Conservation and evolution

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Contents

The study by Rick *et al.* (1977) on genetic variation in *Lycopersicon* provides a tantalizing clue favouring Model 3 versus Model 2. As shown in Fig. 3.10, the excess of observed heterozygotes over the expected appears to be related to percentage cross-pollination. Heterozygosity of these populations and cross-pollination are highly correlated ($r = 0.81$; $P<0.01$), so the negative association of heterozygote excess with outbreeding suggests that the locus-specific enhancement of fitness increases as the overall heterozygosity decreases among populations. This may be the first evidence from natural populations for an asymptotic relationship between genetic variation and fitness. Earlier experimental work by Crow and his colleagues (Temin, Meyer, Dawson and Crow, 1969; Crow, 1970) provides additional support for the asymptotic model. They found that as *Drosophila* were inbred, their viability drops off slowly at first, and more rapidly as inbreeding reached higher levels.

It would be premature to base a conservation strategy on such a subtlety, but if Model 3 turns out to be a general rule in population genetics, it would mean that the absence of obvious inbreeding effects on the viability or fecundity of a managed population during the first few generations of captive breeding or intensive management is no guarantee that such immunity would persist at higher levels of inbreeding.

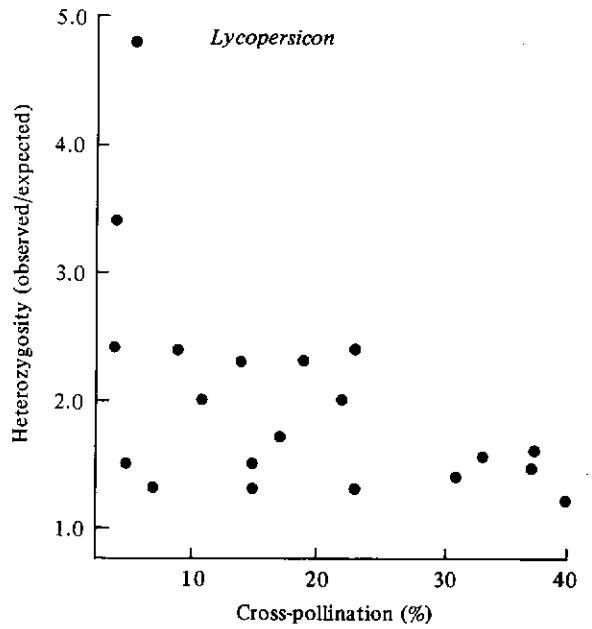

Fig. 3.10 The inverse relationship between cross-pollination and heterozygote excess in *Lycopersicon*. After Rick, Fobes and Holle (1977).

Finally, the correlation of fitness with heterozygosity in natural populations need not signal the existence of single gene overdominance. An alternative is dominance i.e. recessive deleterious genes; this is mentioned (p. 51) in the discussion of the work of Singh and Zouros (1978). One of the major tasks of empirical population genetics is to discover the mechanisms underlying the purported correlation.

3.3 Inbreeding depression

3.3.1 Inbreeding defined

We begin our discussion of inbreeding with an experiment performed on Poland China swine almost forty years ago (McPhee, Russel and Zeller, 1931). The experiment was designed to establish the effects of sib (brother–sister) mating. As it turned out, the experiment was rather short-lived, lasting only two generations. The reason for its discontinuation at this point was a precipitous drop in the fitness of the inbred line. As shown in Table 3.7 the mean number of pigs per litter dropped from 7.15 in the general herd to 4.26 in the second generation of inbreeding. This decline in fecundity was accompanied by a drop in survivorship of pigs by more than half. When the values for fecundity and survivorship are multiplied, one obtains the number of surviving offspring per litter. In the general herd this number is four; it is only one in the F_2 inbreds. Concomitant with this 75% loss in productivity was an equally serious change in the ratio of males to females; it changed from 1.1:1.0 in the general herd to 1.6:1.0 in the F_2 inbreds, thus further aggravating the decline in fecundity and survivorship.

The implications of results like these for the conservation of large organisms are profound, not only for the breeders of endangered species in zoos, but also for the managers of wildlife reserves. But before pursuing this topic we must first define inbreeding in a more quantitative fashion, and explain

TABLE 3.7 *Vital statistics of a herd of Poland China swine and the progeny of two generations of sib mating*

	No.	Inbreeding coefficient		Size of litter	Percentage born alive	Percentage raised to 70 days	Sex ratio
		Dam	Litter				
General herd	694	0+	0+	7.15	97.0	58.1	109.7
F_1 inbred	189	0.09	0.33	6.75	93.7	41.2	126.1
F_2 inbred	64	0.33	0.42	4.26	90.6	26.6	156.0

From McPhee, Russel and Zeller (1931) after Wright (1977).

some of the theories that attempt to account for its effects. Those familiar with inbreeding genetics might wish to skip to section 3.3.4.

One of the most important points to grasp about inbreeding is that it is a *relative* concept. For example, John may be homozygous for deleterious genes at many more loci than Mary, but Mary, strictly speaking, may be more inbred. This is because the formulation of inbreeding is in terms of the proportion of homozygous loci in an individual relative to that proportion in the general population. That is, John may be a member of a tribe having a strong taboo against incest and inbreeding, and yet everyone in the tribe can trace his ancestry back to a single matriarch and her husbands. Mary, on the other hand, might be a typical American of mixed ancestry, as well as being the daughter of first cousins. In genetic terms, John is more homozygous, but Mary is more inbred.

Another way of saying this is that an individual who is a product of inbreeding within a very heterozygous base population can be more heterozygous than a non-inbred individual from a genetically homogeneous population. We will return to this point in the discussion of the relationship between inbreeding and genetic load.

Inbreeding means mating of close relatives, individuals, that is, who are likely to share some of their genes because they have one or more ancestors in common. The most widely used measure of inbreeding, or consanguinity, is the inbreeding coefficient of Wright (1921), denoted by F. This coefficient is most easily understood as the probability that the two alleles of a particular locus in an individual are identical by descent. For example, consider individual E in Fig. 3.11; she is the product of a brother–sister (sib) mating, so she has only one set of grandparents. When considering only one gene (we use gene and locus interchangeably), the total number of copies present in her grandparents was four. We don't mean that the two grandparents had four biochemically distinct alleles; these four copies may have been molecularly identical, but for our purposes they are still considered distinct. Now we ask what is the probability (this probability is called F) that E is homozygous for any one of these four copies present in the grandparents. The answer is 1/4. This can be seen in the following way. The probability that A transmitted a copy of one of his two alleles, say a', to C is 1/2. The probability that another copy of a' was transmitted to D is the same, 1/2. Therefore the probability that C and D both received one copy of the identical gene a' is 1/4. Given that both C and D received one copy of a', the probability that E is homozygous for a' is 1/4; therefore, the probability that E is homozygous $a'a'$, *and* that both of the a' alleles came from A is $1/4 \times 1/4 = 1/16$. Because A and B together had four alleles of the gene in question, and because the probability is 1/16 that E will be homozygous for a *particular* one, the probability that she is homozygous by descent for *any* of the four copies is $1/16 + 1/16 + 1/16 + 1/16 = 1/4$. Precise methods for calculating inbreeding

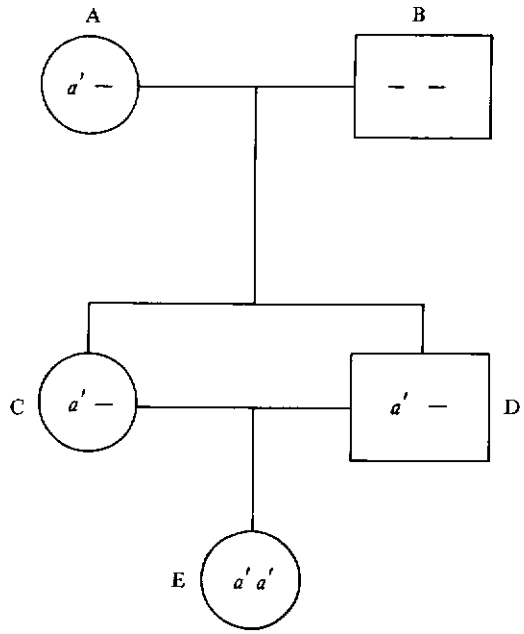

Fig. 3.11 A pedigree showing identity of a gene by descent. See text.

coefficients can be found in Cavalli-Sforza and Bodmer (1971), Crow and Kimura (1970), Pirchner (1969) and Falconer (1960).

Another way to interpret F is in terms of the relative amount of heterozygosity. In the previous example we showed that an individual who is the offspring of a sib mating has 25% chance of being homozygous by descent at a given polymorphic locus. This is equivalent to saying that she will have alleles identical by descent at 25% of all of the loci which are not already fixed (that is, were polymorphic) in the base population. Putting it slightly differently, she will be, on the average, 75% as heterozygous as the average individual in the base or source population. F, then, is a direct estimate of the genetic variability *relative* to the variability in the non-inbred population.

3.3.2 The rate of inbreeding

Next, we examine a question of critical importance in conservation genetics – the *rate of inbreeding*. The rate at which genes become fixed (alternatively, the rate at which heterozygosity is lost) depends on the breeding system. The closer the relationship between parents, the higher the rate of fixation. The most intense form of inbreeding is selfing or self-fertilization. In theory, half of the remaining heterozygous loci become fixed every generation in a

selfing population. Hence, the per generation inbreeding is $F = 0.5$. Sib mating or offspring–parent mating gives a per generation loss of heterozygosity of 0.25, as we saw above. The inbreeding coefficients for sib mating as well as for other systems are given for five generations in Fig. 3.12. Note that the per generation change in the inbreeding coefficient, ΔF, is a constant for any breeding system.

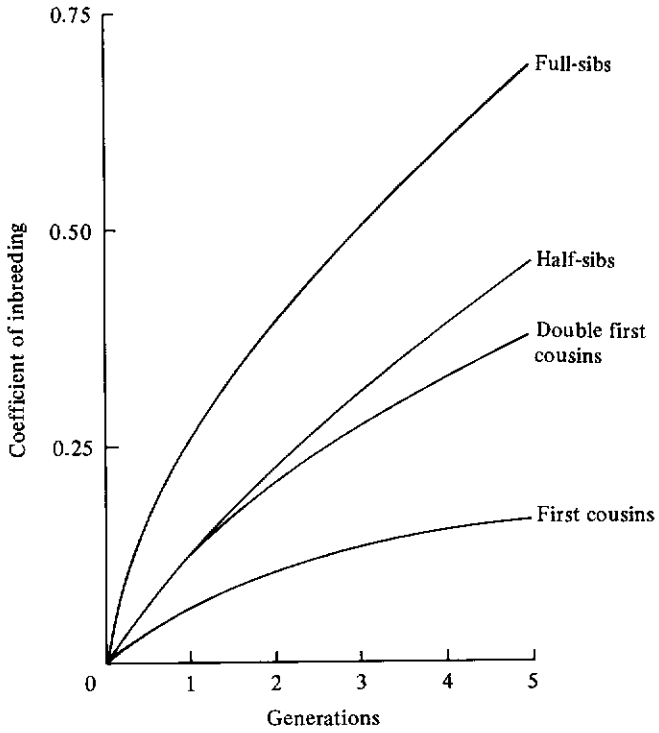

Fig. 3.12 Increase in homozygosity during inbreeding. After Underwood (1979).

3.3.3 Inbreeding depression: theory

Aside from its effect on heterozygosity, phenotypic changes are often observed in inbred lines. For simple Mendelian traits, the changes are random. Sewell Wright (1977) recounts his ability to distinguish his inbred lines of guinea pigs due to unique colour patterns and other traits that had become fixed in each line. More is said on this topic in section 3.5.5.

Inbreeding also causes a change or shift in the means of some genetically determined quantitative characters. But this shift, unlike genetic drift, is directional; *it is always towards the direction of the phenotype expressed by (homozygous) recessive alleles.* This is where the term *inbreeding depression*

comes from. One might ask why this is necessarily so – why do recessive alleles when homozygous, produce inferior or 'depressed' phenotypes? The answer has to do with exposure. Dominant alleles are always exposed, or subject to natural selection; hence, a deleterious dominant is readily eliminated in a population. On the other hand, a deleterious recessive can persist at low frequency indefinitely, since it is rarely 'seen' in the homozygous state. The effect of inbreeding on a trait, in the presence of recessives, can be stated more precisely: if the variation for a particular phenotypic character has any degree of dominance or overdominance (heterozygote superiority), then there will be a shift in the average expression of the character towards the homozygous recessive phenotype, as the following example shows.

In an infinitely large, randomly breeding population, the equilibrium (Hardy–Weinberg) genotype frequencies, considering a single locus, are

$$p^2 + 2pq + q^2 = 1$$

where p is the frequency of allele a_1, the dominant allele, and q is the frequency of a_2, the recessive allele. Upon inbreeding, these Hardy–Weinberg frequencies change to

$$(p^2 + pqF) + (2pq - 2pqF) + (q^2 + pqF) = 1$$

Note that the proportion of homozygotes increases at the expense of the heterozygotes. Now, the reason the mean shifts towards the recessive (a_2) side is illustrated in Fig. 3.13. Assume a population is subdivided into a number of lines, each of which is inbred at a level F. The frequencies of the two alleles a_1 and a_2 within a line are p and q, respectively, and the average frequencies for the whole population (all lines together) are $\bar{p}$ and $\bar{q}$. For the sake of simplicity we have set $p = q = 0.5$ and we have set d, the genotypic value (average phenotype) of the heterozygote, equal to the genotypic value of the dominant, or 1.0; the respective value of the recessive is -1.0. This is merely a mathematical way of expressing complete dominance. Upon inbreeding, the change in the mean is $-2d\bar{p}\bar{q}F$, and the mean genotypic value of the average completely inbred ($F = 1.0$) population is (Falconer, 1960)

$$\begin{aligned} M_F &= M_0 - 2dpqF \\ &= 0.5 - 2(0.25) \\ &= 0 \end{aligned}$$

where M_0 is the mean genotypic value of the randomly breeding population and M_F is the value for the average inbred population. Of course, a completely inbred population will be composed of individuals that either are all a_1a_1 or all a_2a_2; 50% of lines will be monomorphic for one genotype and 50% will be monomorphic for the alternative genotype. It is by averaging together all the inbred lines that we obtain $M_F = 0$. As long as there is any

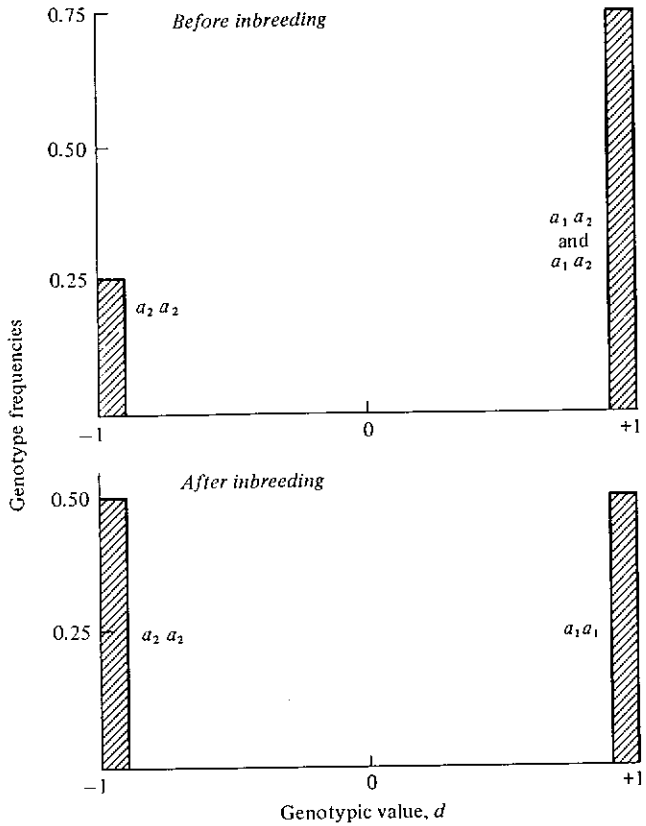

Fig. 3.13 Inbreeding depression as the result of dominance. The genotypic value of the dominant allele is 1; that of the recessive is −1. Before inbreeding, the mean genotypic value for the population is 0.5, as indicated by the symbol M_0. After inbreeding (below), all lines are homozygous, half for allele a_1 and half for allele a_2. Therefore, half of the lines have a genotypic value of −1 and half have a genotypic value of 1; the mean (M_F) is zero. After Falconer (1960, p. 113).

dominance, inbreeding always produces a phenotypic shift towards the recessive.

To counteract this tendency it is common practice for breeders to introduce 'outside' genes from relatively unrelated colonies. This procedure reduces the frequency of unfit homozygous recessives in the mixed population. Bruce (1910) explained this effect mathematically for a single locus. Assume for example, that we have two lines each having a different recessive allele with frequencies q and q', respectively. If we pick at random the same number of individuals from both lines, the overall proportion of unfit homozygous recessives is $(q^2 + q'^2) / 2$. But if we produce a *new* line by

crossing individuals from one line with individuals from the other, the frequency of homozygous recessives becomes $(q/2)^2+(q'/2)^2+2(q/2)\ (q'/2)$ which is less than or equal to $(q^2 + q'^2)/2$. This assumes (conservatively) that qq' is as unfit as qq or $q'q'$.

Crossing has an even more salubrious effect if the two lines tend to have recessives at different loci. For example, line A might have an unfit recessive, m, at locus M, and line B has unfit recessive n at locus N. Assume the frequency of m and n has the value 0.2 in each population respectively. The proportion of homozygous recessives in a new line formed by crossing equal numbers from A and B will be $2(0.1)^2 = 0.02$, in contrast to 0.04 in line A or B.

3.3.4 Inbreeding depression and fitness

In this section we delve more deeply into the actual forms taken by the deleterious effects of inbreeding. The expression of inbreeding depression is biased towards certain kinds of characteristics. That is, inbreeding depression neither affects all characters uniformly, nor are these effects random or unpredictable with respect to the traits in which they appear. In fact, we can predict quite accurately which characteristics will be depressed the most upon inbreeding.

The careful reader will have anticipated this predictability of inbreeding effects. Recall that the average phenotype of traits upon inbreeding moves away from the dominant or overdominant phenotype and towards the recessive. Traits, therefore, which have a significant amount of dominance or overdominance (non-additive genetic variance) will change the most. Furthermore, a shift towards the phenotypic expression of recessive genes is tantamount to a decline in fitness because, as already mentioned, a disproportionate number of deleterious alleles are recessive.

Now, in what kind of characters is dominance typically observed? By and large, they are traits related to reproduction; in the parlance of quantitative genetics, they are 'fitness characters' (Robertson, 1955). This means that we can expect to observe the most inbreeding depression in characters such as fecundity (total reproductive output), fertility (ability to produce viable gametes or zygotes), developmental rate or age at sexual maturity, litter size and analogous traits. In contrast, characters, the states of which are not critical to reproduction, are less affected by inbreeding. Finally, the traits which are least affected by inbreeding are those that can vary greatly without affecting the viability or reproductive contribution of individuals.

Another way of expressing the above breakdown of traits is in terms of *heritability*. A long exegesis on heritability would be inappropriate here (see Bodmer and Cavalli-Sforza, 1976 for an excellent summary). For our purposes it is sufficient to point out that heritability is a measure of the genetic determinism of a trait or of its correlation among close relatives. Such

correlations are high if the genes that control a trait are acting in an additive fashion, that is, if there is relatively little dominance or overdominance. The experience of breeders has led them to generalize that reproductive traits generally have low heritability (little additive variation) while traits which are apparently less relevant to reproduction and survival have high heritabilities.

Table 3.8 summarizes the heritabilities of some traits in animals. Note that the heritabilities for coat pattern, tail length and body conformation all tend to be high (50% to 90%), whereas those for reproductive characters are usually 20% or less. The rule of thumb is that the characteristics with the lowest heritabilities are those which will be the most depressed by inbreeding, assuming a relatively large amount of dominance variation.

At the beginning of this chapter we gave an account of inbreeding depression in Poland China swine. This example is just one of several hundred in the animal and plant breeding literature. A complete review of the literature would serve no purpose here, but some additional examples are helpful in reaching some general conclusions.

Sewell Wright (1977) recently summarized his extensive and long-term studies on inbred guinea pigs. Three points are especially significant. First, out of thirty-five original lines undergoing inbreeding, only half survived for nine years, and only five were vigorous enough to warrant intensive study. Second, in these five relatively vigorous lines, the effective fecundity, compared to non-inbred controls (young raised per mating year) was only 30%.

TABLE 3.8 *Heritabilities of various traits*

Trait	Heritability	Source
Finger print ridges in humans	0.95	Holt (1961)
Amount of spotting in Friesian cattle	0.95	Falconer (1960)
Stature in human males	0.79	Osborne and De George (1959)
Femur length in mice at 3 months (male-son)	0.79	Leamy (1974)
Tail length in mice	0.6	Falconer (1960)
Length of wool in sheep	0.55	Falconer (1960)
Abdominal bristle number in *Drosophila*	0.5	Falconer (1960)
Skull length in mice at 3 months (male-son)	0.50	Leamy (1974)
Combined molar length in mice	0.30	Bader (1965)
Milk yield in cattle	0.3	Falconer (1960)
Egg production in poultry and *Drosophila*	0.2	Falconer (1960)
Litter size in pigs and mice	0.15	Falconer (1960)

Third, the overall fitness of the inbred lines compared to the controls is worst in unfavourable (nutritionally poor) environments. That is, the inbreds are less resistant to stressful conditions (Lerner, 1954; Parsons, 1971).

Bowman and Falconer (1960) studied the effects of inbreeding on litter size in mice. The decline was from 7.77 to 4.58 in five generations; expressed in terms of F, this is 0.6 of a mouse (7.7%) per 10% increase in F. (Recall that ΔF, equal to 10% is slightly less than the ΔF in a half-sib mating scheme.) Only one out of twenty inbred lines remained at the control level for litter size after generation twelve.

The difference between inbred and outbred strains of Holstein-Friesian cows was studied by Tyler, Chapman and Dickerson (1949). They found little effect of inbreeding on body dimensions (note that body dimensions are not fitness characters) but recorded a depression in milk and butterfat production of 6.2% and 5.8%, respectively, for 10%ΔF.

In gallinaceous birds, the effect of inbreeding appears to be related to the history of the species in captivity; the longer they have been domesticated, the less the inbreeding depression. Table 3.9 summarizes the results of several studies of Abplanalp and coworkers (Abplanalp, 1974). Note that the decline in fitness is least in chickens and turkeys, and most in Japanese quail and chukar partridge; the latter two species are much less domesticated than the former two. Domesticated species have a history of selection and inbreeding; i.e. they have been partially purged of their deleterious genes, so inbreeding depression concomitant with further inbreeding is relatively less severe.

Wright (1977), in reviewing the history of inbreeding in maize, reiterates that the fitness characters show the most drastic decline. Most lines

TABLE 3.9 *Effect of 25% inbreeding on the relative performance of four gallinaceous species; performance of non-inbred lines equals 100*

	Performance as percentage of non-inbred birds			
Trait	Chicken	Turkey	Japanese Quail	Chukar
Hatchability: embryo inbred	90.0	83.4	72.2	71.3
hen inbred	97.0	92.1	89.3	89.1
Fertility	99.1	98.8	79.2	71.1
Viability of females	94.3	90.7	81.5	92.1
Egg production	90.4	89.5	83.9	84.1
Total reproduction	74.4	61.6	35.9	34.1
'B' = inbreeding depression	1.183	1.938	4.098	4.303

From Abplanalp (1974).

deteriorated so rapidly upon inbreeding that they could not be saved. Those that survived to nine or ten generations of selfing deteriorated in height (27%), length of ear (28%) and, most dramatically, in yield (61%).

In sum, these studies point to the universality of inbreeding depression. More particularly, there is remarkable agreement between the inbreeding coefficient and loss of fitness, namely, a ΔF of 10% generally corresponds to a 5–10% decline for a particular reproductive trait. When considering *total reproductive performance* (rather than isolated characteristics) the decline in fitness jumps to a stunning 25% or so for species or lines that have not been extensively inbred in the past (Tables 3.7 and 3.9).

The decrease in fitness is less for most domesticated species and for previously inbred or selected lines of experimental organisms. As suggested above, this apparently less severe inbreeding depression is probably the result of the 'purging' effect of selection and inbreeding. Any inbreeding is likely to remove some of the deleterious genes from a line. For example, Roberts (results cited in Falconer, 1960) performed inbreeding experiments on mice stocks obtained by crossing inbred laboratory strains. Only two out of thirty lines were lost after three generations of sib mating. Such a high survival rate of lines is probably a consequence of the rarity of deleterious recessives in the original inbred strains.

The experience of plant breeders also leads to the conclusion that a history of slow or episodic inbreeding tends to decrease the severity of later inbreeding episodes. Many more plants than animals are prevailingly self-fertilizing, and it is possible to ask if species which are predominantly selfing have less inbreeding depression than would be expected. Young and Murray (1966) noted that the degree of inbreeding depression in domesticated plants correlated with the typical amount of self-pollination; in order of increasing cross-pollination and inbreeding depression, these plants are barley, cotton, tomato and corn. In general, self-pollinating species show less heterosis and less inbreeding depression than do out-crossing forms.

There are 'costs', however, to the purging of a stock by inbreeding. First, most attempts fail, so one must start with many lines if one hopes to pull some through the 'inbreeding crisis'. Second, a successfully purged inbred line, once obtained, will be genetically different from the outbred population in many, random, directions. Notwithstanding such costs, it is apparent that once a line has safely passed through one bout of inbreeding, it is more likely to make it through others because it will retain progressively fewer deleterious recessives.

It is clear from the previous paragraphs that inbreeding can be seen to produce two opposing effects: on the one hand it can purge some deleterious genes; on the other hand, it can fix some deleterious genes. Whether a line survives a period of inbreeding, therefore, is a matter of chance. If by chance no lethals or subvital genes are fixed, the line will survive. If, however, some

such genes become fixed or reach a high frequency, then the line will probably be lost. *The critical question then is what is the ratio between lines that are purged and survive versus lines that go extinct?* The value of this ratio determines whether inbreeding is a potentially useful tool for conservation.

On this question the data speak rather loudly. Above we noted that only half of Rommel and Wright's guinea pig lines survived for nine years (approximately 11.5 generations of sib-mating). Bowman and Falconer (1960) inbred twenty lines of house mice; only one line survived after generation 12, and only ten lines survived to generation 5. Using wild mice, Lynch (1977) found that only two out of fourteen lines survived for six generations of sib-mating. The results for *Drosophila* are essentially the same; only about 10% of lines survive more than ten or twenty generations of sib-mating (Clayton, Knight, Morris and Robertson, 1957; Wallace and Madden, 1965).

Abplanalp reported that only eight out of 279 lines survived a large inbreeding experiment with white leghorn chickens. The material for the above studies was domesticated stocks, so it is probable that they were all partially purged of their deleterious recessives by population bottlenecks and selection. Thus, one could anticipate that the survivorship of inbred lines would be even less with a foundation stock fresh from nature. In summary, between 5% and 20% of lines survive after F reaches values above 0.80.

One of the myths about inbreeding is that there exists an inbreeding depression minimum, and that once a line succeeds in traversing safely this genetic purgatory, it is cleansed of deleterious genes. The basis for this misconception is the existence of apparently fit inbred domesticated lines such as white rats. Indeed, there exist apparently* homozygous lines, but it is not usually appreciated that these lines typically are severely handicapped with respect to many traits, and would certainly not survive in nature. For example, Lindstrom (1941) found that 677 *useful* lines of inbred maize had been produced by experimental stations in the US, but these were only 2.5% of all those lines that had been started. Further, the average yield of these was only 30% that of F_1 hybrids. It is probably extraordinarily rare for an inbred line to be as fit overall as an outbred population.

3.3.5 Inbreeding depression and behaviour

Up to this point we have stressed the effects of inbreeding depression on the reproductive functions. Certainly reproductive characters are usually profoundly depressed by inbreeding, but other kinds of traits can be seriously

* It is now accepted that inbred lines are never as homozygous as predicted from simple theory (e.g. Enfield, 1977; Eriksson, Halkka, Lokki and Saura, 1976); natural selection apparently acts through heterozygote superiority to impede fixation.

affected as well. The reason that we have emphasized reproductive depression is that breeders and experimenters find it practical and relevant to study these. But an overemphasis on such traits as fecundity could lead one to believe that if an inbred stock is relatively fecund, for example, then it should be relatively fit overall, a very misleading assumption. Fecundity and viability in the laboratory, farm or zoo is probably useless in predicting how a stock will perform in nature. This because viability and reproduction in nature depend on a vast and subtle integration of physiological and behavioural phenomena. Whereas an inbred stock might perform well in a sheltered environment, it is likely to be severely handicapped when completely on its own. White leghorn poultry, for example, are champion egg layers, but no farmer would bet on their success in a hedgerow.

To take a more concrete example, laboratory mice (*Mus musculus*) are rarely exposed to the environmental extremes encountered by their wild cousins. Hence, a deterioration of certain abilities necessary for survival in the wild might be expected in laboratory mice, and many such evolutionary changes would go unnoticed. For example, the loss of the capacity to make a warm, protective nest would, in a laboratory strain, have little or no effect on fitness, but such a change in a wild population would lead to death of young from exposure, and thus to extinction. Are behavioural traits of this type likely to be depressed by inbreeding?

They are. Lynch (1977) determined that the amount of nesting material used by mice each day (nesting score) behaves genetically in the same way as fitness characters such as litter size. That is, inbreeding decreases nesting scores within lines, and crosses between inbred lines give substantial heterosis. Fig. 3.14 shows how nesting score varies among some inbred laboratory strains in comparison with a cross between them and in comparison to a wild strain. One of the six inbred strains, *BALB*, builds much bigger nests than the wild strain (this could be as non-adaptive as building too small a nest), and two of the inbred strains build very small nests. Such aberrant behaviour, while perhaps of little significance in the laboratory, could prove disastrous in nature.

There is a rich supply of anecdotes regarding inbreeding effects. A recent example concerns the brown-eared pheasant *Crossoptilon mantchuricum*. In the West all brown-eared pheasants (descended from a male and two females captured in northern China in 1864) have been producing mostly non-fertile eggs. Eggs obtained from artificially inseminated hens are fertile, and the infertility was traced to the low libido of the cocks, according to researchers at Cambridge University (Anon., 1977). Inbreeding problems are notorious in popular breeds of dogs; hip dysplasia and ocular diseases are particularly common.

Behavioural deterioration in traits related to competition will be detrimental in nature, if not on the farm or in the field. Several studies suggest

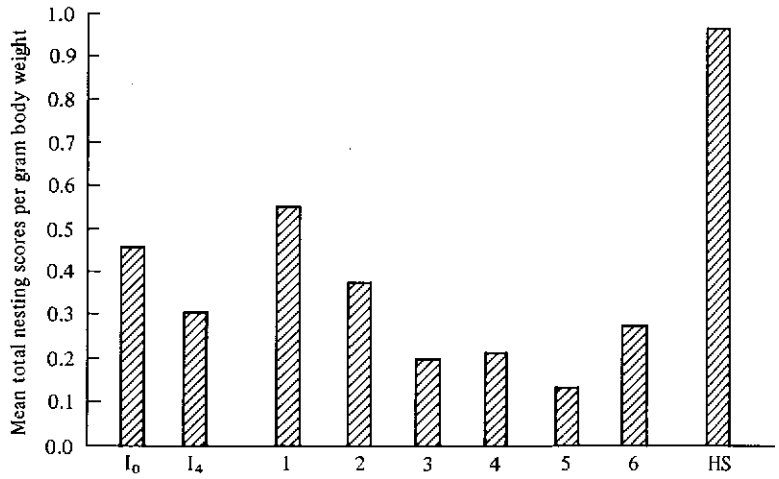

Fig. 3.14 Mean nesting scores for matings between wild caught mice (I_0), the fourth generation of full-sib matings derived from this natural population (I_4), six inbred strains (1 is *BALB/cJ*, 2 is *CBA/J*, 3 is *C3H/HeJ*, 4 is *C57BL/6J*, 5 is *DBA/1J* and 6 is *A/J*) and an eight-way cross among inbred strains (HS). After Lynch (1977).

that inbreeding severely depresses competitive ability. Latter and Robertson (1962) showed a strong effect of inbreeding on competitive ability in *Drosophila*. Mertz, Cawthorn and Park, (1976) ran competition experiments between two species of flour beetle, *Tribolium castaneum* and *T. confusum*. One of the variables in these experiments was the level of inbreeding. In a reanalysis of their results, J. W. Senner (in preparation) demonstrated a very strong depression of competitive ability, apparently attributable to inbreeding. In these examples the depression in competitive ability could be just another expression of a loss of fecundity, since any decline in reproductive fitness should reduce competitive ability.

The results of Garten (1976), discussed on p. 54 are also relevant here. Garten found that intraspecific competitiveness, as measured by aggression in oldfield mice, was positively correlated with mean heterozygosity in the population from which the mice were collected. Such studies reinforce our suspicion that *any* loss of genetic variability, whether due to natural causes (small population size, bottlenecks, directional selection) or to artificial inbreeding, is going to reduce the chances of survival in the wild.

3.3.6 Advantages of inbreeding

For certain purposes, inbred lines have clearcut advantages over non-inbred lines. Their most attractive characteristic is phenotypic uniformity under controlled conditions. In biological experimentation, the most repeatable

results are obtained when the experimental material is genetically homogeneous. Much pure and applied research in physiology, developmental biology, immunology, endocrinology and pharmacology depends on inbred strains. The problem with inbred organisms, however, is that they are highly sensitive to variation in the environment, particularly nutrition and temperature. The best of both worlds, that is homeostasis in a fluctuating environment and phenotypic uniformity, is achieved by crossing two or more inbred lines with good 'combining' characteristics. The hybrid offspring are genetically identical and heterozygous, the latter tending to minimize their sensitivity to environmental heterogeneity.

Another apparent advantage of hybrids between some inbred lines is their superior productivity. The best known example is hybrid corn. This brings us to a very important question: Why isn't it possible to assemble all the best genes together in a single homozygous line, assuming heterosis results from the masking of deleterious recessives? Indeed, this is a possibility for specific agricultural or laboratory purposes, but because of the thousands upon thousands of genes involved, a project of this magnitude would be extraordinarily tedious and difficult, especially for slowly reproducing species. Even for maize, which has been bred for many decades, such an ideal strain is unlikely to replace hybrids between inbred lines for the forseeable future (Eberhart, 1977).

Whereas it is our opinion that inbreeding under most circumstances is anathema for conservation programmes, special circumstances may permit special means. In an organism, for example, that for some reason is already very homozygous, further inbreeding might do little harm and could make it easier to maintain in a domesticated or semi-domesticated state. Just such a protocol was recommended by Slatis (1960) who found that inbreeding depression in the European bison, *Bison bonasus* was nearly absent. Perhaps this is because this species has never been abundant in historical times and recently passed through a bottleneck of seventeen *related* individuals. It should be recognized, however, that the consequence of intense inbreeding, assuming the unlikely result of survival without serious depression in viability and fecundity, is the relinquishing of future adaptive options, as well as the appearance of random changes in the phenotype.

3.4 The basic rule of conservation genetics

It would appear that there is no safe amount of inbreeding for normally outbred organisms. And even changes in level of heterozygosity, such as occur normally in natural populations, might cost populations a certain amount of fitness. Of course, all species cannot be maintained in a completely outbred state, but it behoves us to minimize inbreeding and the loss of genetic variation. Notwithstanding this, inbreeding at low intensities is

quite common in nature, as well as in domestic stock, so, *a priori*, it would appear that deleterious genes can be eliminated by natural selection before being fixed, given that the rate of inbreeding is low enough. Theory and experience tell us that the smaller the population, the more difficult is selection's task in the weeding out of such genes; the reason is that selection is no match for genetic drift at very small population sizes.

Is there, then, a threshold rate of inbreeding, above which fitness relentlessly declines, and below which fitness can be maintained? The answer is a qualified 'yes'. It is a qualified response because no two populations are alike: those with a large load of deleterious genes will tolerate less inbreeding than those with a lower genetic load. Incidentally, species with high levels of heterozygosity may have relatively high genetic loads (Soulé and J. W. Senner, in preparation) so there is some hope of making rough predictions of the inbreeding tolerance of different species or stocks.

The basis for our 'yes' answer regarding a maximal rate of inbreeding is an empirical rule of thumb used by animal breeders. The rule is that natural selection for performance and fertility can balance inbreeding depression if ΔF per generation is no more than about 1% (Franklin, 1980) or 2–3% (Dickerson *et al.*, 1954; Stephenson, Wyatt and Nordskog, 1953). If ΔF values are higher than this, natural selection is unable to offset the tendency for the fixation of deleterious recessives. Apparent exceptions are very rare. We prefer the lower (1%), more conservative value, for two reasons. First, domestic animals from which the rule is derived are already inbred to some degree and therefore have less genetic load than their wild progenitors. As any genetics student knows, it is virtually impossible thoroughly to eliminate a complete recessive from a population, even if it is lethal as a homozygote; nevertheless, such genes can be driven to relatively low frequencies, and in small populations, genes at low frequencies tend to be lost.

Second, domestic animals can tolerate more random phenotypic change than can stocks destined for reintroduction into unmanaged habitats. Breeders of domestic animals may safely ignore genetic and phenotypic changes in their organisms that would completely debilitate a species that had to cope with predators, inclement weather and other conditions occurring in nature. Egg ranchers for example, do not mind if their animals have genes that cause a poor body configuration, poor feather distribution, myopia, or poor predator escape behaviour. These sorts of genes often accumulate in a stock during the course of inbreeding and artificial selection.

We refer to the 1% rule as the *basic rule of conservation genetics* because it serves as the basis for calculating the irreducible minimum population size consistent with short-term preservation of fitness. An even more stringent criterion for conservation genetics is given in Chapter 4 in the context of long-term protection of adaptive potential.

How does the basic rule translate into population size? Fortunately, the

relationship between ΔF and N_e is a simple one. The per generation rate of loss of heterozygosity is related to population size as given by Wright's (1931) expression

$$\Delta F = \frac{1}{2N_e} = \frac{1}{8N_m} + \frac{1}{8N_f} \qquad (3.7)$$

so that the effective size must be fifty or more if the loss of heterozygosity (or increase in homozygosity) is not to surpass 1%.

As discussed in section 3.1, a census size = 50 individuals does not always mean an effective size of fifty. To take only one example, the sexes of the reproducing adults, if not equal, will require a larger census if our goal is $N_e \geqslant 50$. This is illustrated in Fig. 3.15. Note that the *minimum* number for the least numerous sex is fifteen. In other words, if a herd has less than fifteen breeding males (and between seventy and eighty females), ΔF will be above 1%, which is in the danger zone according to the rule. If a slightly more

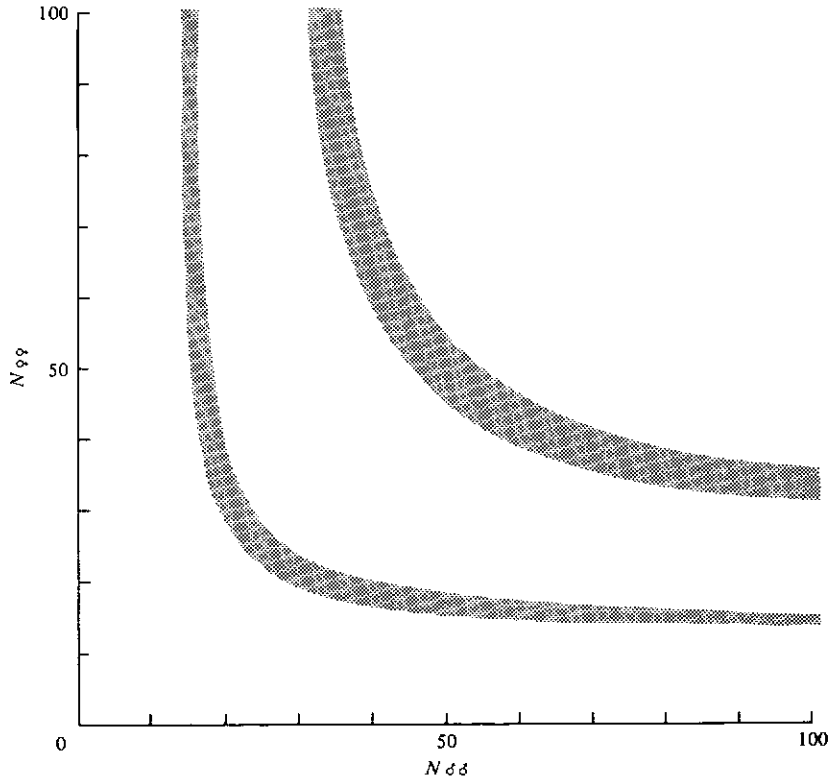

Fig. 3.15 The numbers of breeding males and breeding females needed to satisfy the 1% or $N_e = 50$ criterion (the lower left shaded region) and the 0.5% or $N_e = 100$ criterion (the upper right shaded region).

conservative criterion is employed, say ΔF of 0.5%, the equivalent minimum numbers are thirty males and ninety females. Similar effects result from population fluctuations and progeny distributions when $V_k > \bar{k}$. In the following chapters we shall apply the basic rule to various hypothetical and real conservation problems.

3.5 Mutation–selection equilibria

One final point. We have chosen to discuss the mutational origin and replacement of variation in the following chapter, so only a brief comment is made here. One might hope that the alleles that are lost in small populations could be regenerated by mutation. Such a sanguine expectation is untenable. Wright (1937) discussed this problem while examining the effects of joint pressures on the distribution of gene frequencies at single loci. The essential conclusion is that selection pressure is relatively powerless to prevent loss or fixation of alternative alleles (including new mutations) in a small population. Not only will small populations rapidly lose genetic variation, but *the beneficial alleles have roughly the same probability of being fixed as do the deleterious ones.*

3.6 Summary

1. A population bottleneck, here defined as a single generation event during which a population is severely reduced in size, has the following effects on genetic variability:
   (a) The loss of genetic variation or heterozygosity is not severe; even two individuals retain 75% of a population's genetic variance;
   (b) In contrast, the loss of alleles, especially those at low frequencies, is serious. If these alleles are ever important for survival, such as during an epidemic, then their attrition would increase the expectation of extinction;
   (c) The total amount of genetic variation that is lost following a bottleneck depends on how fast the population grows to a moderate (several hundred or more) size.
2. Chronically small population size produces random gene frequency changes and fixation or loss of alleles; such random fluctuations are referred to as genetic drift. Small populations continually 'leak' alleles and genetic variance, and a few generations of genetic drift is much more erosive of genetic variation than a bottleneck followed by a rapid recovery of numbers.
3. The impact of genetic drift is directly related to the effective population size, not the census number of individuals. The effective size, N_e, is extremely sensitive to unbalanced sex ratio among breeding adults. In

a polygynous system in which the females are monogamous (e.g. Pere David's deer, hamadryas baboon, elephant seal, zebra) N_e can be an order of magnitude smaller than the census number.

4. Large fluctuations in population size can severely depress N_e. The reason is that N_e is most strongly influenced by sampling error (drift) occurring during the 'crash' part of the sequence of generations. Mathematically, N_e is the harmonic mean, not the arithmetic mean, of the numbers in each generation.
5. If for any reason the reproductive output of some families is especially great, and/or that of other families is especially poor, N_e will be less than it would be if the number of progeny were randomly distributed among families. The converse is also true: as the variance of progeny per family approaches zero, N_e approaches twice the actual number of breeding adults.
6. It is incumbent on managers to be aware of these effects and to maximize N_e to the extent consistent with other management programme criteria.
7. The central problem of conservation genetics is the relationship between change in genetic variation and fitness. In natural populations, genetic variation can vary in space (between populations) as well as time (within populations). A review of the literature leads us to conclude that fitness within and between *natural* populations is often correlated with measures of heterozygosity. The superior viability of relatively heterozygous individuals is most apparent when sampling different age classes, especially in species with 'structured' populations. There is also some evidence that homozygous individuals are phenotypically more extreme compared to heterozygotes. Among populations of the same species, at least for non-flying vertebrates, there is evidence for a correlation between mean heterozygosity and fitness.
8. Predominantly inbreeding plants span virtually the entire range of heterozygosity levels observed in outcrossing species, though a relatively high proportion of them have no detectable allelic variation. In those populations in which heterozygosity is retained there is some prima-facie evidence for heterozygote superiority.
9. The existence of some populations of plants and animals lacking detectable genetic variation means that survival is possible without heterozygosity, at least under some circumstances. Since such populations cannot evolve, however, their extinction probability is very high.
10. There is some evidence for the asymptotic model of genetic variation and fitness, suggesting that the more genetic variation is lost, the more deleterious the losses become.
11. Inbreeding always reduces fitness in animals; the decline for reproductive traits (inbreeding depression) resulting from a 10% increase in the inbreeding coefficient is usually between 5% and 10%. For total reproductive performance, the decline may be two to five times this high. Behaviour traits and competitive ability are also depressed by inbreeding.
12. Intense inbreeding results in the loss of 90% or more of lines unless the stock has a long history of domestication or slow inbreeding.
13. The basic rule of conservation genetics, based on the experience of animal breeders, is that the maximum tolerable rate of inbreeding is 1%. This translates into an effective population size of fifty.