to 0.07. Although the mortality rate appeared to decrease with increasing group size (see Table 7), the 95% confidence intervals overlapped extensively between the 4 group size classes. Data was not sufficient to test for significance of difference between the group size classes.

Group size	Sample	Deaths	Mortality	95% limit (±)
11.6-16.5	57.41	4	0.0697	0.0336
16.6-21.5	41.51	2	0.0482	0.0332
21.6-26.5	37.25	1	0.0268	0.0265
26.6-31.5	28.25	1	0.0354	0.0348

Table 7: The immature or pre-reproductive mortality rates in four group size classes.

DISCUSSION

The group size of the lion-tailed macaque is similar to that reported for the other macaques, in the range of 20-30 animals. However, the adult sex ratio in a group is more biased towards the females, with about 5-6 females per male compared to 1-3 females per male reported for most other macaques. A similar sex ratio also occurs in the pig-tailed macaque, *M.nemestrina* (Caldecott 1986). There is no indication of any significant geographical variation in group size and composition. The populations south and north of the Palghat Gap have similar group sizes (18.4 and 19.1, respectively). Their age/sex compositions are also not significantly different.

The demographically important life history parameters of the lion-tailed macaque seem to be exceptional compared to the other macaques. The high age at first birth, low birth rate, and low mortality rates at all stages of life are noteworthy features. These indicate that the life history parameters of the lion-tailed macaque are highly adapted to its relative stable habitat or are K-selected. The inevitable consequence of the above suit of parameters is that the lion-tailed macaque would have very low capability to track rapid changes in resources and to recover from population perturbations caused for example by diseases or hunting.

There is strong evidence that growth rate of groups is strongly and negatively related to group size and the number of adult females in the group. This density dependent growth is brought about by a reduction in birth rate as the group size increases. A similar decrease in birth rate with increasing group size and number of adult females in the group has been reported from a number of primates (Van Schaik 1983). Increasing competition between females for food is probably the major factor which cause such a decrease in birth rate (Wrangham 1980; van Schaik 1983). This is also the most likely explanation for the reduction in birth rate in the lion-tailed macaque. An increase in group size in the lion-tailed macaque causes (a) A reduction in time spent feeding on foods of animal origin which form the primary source of protein; (b) Selection of less preferred plant food items; (c) A reduction in the time spent resting; (d) An increase in the time spent ranging and foraging; (e) An

increase in the distance travelled per day; and (f) An increase in the time spent on agonistic interactions (Kumar 1987). Some of these effects of increase in group size have also been reported for the long-tailed macaque, *M.fascicularis* (van Schaik 1983, 1986; van Schaik *et al* 1983).

Immature mortality has been hypothesised to decrease initially with increasing group size due to increased protection from predators, and then increase as intra-group competition for food adversely affect immature survival. Evidence for this has been reported for some species (van Schaik 1983). The present data on the lion-tailed macaque is not sufficient to test this hypothesis. The density dependent growth rate in the lion-tailed macaque, therefore, is largely driven by the increasing competition among females for resources which cause a reduction in birth rate.

REFERENCES

Ali, R. (1985). An overview of the status and distribution of the lion-tailed macaque. In, *The Lion-tailed Macaque: Status and Distribution* (Ed, by P.G.Heltne), pp. 13-26. Alan R.Liss, New York.

Caldecott, J.O. (1986). *An Ecological Study of the Pig-tailed Macaque.* Contributions to Primatology, Vol.21, 1-259. Karger, Basel.

Graham, C.E., Kling, O.R. & Steiner. (1979). Reproductive senescence in female non-human primates. In, *Ageing in Non-human Primates* (Ed. by D.Bowden), pp.183-202. van Nostrand Reinhold, New York.

Green, S.M. & Minkowski, K. (1977). The lion-tailed macaque and its South Indian rain forest habitat. In, *Primate Conservation* (Ed. by H.S.H.Rainier, III & G.H.Bourne), pp. 290-338. Academic Press, New York.

Karanth, U. (1985). Ecological status of the lion-tailed macaque and its rain forest habitats in Karnataka, India. *Primate Conservation*, 6: 73-84.

Kumar, A. (1987). *The Ecology and Population Dynamics of the Lion-tailed Macaque (Macaca silenus) in South India.* Ph.D. Dissertation, Cambridge University, U.K.

Kurup, G.U. (1978). Distribution, habitat and status of the lion-tailed macaque, *Macaca silenus. Journal of the Bombay Natural History Society*, 75: 321-340.

Kurup, G.U. & Kumar, A. (1993). Time budget and activity patterns of the lion-tailed macaque (*Macaca silenus*). *International Journal of Primatology, 14:27-39.*

Rowell, T.E. (1969). Long-term changes in a population of Ugandan baboons. *Folia Primatologica,* 11:241-254.

van Schaik, C.P. (1983). Why are diurnal primates living in groups? *Behaviour*, 87: 120-144.

----- (1986). Exploitation and interference in feeding competition among female long-tailed macaques (*Macaca fascicularis*). Paper presented at the XI Congress of the International Primatological Society, Gottingen, Germany.

van Schaik, C.P., Noordwijk, M.A., van De Boer, R.J. & Den Tonkelaar, I. (1983). The effect of group size on time budget and social behaviour in wild long-tailed macaques. *Behavioral Ecology and Sociobiology,* 13: 173-181.

Strong, D.R. (1986). Density-vague population change. *TREE*, 1:39-45.

Tanner, J.T. (1966). Effects of population density on growth rates of animal populations. *Ecology*, 47:733-745.

Wittenberger, J.F. (1980). Group size and polygyny in social mammals. *American Naturalist*, 115: 197-221.

Wrangham, R.W. (1980). An ecological model of female-bonded primate groups. *Behaviour*, 75:262-300.

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DEMOGRAPHY GLOSSARY

Age Age class in years.

Px Age-specific survival.

Probability that an animal of age x will survive to next age class.

Lx Age-specific survivorship.

Probability of a newborn surviving to a age class x.

Mx Age-specific fertility.

Average number of offspring (of the same sex as the parent) produced by an animal in age class x. Can also be interpreted as average percentage of animals that will reproduce.

r Instantaneous rate of change.

If $r < 0$ ..... Population is declining

If $r = 0$ ..... Population is stationary (no change in number)

If $r > 0$ ..... Population is increasing

lambda Percent of population change per year.

If lambda < 1 ..... Population is declining

If lambda = 1 ..... Population is stationary

If lambda > 1 ..... Population is increasing

R_o Net reproductive rate. The rate of change per generation.

If $R_o < 1$ ..... Population is declining

If $R_o = 1$ ..... Population is stationary

If $R_o > 1$ ..... Population is increasing

G Generation Time.

Average length of time between the birth of a parent and the birth of its offspring. Equivalently, the average age at which an animal produces its offspring)

What is Population (and Habitat) Viability Analysis?

by Robert C. Lacy

Some history of concepts, terms, and approaches

The papers presented in this symposium refer both to "Population Viability Analysis (PVA)" and to "Population and Habitat Viability Analysis (PHVA)." In the conservation literature, one can find reference also to "Population Vulnerability Analysis", "Population Viability (or Vulnerability) Assessment", and other variants on the name. This diversity of terminology has caused some confusion among practitioners of the PVA (or PHVA) approach, and probably even more confusion among conservationists and wildlife managers who have tried to understand what analysis was being described, and whether it could be a useful tool in their efforts to conserve biodiversity. The diversity of perceptions about the PVA approach is not limited to its name. Different people mean different things by PVA, and the definitions and practice of PVA are constantly evolving. It is not the case, as has sometimes been suggested, that some people are doing PVA correctly, and others incorrectly, but rather that people are using different (if related) kinds of analyses and labelling them with the same (or similar) terms. What analysis is correct depends on the need and the application. In this paper, I will attempt to clarify what PVA is, by suggesting a more consistent terminology and by describing the features that characterize the application of the PVA approach to conservation. The perspective I offer is necessarily biased by my experiences in conservation. I will not attempt an exhaustive historical account of this field.

Population viability analysis originally described methods of quantitative analysis to determine the probability of extinction of a population. Shaffer (1981) identified multiple factors that can threaten viability or persistence of a population. He usefully categorized these factors into demographic stochasticity, environmental variability, catastrophic variation, and genetic drift and inbreeding. Any or all of these factors could threaten the persistence of a population that falls below a "minimum viable population size", or MVP. Each of these factors entails a stochastic, or random probabilistic, process which causes small populations to undergo fluctuations and possibly further decline. The smaller and more erratic is the size of a population, the greater the probability that the number will, by chance, hit zero -- the extirpation of the local population, or ultimate extinction if the local population was all that remained of the taxon. Gilpin and Soulé (1986) stressed that the destabilizing stochastic processes are synergistic: various of demographic, environmental, and genetic processes can exacerbate the instability caused by other factors, causing what has been depressingly referred to as "extinction vortices". Shaffer (1981) first defined an MVP as the size at which a population has a 99% probability of persistence for 1000 years, but it might be more meaningful biologically to consider it to be the size below which a population's fate becomes determined largely by the stochastic factors that characterize extinction vortices.

One concept of population viability analysis is any methodology used to determine an MVP. Shaffer had suggested several ways to determine an MVP. Perhaps the most rigorous method, and the one that would produce the most defensible estimates, would be an empirical observation of the stabilities and long term fates of a number of populations of various sizes. Berger (1990) presented a good example of this approach, in which he observed that populations of bighorn sheep in the mountains of the western USA persisted only when the populations consisted of more than 100 animals. More empirical studies are needed, but the time and numbers of populations required for such studies are precluded in the cases of most species threatened with extinction -- exactly those for which estimates of MVP are most urgently needed. A more elegant and general approach to PVA is to develop analytical models of the extinction process that will allow calculation of the probability of extinction from a small number of measurable parameters. Goodman's (1987) model of demographic fluctuations, and applications to conservation of the classic population genetic models of loss of genetic diversity by genetic drift (Franklin, 1980; Soulé *et al.*, 1986; Lande and Barrowclough, 1987) are valuable efforts in this direction. Kinnaird and O'Brien (this issue) describe the use of some of these models in a PVA of the Tana River crested mangabey (*Cercocebus galeritus galeritus*).

Unfortunately, our understanding of population biology is not yet sufficient to provide fully adequate analytical models of the extinction process. For example, none of the existing analytical models incorporate all three of demographic, environmental, and genetic fluctuations, and thus they do not begin to model the array of extinction vortices described by Gilpin and Soulé (1986). Moreover, the analytical models make extremely simplifying assumptions about a number of the intricacies of population structure. For example, social groupings or preferences are often assumed to be invariant or lacking, resulting in random mating; and dispersal is usually assumed to be

random between all sites (the "island model") or only to occur between adjacent sites (the "stepping stone model"). Much more work is needed either to develop more complex and flexible models or to demonstrate that the simple models are sufficient to provide guidance for conservation.

A third method of estimating an MVP described and demonstrated by Shaffer (1981) is the use of computer simulation modeling to project the probability distribution of possible fates of a population. Simulation models can incorporate a very large number of threatening processes and their interactions, if the processes can be described in terms of quantitative algorithms and parameterized. The flexibility of such models is both an strength and a weakness of the approach (Lacy, 1993a; Lindenmayer *et al.*, 1993a). The extinction process is exceedingly complex (Clark *et al.*, 1990), and it can be difficult to focus on the key threatening processes when using a model that allows for testing of almost everything (Lacy and Clark, 1990; Maguire *et al.*, 1990; Lindenmayer *et al.*, 1991, 1993a, 1993b).

Shaffer's (1981) original term "minimum viable population" (MVP) has fallen into disfavor (Soulé, 1987), even as the PVA approach has risen in popularity. Shaffer stressed that an MVP was an estimate of the population size below which the *probability* of extinction was unacceptably high, that different populations would have different MVPs, and that the MVP determined for a population would depend on the threatening factors that were considered. However, the term implied to some people that there was a well-defined number below which extinction was certain and above which persistence was assured. Re-emphasizing the probabilistic nature of the extinction process, a number of conservation biologists have focused on methods for estimating the probability of extinction over defined time periods for a designated population exposed to a specific scenario of environmental conditions, threats to persistence, and future management actions and other foreseeable events (Brussard, 1985; Starfield and Bleloch, 1986; Soulé, 1987; Simberloff, 1988; Gilpin, 1989; Shaffer, 1990; Boyce, 1992; Burgmann *et al.*, 1993; Lacy, in press). Thus, "Population Viability Analysis" (or the synonymous "Population Viability Assessment" and "Population Vulnerability Analysis") came to describe any of the array of methods for quantifying the probability of extinction of a population. Although PVA has been extended by some to encompass a broader approach to conservation (see below), the term "Population Viability Analysis", or PVA, should perhaps be reserved for its original, yet still rather broad, meaning.

Beginning in about 1989 (Lacy *et al.*, 1989; Seal and Lacy, 1989; Seal *et al.*, 1990), it became increasingly recognized that PVA can often be most usefully incorporated into a strategy for the conservation of a taxon if it is part of, and often central to, a conservation workshop that mobilizes collaboration among the array of people with strong interest in or responsibility for a conservation effort (e.g., governmental wildlife agencies, conservation NGOs, and the local people who interact with the species or its habitat) or with particular expert knowledge about the species, its habitats, or the threats it faces (e.g., academic biologists, conservation professionals, other wildlife biologists, experts on human demographics and resource use). Conservation problems are almost always multi-faceted, involving not only complex dynamics of biological populations, but also interactions with human populations, the past, present, and future impacts of humans on habitats, and human political, social, and economic systems (Alvarez, 1993; Bormann and Kellert, 1991; Clark, 1989, 1993). Many people need to contribute knowledge, expertise, and ideas in order to achieve the recovery of threatened species. Population viability analyses can provide a framework for incorporating the many needed kinds of knowledge into species conservation efforts, because PVAs do allow the assessment of many kinds of factors that threaten the persistence of populations (Lacy, 1993a; Lindenmayer *et al.*, 1993a).

The Conservation Breeding Specialist Group (CBSG, formerly called the Captive Breeding Specialist Group) of the IUCN Species Survival Commission especially has advocated and used workshops centered on PVAs to provide guidance to conservation assessment and planning (e.g., Seal *et al.*, 1990; Ellis *et al.*, 1992a; Foose *et al.*, 1993). Over the past few years, the PVA workshop as an approach to species conservation has expanded considerably beyond the quantitative analysis of extinction probabilities as advanced by Shaffer (1981, 1990), Soulé (1987), Gilpin (1989), Clark *et al.* (1991), Boyce (1992), and others. PVA workshops have incorporated consideration of resource use and needs by local human populations (Seal *et al.*, 1991), education programs for the local human populations (Odum *et al.*, 1993), trade issues (Foose *et al.*, 1993), and trends in human demographics and land use patterns (Walker and Molur, 1994). Recognizing that the conservation assessment workshops increasingly incorporated more than just the population biology modeling (which still formed a core organizing and analysis framework for the workshop), the CBSG has more recently termed their workshops Population *and Habitat* Viability Analyses (PHVA) (e.g., Ellis *et al.*, 1992a; Odum *et al.*, 1993). I would recommend that the term Population and Habitat Viability Analysis (PHVA) be used to describe the collaborative workshop approach to species conservation that centers on, but encompasses more than, a Population Viability Analysis (in the narrow sense). The concept of a PHVA continues to expand and evolve, as it should considering the need for more holistic and flexible approaches to conservation

(e.g., Ruggiero *et al.*, 1994; and see the debates in almost any issue of Conservation Biology). Thus, in the usage I recommend, PVA is a quantitative analysis of the probability of population persistence under defined sets of assumptions and circumstances. PHVA is a workshop process that brings to bear the knowledge of many people on species conservation, eliciting and assessing multiple options for conservation action, principally by using the tool of PVA as a way evaluate present threats to population persistence and likely fates under various possible scenarios. In the IPS symposium on the application of P(H)VAs to primate conservation, there were presented discussions and examples of PVAs, in the narrow sense (e.g., Kinnaird and O'Brien, this volume), and PHVAs, in the broadest sense (e.g., Rylands, this volume; Brockelman, this volume).

Population Viability Analysis (PVA)

Two defining characteristics of a PVA are an explicit model of the extinction process and the quantification of threats to extinction. These features set PVA apart from many other analyses of the threats facing species, including, for example, the IUCN Red Books of Threatened Species published in the past. As a methodology to estimate the probability of extinction of a taxon, PVA necessarily must start with an understanding, or model, of the extinction process (Clark *et al.*, 1990).

Generally, the model of extinction underlying a PVA considers two categories of factors: deterministic and stochastic. Deterministic factors, those that can shift species from long-term average population growth to population decline include the well-known threats of over-harvest, habitat destruction, pollution or other degradation of environmental quality, and the introduction of exotic predators, competitors, and diseases. Singly or combined, these forces have driven many wildlife populations to low numbers and, for some, to extinction. Once a population becomes small, and isolated from conspecific populations that might serve as sources for immigrants that could stabilize demographics and genetics, its dynamics and fate can become dominated by a number of random or stochastic processes (as outlined above and by Shaffer, 1981). Thus, even if the original deterministic causes of decline are stopped or reversed, the instability caused by the action of stochastic processes acting on small populations can cause the extinction of a population.

In nature, most threatening processes have both deterministic and stochastic features. For example, a high level of poaching might be seen as a deterministic factor driving a wildlife population toward extinction, but whether an individual animal is killed might be largely a matter of chance. In a PVA, poaching might be modelled as a deterministic process by killing a determined proportion of the animals, or it might be modelled as a stochastic process by giving each animal that probability of being killed but allowing the exact numbers killed to vary over time. If the population is large and the percent of animals killed is high, then these two ways of modelling the effects of poaching will yield the same results: the deterministic component of poaching dominates the population dynamics. If the population is small or the percent of animals killed is very low, then the numbers killed in a stochastic model (and in nature) might vary substantially from year to year: the stochastic nature of poaching further destabilizes the population.

Which of the various deterministic and stochastic factors are important to consider in a PVA will depend on the species biology, the present population size and distribution, and the threats it faces. For example, orang utans may be threatened by forest destruction and other largely deterministic processes, but inbreeding and randomly skewed sex ratios resulting from highly stochastic processes are unlikely to be problems, at least not on a species-wide basis. On the other hand, even if the remnant Atlantic coastal rainforest of Brazil is secured for the future, the populations of golden lion tamarins (*Leontopithecus rosalia*) which can persist in that remnant forest are not sufficiently large to be stable in the face of stochastic threats (Seal *et al.*, 1990; Rylands, this volume). The identification of the primary threats facing a taxon via a comprehensive PVA is important for conservation planning. For example, tamarin populations might be stabilized by the translocations and reintroductions that are underway and planned, but the orang utan PHVA recognized that releases of confiscated "pet" orang utans are unlikely to have a conservation benefit for those populations which are facing habitat destruction, not stochastic fluctuations and inbreeding. For many species, such as the whooping crane (*Grus americana*), the temporarily extinct-in-the-wild black-footed ferret (*Mustela nigripes*), and the Puerto Rican parrot (*Amazona vitatta*), only a single population persisted in the wild. Although those populations may have been maintained or even increased for a number of years, the principal threat was that a local catastrophe (e.g., disease epidemic, severe storm) could decimate the population (Clark, 1989; Lacy *et al.*, 1989; Mirande *et al.*, 1991). The primary recovery actions therefore needed to include the establishment of additional populations. Tragically, some taxa, such as the Florida panther (*Felis concolor coryi*) and

the eastern barred bandicoot (*Perameles gunnii*) in Victoria, Australia, are critically threatened simultaneously by deterministic factors, stochastic processes, and the possibility of catastrophic loss (Seal and Lacy, 1989; Lacy and Clark, 1990).

PVA is formally an assessment of the probability of extinction, but PVA methods often focus on other indicators of population health. Mean and variance in population growth (Lindenmayer and Lacy, 1995a, 1995b, 1995c), changes in range, distribution, and habitat occupancy (Hanski and Gilpin, 1991), and losses of genetic variability (Soulé *et al.*, 1986; Lande and Barrowclough, 1987; Seal, 1992; Lacy and Lindenmayer, 1995) can be analyzed and monitored. Although not yet common, monitoring of population health could also utilize measures of developmental stability (Clarke, 1995), physiological parameters such as body condition (Altmann *et al.*, 1993) or levels of the hormones related to stress and reproduction (Sapolsky, 1982, 1986), or the stability of behavior and the social structure of the population (Samuels and Altmann, 1991).

The interactions and synergism among threatening processes will often cause numerical, distributional, physiologic, behavioral, and genetic responses to concordantly reflect species decline and vulnerability. It remains important, however, to understand and target the primary causal factors in species vulnerability. The recent proposal to base IUCN categories of threat on quantified criteria of probability of extinction, or changes in such indicators as species range, numbers, and trends (Mace and Lande, 1991; Mace *et al.*, 1992; Mace and Stuart, 1994; IUCN Species Survival Commission, 1994) reflects the increased understanding of the extinction process that has accompanied the development of PVA, and simultaneously demands that much more progress be made in developing predictive models, gathering relevant data on status and threats, and applying the PVA techniques.

Population and Habitat Viability Analysis (PHVA)

Population and Habitat Viability Analysis is a multi-faceted process or framework for assisting conservation planning, rather than a singular technique or tool. It is often interwoven with other techniques for managing complex systems, such as decision analysis (Maguire, 1986; Maguire *et al.*, 1990). Even when viewed as "the" PHVA workshop, all such conservation workshops involved and required substantial pre-workshop and post-workshop activities. Some PHVA workshops have been extended into multiple workshops and less formal, smaller collaborative meetings, often focused on subsets of the larger problems of species conservation.

Although PHVAs are diverse and not well defined, the PHVA process contains a number of critical components (Lacy, in press), many of which were illustrated well in the PHVA workshops described in this symposium. First, it is essential to gather an array of experts who have knowledge of the species or problem. A PHVA is not required to bring together experts, but it often facilitates such sharing of expertise because the collective knowledge of many is essential for a useful PVA (in the narrow sense) to be completed. In addition to a diversity of people, a PHVA workshop also requires and therefore facilitates the involvement of a number of agencies and other concerned organizations. For example, the PVA on the two endemic primates of the Tana River Primate Reserve in Kenya (Seal *et al.* 1991) was convened by the Kenya Wildlife Service, facilitated by the IUCN SSC Captive Breeding Specialist Group, benefitted from the expertise contributed by members of the IUCN SSC Primate Specialist Group, and was sponsored by the World Bank. The involvement of many agencies and interested parties is critical to endangered species recovery.

An early requirement, or prerequisite, of a PHVA workshop is to determine the conservation problem to be addressed, and to state the goals of the management plan. Many endangered species programs have not clearly identified their goals. For example, at a PHVA and Conservation Assessment and Management Plan workshop on the forest birds of the Hawaiian islands (Ellis *et al.*, 1992a, 1992b), it became apparent that the agencies responsible for the conservation of Hawaii's bird fauna had not determined whether their goal was to prevent species extinctions, prevent taxa (species or subspecies) from becoming extirpated on any of the islands they presently inhabit, preserve species in sufficient numbers and distribution to allow them to continue to fill ecological roles in the biological communities, or the restoration of taxa to most or all parts of the original ranges. The management actions required to achieve these various levels of conservation are quite different.

PHVA workshops facilitate the assembly of all available data. Often, important information is found in the field notes of researchers or managers, in the heads of those who have worked with and thought about the problems of the species, and in unpublished agency reports, as well as in the published scientific literature. A pending PHVA can be the impetus that encourages the collection of data in anticipation of presentation, review, and analysis at the workshop. For example, a Sumatran Tiger PHVA helped stimulate the systematic collection of data on sightings and

signs of tigers in protected areas throughout the island of Sumatra, and collation and integration with a Geographic Information System (GIS) map of habitats and human pressures on those habitats.

It is important to specify the assumptions that underlay a PHVA, and any consequent management recommendation. For example, the Hawaiian bird conservation efforts are constrained by a belief that no birds bred outside of the islands should ever be brought back to the islands for release. While this position derives from a reasonable concern for disease transmission (much of the decline of Hawaii's native birds is thought to be due to introduced avian diseases) as much as from any political or philosophical stand, any justification for the restriction must be questioned in light of the fact that wildlife agencies import and release, without quarantine, 1000s of exotic gamebirds onto the islands annually.

Once experts are assembled, problems stated and goals set, data gathered, and assumptions specified, then the PHVA process can proceed with what I describe as PVA in the narrow sense: estimation of the probability of population persistence. The available data are used to estimate the parameters that are needed for the model of population dynamics to be applied. Often, data are not available from which to estimate certain key parameters. In those cases, subjective and objective, but non-quantified, information might be solicited from the assembled experts, values might be obtained from data on related species, or a factor might simply be omitted from the model. While such a non-precise process might consist simply of intuitive judgements made by experts, it is important to specify how values for the parameters in the model were obtained. The resulting limitations of the analyses should be acknowledged, and a decision made if, how, by whom, and when the missing data would be collected so that more refined analyses could be conducted. With the PVA model, projections of the most likely fate, and distribution of possible fates, of the population under the specified assumptions are made.

Because so much of a PVA -- the data, the model, and even the interpretation of output -- is uncertain, a PVA that provides an estimate of the probability of extinction under a single scenario is of very limited usefulness. An essential component of the PHVA process, therefore, is sensitivity testing. Ranges of plausible values for uncertain parameters should be tested, to determine what effects those uncertainties might have on the results. In addition, several different PVA models might be examined at a PHVA workshop, or the same general model tested under different structural assumptions. Different participants in the process should assess and interpret the results. Such sensitivity testing reveals which components of the data, model, and interpretation have the largest impact on the population projections. This will indicate which aspects of the biology of the population and its situation contribute most to its vulnerability and, therefore, which aspects might be most effectively targeted for management. In addition, uncertain parameters that have a strong impact on results are those which might be the focus of future research efforts, to better specify the dynamics of the population. Close monitoring of such parameters might also be important for testing the assumptions behind the selected management options and for assessing the success of conservation efforts.

Closely parallel to the testing of uncertainties in the present situation is the testing of options for management. PVA modeling allows one to test the expected results of any given management action, under the assumptions of the model and within the limitations of present knowledge, on the computer before implementation in the field. This process can guide selection of the management options most likely, given current knowledge, to be effective, and will define target recovery goals that should be obtained if our knowledge is adequate and the recommended actions are followed. A PHVA workshop on the Black Rhinoceros in Kenya's 11 rhino sanctuaries (Foose *et al.*, 1993) suggested that periodic movement of rhinos between fenced sanctuaries to reduce inbreeding and demographic fluctuations would be necessary to stabilize the populations in the smaller parks. Moreover, the modeling provided estimates of the rate at which the larger populations would be able to provide surplus animals for translocation.

It would be an error to assume that any PVA model incorporates everything of interest (Lacy, in press). A PVA simulation program can only include those processes that are known to the programmer. This will likely be a subset of what might be known to the field biologists, which in turn will definitely be a subset of those processes that impact natural populations. A number of variables affecting population dynamics and viability are not yet commonly examined in PVA models. These include: social and ecological determinants of dispersal; complex social processes, such as the role of non-breeders in group stability and the impacts of other aspects of the social environment on reproductive success and survival; competitive, exploitative, or mutualistic interactions with other species experiencing their own population dynamics; and the effects of changes in the global environment. To date, most PVA models treat organisms as independent actors in spatially homogeneous physical, biotic, and social

environments. There is tremendous opportunity and need for elaboration of PVA models, and it is likely that increasingly sophisticated models will also become more specific to the individual taxa and environments under study.

PHVA workshops must incorporate consideration of the assumptions of the PVA model used and the biases or limitations in interpretation that could result. PHVAs consider only those threatening processes of which we have knowledge, for which we can develop algorithms for modeling or other methods for analysis, and for which we have some data. As a result, it is likely that PVAs will underestimate the vulnerability of most populations to extinction, and that PHVA workshops will be less comprehensive than is desirable. We need always to be cognizant of the limits of our understanding of wildlife populations, and to include appropriate margins for error in our conservation strategies.

PVA is, by definition, an assessment of the probability of persistence of a population over a defined time frame. Yet, persistence of a population, while a necessary condition for effective conservation of natural systems, is often not sufficient. Prevention of extinction is the last stand of conservationists, but the goals should be higher: conservation of functional biological communities and ecosystems. PVA usually ignores the functional role of a species in a community, but a PHVA workshop should consider much more than the prevention of the final biological extinction of the taxon. A species, such as the American Bison (*Bison bison*), can be functionally extinct in terms of no longer filling its original role in nature, even as it is praised as a conservation success story and would, by PVA, be considered safe from extinction and viable.

The use of the PHVA process to help guide conservation decisions is not a singular event, in which an analysis can be completed, management actions recommended and implemented, and conservation thereby assured. The many uncertainties in the process mandate that PVA be used as a tool in an adaptive management framework, and a PHVA workshop is just one stage of an effective conservation strategy. In adaptive management, the lack of knowledge adequate to predict with certainty the best course of action is recognized, management actions are designed in such a way that monitoring will allow testing of the adequacy of our model and understanding, and corrective adjustments to management plans are made whenever the accumulating data suggest that the present course is inadequate to achieve the goals and that a better strategy exists (Holling, 1978). The urgency of the biodiversity crisis will not permit us ethically to refrain from aggressive conservation action until we have scientifically sound understanding of all the factors that drive population, community, and ecosystem dynamics. PHVA provides a forum for making use of the information we do have, in a well-documented process that is open to challenge and improvement. PHVA workshops can, therefore, assist wildlife managers in the very difficult and important job of using science to safeguard the future of wildlife populations.

Summary

Population Viability Analysis (PVA) and Population and Habitat Viability Analysis (PHVA) refer to an array of interrelated and evolving techniques for assessing the survival probability of a population and possible conservation actions. I suggest that it might be useful to restrict the term PVA to its original meaning -- the use of quantitative techniques to estimate the probability of population persistence under a chosen model of population dynamics, a specified set of biological and environmental parameters, and enumerated assumptions about human activities and impacts on the system. PHVA refers to a workshop approach to conservation planning, which elicits and encourages contributions from an array of experts and stakeholders, uses PVA and other quantitative and non-quantitative techniques to assess possible conservation actions, and strives to achieve consensus on the best course of action from competing interests and perspectives, incomplete knowledge, and an uncertain future.

Many of the components of PVAs and PHVAs, even when used in isolation, can be effective educational and research tools. To be a useful framework for advancing the conservation of biodiversity, however, PHVA must incorporate all of: (1) collection of data on the biology of the taxon, status of its habitat, and threats to its persistence, (2) quantitative analysis of available data, (3) input of population status and identifiable threats to persistence into analytical or simulation models of the extinction process, (4) assessment of the probability of survival over specified periods of time, given the assumptions and limitations of the data and model used, (5) sensitivity testing of estimates of extinction probability across the range of plausible values of uncertain parameters, (6) specification of conservation goals for the population, (7) identification of options for management, (8) projection of the probability of population survival under alternative scenarios for future conservation action, (9) implementation of optimal actions for assuring accomplishment of conservation goals, (10) continued monitoring of the population, (11) reassessment of assumptions, data, models, and options, and (12) adjustment of conservation strategies to respond to the best information available

at all times. There are many uncertain aspects of population dynamics, especially of endangered taxa, including few data on species biology and habitats, uncertain political and social climate for implementing conservation actions, and the unpredictability inherent in small populations due to the many stochastic forces that drive population dynamics.

The rapid development of PVA as a research and management tool, and the concurrent but not always parallel expansion of the scope of what conservation threats, options, and actions are considered in PHVA workshops, has led to confusion. Different people can describe rather distinct kinds of analyses with the same terminology, while others use different terms to describe nearly identical approaches. The ever-changing concepts of PVA and PHVA are confusing, but the flexibility of the processes is also their strength. Current tools are inadequate to address fully the challenges of stemming the losses of biodiversity. The PVA/PHVA framework allows and encourages rapid application of new tools, data, and interpretations into increasingly effective conservation programs.

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Literature Cited

Altmann, J., D. Schoeller, S.A. Altmann, P. Muruthi and R.M. Sapolsky. 1993. Body size and fatness of free-living baboons reflect food availability and activity levels. *Am. J. Primatol.* 30:149-161.

Alvarez, K. 1993. *Twilight of the panther. Biology, bureaucracy, and failure in an endangered species program.* Myakka River Publ., Sarasota, Florida.

Berger, J. 1990. Persistence of different-sized populations: an empirical assessment of rapid extinctions in bighorn sheep. *Conserv. Biol.* 4:91-98.

Bormann, F.H. and S.R. Kellert. 1991. *Ecology, Economics, and Ethics. The Broken Circle.* Yale University, New Haven.

Boyce, M.S. 1992. Population viability analysis. *Annu. Rev. Ecol. Syst.* 23:481-506.

Brussard, P. 1985. Minimum viable populations: how many are too few? *Restoration and Management Notes* 3:21-25.

Burgman, M., S. Ferson and H.R. Akçakaya. 1993. *Risk assessment in conservation biology.* Chapman and Hall, New York.

Clark, T.W. 1989. *Conservation biology of the black-footed ferret.* Wildlife Preservation Trust International, Philadelphia.

Clark, T.W. 1993. Creating and using knowledge for species and ecosystem conservation: Science, organizations, and policy. *Perspect. Biol. Med.* 36:497-525.

Clark, T.W., R.M. Warneke and G.G. George. 1990. Management and conservation of small populations. In: *Management and conservation of small populations.* T.W. Clark and J.H. Seebeck, eds. Chicago Zoological Society, Brookfield, Illinois, pp. 1-18.

Clark, T.W., G.N. Backhouse and R.C. Lacy. 1991. The population viability assessment workshop: A tool for threatened species management. *Endangered Species Update* 8:1-5.

Clarke, G.M. 1995. Relationships between developmental stability and fitness: Application for conservation biology.

Conserv. Biol. 9:18-24.

Ellis, S., K. Hughes, C. Kuehler, R.C. Lacy and U.S. Seal. 1992a. `Alala, Akohekohe, and Palila Population and Habitat Viability Assessment Reports. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Ellis, S., C. Kuehler, R.C. Lacy, K. Hughes and U.S. Seal. 1992b. Hawai`ian forest birds conservation assessment and management plan. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Franklin, I.R. 1980. Evolutionary change in small populations. In: *Conservation biology: An ecological/evolutionary perspective*, M.E. Soulé and B.A. Wilcox, eds. Sinauer Associates, Sunderland, Massachusetts, pp. 135-149.

Foose, T.J., R.C. Lacy, R. Brett and U.S. Seal. 1993. Kenyan Black Rhino Metapopulation Workshop Report. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Gilpin, M.E. 1989. Population viability analysis. *Endangered Species Update* 6:15-18.

Gilpin, M.E. and M.E. Soulé. 1986. Minimum viable populations: processes of extinction. In: *Conservation biology. The science of scarcity and diversity*, M.E. Soulé, ed. Sinauer Associates, Sunderland, Massachusetts, pp. 19-34.

Goodman, D. 1987. The demography of chance extinction. In: *Viable populations for conservation*, M.E. Soulé, ed. Cambridge University Press, Cambridge, pp. 11-34.

Hanski, I. and M. Gilpin. 1991. Metapopulation dynamics: a brief history and conceptual domain. *Biol. J. Linnean Soc.* 42:3-16.

Holling, C.S. (ed.) 1978. *Adaptive environmental assessment and management*. International Series on Applied Systems Analysis 3, International Institute for applied systems analysis. John Wiley and Sons, Toronto.

IUCN Species Survival Commission. 1994. IUCN Red List Categories. IUCN, Gland, Switzerland.

Lacy, R.C. 1993a. VORTEX: A computer simulation model for Population Viability Analysis. *Wildlife Research* 20:45-65.

Lacy, R.C. 1993b. Impacts of inbreeding in natural and captive populations of vertebrates: Implications for conservation. *Perspect. Biol. Med.* 36:480-496.

Lacy, R.C. (in press). Putting population viability analysis to work in endangered species recovery and small population management. In: *Conserving species dependent on older forests: A population viability workshop*, S. Fleming, ed. Parks Canada, Fundy National Park, Alma, New Brunswick.

Lacy, R.C. and T.W. Clark. 1990. Population viability assessment of the eastern barred bandicoot in Victoria. In: *Management and Conservation of Small Populations*, T.W. Clark and J.H. Seebeck, eds. Chicago Zoological Society, Brookfield, Illinois, pp. 131-146.

Lacy, R.C. and D.B. Lindenmayer. 1995. A simulation study of the impacts of population sub-division on the mountain brushtail possum, *Trichosurus caninus* Ogilby (Phalangeridae: Marsupialia), in south-eastern Australia. II. Loss of genetic variation within and between sub-populations. *Biol. Conserv.* 73:131-142.

Lacy, R.C, N.R. Flesness and U.S. Seal. 1989. Puerto Rican parrot population viability analysis. Report to the U.S. Fish and Wildlife Service. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Lande, R. and G.F. Barrowclough. 1987. Effective population size, genetic variation, and their use in population

management. In: *Viable populations for conservation*. M.E. Soulé, ed. Cambridge University Press, Cambridge, pp. 87-123.

Lindenmayer, D.B. and R.C. Lacy. 1995a. Metapopulation viability of Leadbeater's Possum, *Gymnobelideus leadbeateri*, in fragmented old-growth forests. *Ecological Applications* 5:164-182.

Lindenmayer, D.B. and R.C. Lacy. 1995b. Metapopulation viability of arboreal marsupials in fragmented old-growth forests: comparison among species. *Ecological Applications* 5:183-199.

Lindenmayer, D.B. and R.C. Lacy. 1995c. A simulation study of the impacts of population sub-division on the mountain brushtail possum, *Trichosurus caninus* Ogilby (Phalangeridae: Marsupialia), in south-eastern Australia. I. Demographic stability and population persistence. *Biol. Conserv.* 73:119-129.

Lindenmayer, D.B., V.C. Thomas, R.C. Lacy and T.W. Clark. 1991. Population Viability Analysis (PVA): The concept and its applications, with a case study of Leadbeater's Possum, *Gymnobelideus leadbeateri* McCoy. Report to the Forest and Timber Inquiry (Resource Assessment Commission), Consultancy Series No. FTC91/18, Canberra, Australia. 170 pp.

Lindenmayer, D.B., T.W. Clark, R.C. Lacy and V.C. Thomas. 1993a. Population viability analysis as a tool in wildlife conservation policy: With reference to Australia. *Environ. Manage.* 17:745-758.

Lindenmayer, D.B., R.C. Lacy, V.C. Thomas and T.W. Clark. 1993b. Predictions of the impacts of changes in population size and environmental variability on Leadbeater's Possum, *Gymnobelideus leadbeateri* McCoy (Marsupialia: Petauridae) using Population Viability Analysis: an application of the computer program VORTEX. *Wildlife Research* 20:67-86.

Mace, G.M. and R. Lande. 1991. Assessing extinction threats: Toward a re-evaluation of IUCN threatened species categories. *Conserv. Biol.* 5:148-157.

Mace, G. *et al.* 1992. The development of new criteria for listing species on the IUCN Red List. *Species* (19):16-22.

Mace, G. and S. Stuart. 1994. Draft IUCN Red List Categories, Version 2.2. *Species* (21-22):13-24.

Maguire, L.A. 1986. Using decision analysis to manage endangered species populations. *J. Environ. Manage.* 22:345-360.

Maguire, L.A., R.C. Lacy, R.J. Begg and T.W. Clark. 1990. An analysis of alternative strategies for recovering the eastern barred bandicoot in Victoria. In: *Management and Conservation of Small Populations*, T.W. Clark and J.H. Seebeck, eds. Chicago Zoological Society, Brookfield, Illinois, pp. 147-164.

Mirande, C., R. Lacy, and U. Seal. 1991. Whooping crane (*Grus americana*) conservation viability assessment workshop report. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota, USA.

Odum, A., et al. 1993. Aruba Island Rattlesnake Population and Habitat Viability Assessment (PHVA) Workshop. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Ruggiero, L.F., G.D. Hayward and J.R. Squires. 1994. Viability analysis in biological evaluations: Concepts of population viability analysis, biological population, and ecological scale. *Conserv. Biol.* 8:364-372.

Samuels, A. and J. Altmann. 1991. Baboons of the Amboseli basin: demographic stability and change. *Int. J. Primatol.* 12:1-19.

Sapolsky, R.M. 1982. The endocrine stress-response and social status in the wild baboon. *Hormones and Behavior* 15:279-292.

Sapolsky, R.M. 1986. Endocrine and behavioral correlates of drought in the wild baboon. *Amer. J. Primatol.* 11:217-227.

Seal, U.S. 1992. Genetic management strategies and population viability of the Florida panther (*Felis concolor coryi*). IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Seal, U.S. and R.C. Lacy. 1989. Florida panther population viability analysis. Report to the U.S. Fish and Wildlife Service. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Seal, U.S., J.D. Ballou and C.V. Padua. 1990. *Leontopithecus* population viability analysis workshop report. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

Seal, U.S., R.C. Lacy, K. Medley, R. Seal and T.J. Foose. 1991. Tana River Primate Reserve Conservation Assessment Workshop Report. IUCN SSC Captive Breeding Specialist Group, Apple Valley, Minnesota.

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

Shaffer, M.L. 1990. Population viability analysis. *Conserv. Biol.* 4:39-40.

Simberloff, D.A. 1988. The contribution of population and community biology to conservation science. *Annu. Rev. Ecol. Syst.* 19:473-511.

Soulé, M.E. (ed). 1987. *Viable populations for conservation*. Cambridge University Press, Cambridge.

Soulé, M., M. Gilpin, W. Conway and T. Foose. 1986. The millenium ark: How long a voyage, how many staterooms, how many passengers? *Zoo Biology* 5:101-113.

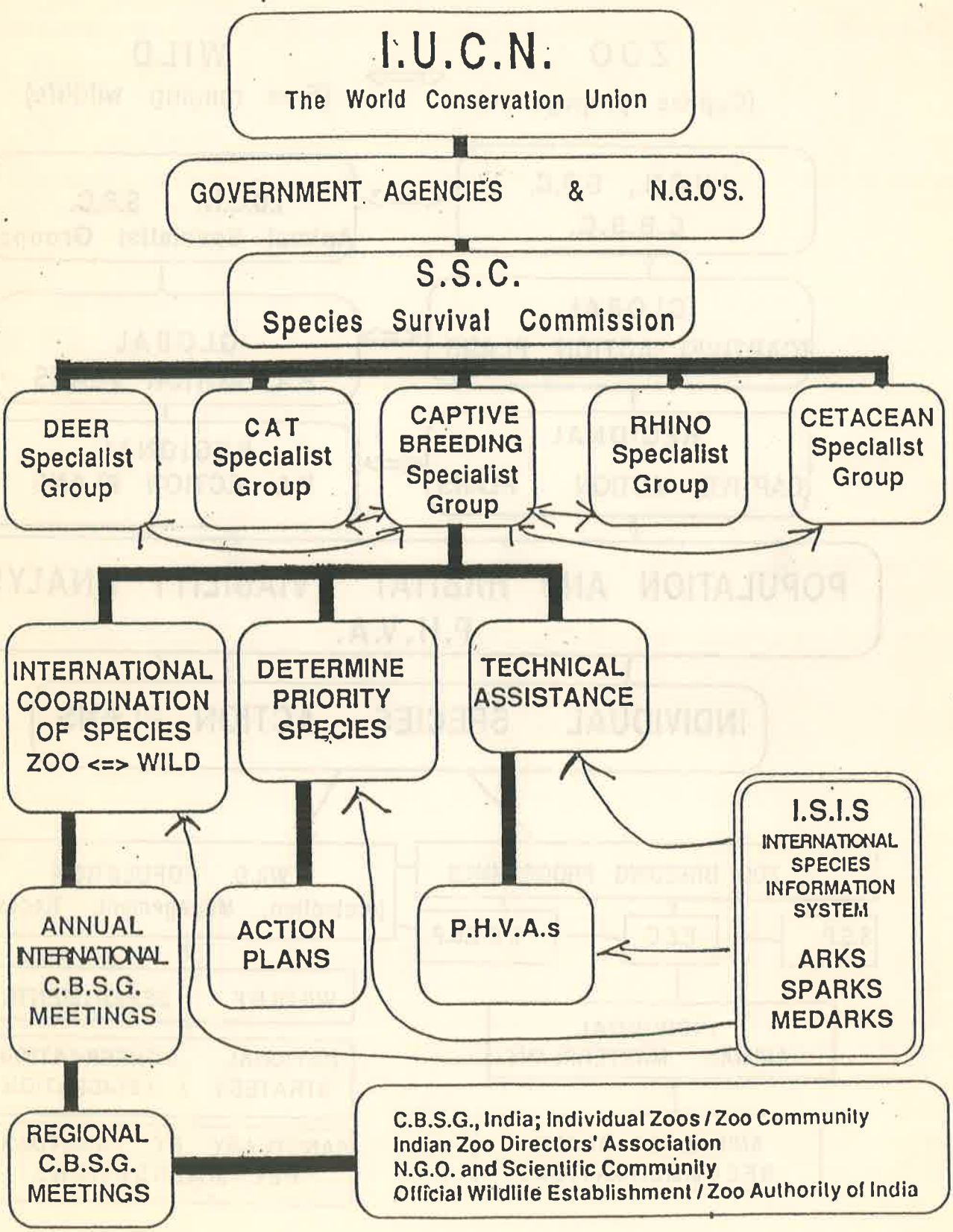
Starfield, A.M. and A.L. Bleloch. 1986. *Building models for conservation and wildlife management*. Macmillan, New York.

Walker, S. and S. Molur. 1994. Population and Habitat Viability Analysis (PHVA) workshop for Indian/Nepali Rhinoceros. Zoo Outreach Organisation/CBSG India, Coimbatore.

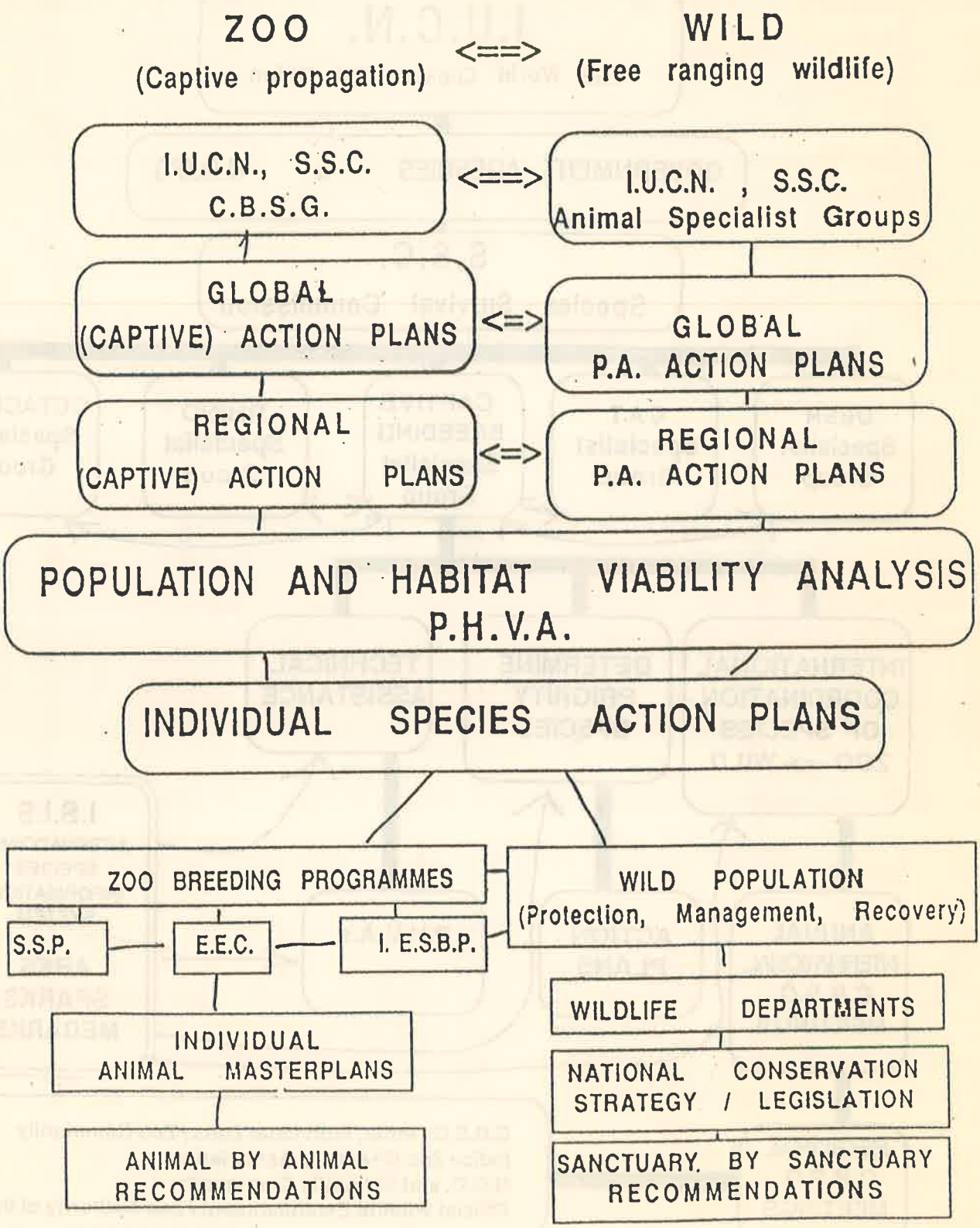
THE THREE "C's" OF CONSERVATION
HOW IT WORKS IN THE WORLD

COOPERATION. COORDINATION COMMUNICATION

ZOO <=> WILDLIFE ESTABLISHMENT <=> GOVERNMENT <=> N.G.O.'s



HOW IT WORKS
ZOO <= TO => WILD



IUCN/SSC RE-INTRODUCTION SPECIALIST GROUP
DRAFT GUIDELINES FOR RE-INTRODUCTIONS

IUCN
The World Conservation Union

INTRODUCTION

The policy guidelines have been drafted by the Reintroductions Specialist Group of the Species Survival Commission. They are intended to act as guidelines only and do not represent an inflexible code to conduct. Each reintroduction proposal should be reviewed on it's individual merits. It is planned that this document be regularly revised. Versions of this document will be drafted for specialist group e.g. plants, invertebrates.

1. DEFINITION OF TERMS

* "Re-introduction" : an attempt to establish a species in an area which was once part of its historical range, but from which it has become extinct. ("Re-establishment" is a synonym, but implies that the re-introduction has been successful")

* "Translocation" : movement of individuals or populations from one part of their range to another.

* "Re-enforcement/Supplementation" : addition of indivduals to an existing population of conspecifics.

* "Conservation Introductions" : the attempt to establish a species, for the purpose of species conservation, outside of the recorded distribution but within the appropriate habitat and eco-geographical area. Such activity should be undertaken only as a last resort when no opportunities for reintroduction into the original site or area exist.

2. AIMS AND OBJECTIVES OF RE-INTRODUCTION

A re-introduction should aim to establish a viable, free-ranging population in the wild, of a species or subspecies which was formerly locally extinct (extirpated). In some circumstances, a re-introduction may have to be made into an area which is fenced or otherwise protected, but it should be within the species' natural habitat and range, and require a minimum of long-term management.

The objectives of a re-introduction will include: to enhance the long-term survival of a species; to re-establish a keystone species (in the ecological or cultural sense) in an ecosystem; to increase biodiversity, provide long term economic benefits to local people, or a combination of these.

Re-introductions or translocations of species for short-term sporting or commercial purposes are not a concern of the RSG.

Re-introductions are generally long-term projects that require the commitment of long-term financial and political support.

3. MULTIDISCIPLINARY APPROACH

A re-introduction requires a multidisciplinary approach involving a team of persons drawn from a variety of backgrounds. They should include persons from: governmental natural resource management agencies; non-governmental organisations; funding bodies; universities; zoos or botanic gardens. Team leaders should be responsible for coordination between the various bodies and provision should be made for publicity and public education about the project.

4. PRE-PROJECT ACTIVITIES

4a. Biological

Feasability study and background research.

Assessment of taxonomic status of individuals to be re-introduced. The re-introduced individuals should be of the same taxonomic unit (and ideally closely related genetically) as those that were extirpated. Investigation of historical information about the loss and fate of individuals from the re-introduction area, as well as molecular genetic studies, should be undertaken in case of doubt. A study of genetic variation within and between populations of this and related taxa can also be helpful. Special care is needed when the population has long been extinct.

Detailed studies of the status and biology of wild populations (if they exist) to determine species' critical needs; for animals, this would include descriptions of habitat preferences, social behaviour, group composition, home range size, shelter and food requirements, foraging and feeding behaviour, predators and disease. For plants it would include biotic and abiotic habitat requirements, dispersal mechanisms, reproductive biology, symbiotic relationships (eg with mycorrhizae, pollinators), insect pests and diseases. A Population and Habitat Viability Analysis will aid in assessing significant environmental and population variables, and allow for long term population management.

Previous Re-introductions

Thorough research into previous re-introductions of same or similar species and wide-ranging contacts with experts in the field should be conducted prior to and while developing re-introduction protocol.

Choice of site

Should be within historic range of species and for an initial re-introduction have few, or no, resident wild population (to prevent disease spread, social disruption and introduction of alien genes). The re-introduction site should have assured, long-term protected status.

Evaluation of re-introduction site
Availability of suitable habitat: Reintroductions should only take place where the habitat and landscape requirementsof the species are satisfied, and likely to be sustained in the forseeable future. The area should have sufficient carrying capacity to sustain growth of the re-introduced population and support a viable population in the long run.
Identification and control of previous causes of decline: could include disease; over-hunting; over-collection; pollution; competition with or predation by introduced species; habitat loss. Where the release site has undergone degredation a habitat restoration programme should be initiated.

Availability of suitable release stock
Release stock ideally should be closely-related genetically to the original native stock.

If captive or artificially propagated stock is to be used, it must be from a self-sustaining population which has been soundly managed both demographically and genetically. Removal of individuals for re-introduction must not endanger the captive stock population. Stock must be guaranteed available on a regular and predictable basis.

4b. Socioeconomic and legal activities

Socioeconomic studies to assess costs and benefits of re-introduction programme to human population. Each reintroduction should be fully integrated with, and supported by, local communities, except where the security of the reintroduced population is at risk.

If cause of species decline due to human factors (eg. over-hunting, over-collection, loss of habitat), a thorough assessment of attitudes of local people to the proposed project is necessary to ensure long term protection.

Policy of country to re-introductions and to species concerned. Checking of existing national and international legislation and regulations, and provision of new measures as necessary. Reintroduction must take place with the full permission and involvement of the governmental agency of the receipient or host country.

If species pose a potential risk to life or porperty, these risks should be minimised and adequate provision made for compensation where necessary. Provisions made for crossing of international / state boundaries.

5. PLANNING, PREPARATION AND RELEASE STAGES

Construction of multidisciplinary team with access to expert technical advice for all phases of the programme.

* Approval of all relevant government agencies and land owners, and coordination between national and international conservation organizations.

* Develop transport plans for stock to country and site of re-introduction.

* Identification of short - and long-term success indicators and prediction of programme duration.
* Securing adequate funding for all programme phases.

* Design of pre- and post-release monitoring programme

* Appropriate health screening and genetic assessment of release stock. Health screening of related congenerics to be undertaken.

* Appropriate veterinary or horticultural measures to ensure health of released stock throughout programme. This to include adequate quarantine arrangements.

* Determination of release strategy (acclimatization of release stock to release area; behavioural training group composition, number, release patterns and techniques; timing).

* Establish policies on interventions.

* Conservation education for long-term support; professional training of individual involved in long-term programme; public relations through the mass media and in local community; involvement where possible of local people in the programme.

6. POST-RELEASE ACTIVITIES

* Post release monitoring.

* Demographic, ecological, genetic and behavioral studies of released stock.

* Restocking policy established and undertaken.

* Study of processes of long-term adaption by individuals and the population.

* Collection and investigation of mortalities.

* Interventions (eg supplemental feeding; veterinary aid; horticultural aid) when necessary.

* Decisions for revision or discontinuation of programme where necessary.

* Habitat protection or restoration to continue where necessary

* Continuing public relations activities including education and mass media coverage.