Lecture 12. Natural Selection and Population Regulation.

Reading:

Shields, W. M. Chapter 8. Optimal inbreeding and the evolution of philopatry. Pages 132-159 *in* I. R. Swingland and P. J. Greenwood, eds. The ecology of animal movement. Clarendon Press, Oxford, United Kingdom.

Meffe, G. K. 1986. Conservation genetics and the management of endangered fishes. Fisheries 11(1):14-23.

Optional:

Hedrick, P. W. and P. S. Miller. 1992. Conservation genetics: techniques and fundamentals. Ecological Applications 2:30-46.

Lande, R. and G. Barrowclough. 1987. Effective population size, genetic variation, and their use in population management. Pages 87-123 *in* M. E. Soulé, ed. Viable populations for conservation. Cambridge University Press, New York, New York, USA.

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.

Genetic models are much like the predator-prey and competition models we have been studying -- they appear to be very naive, and we don't have the hard data to support their predictions. Most of the following material on methods and models comes from Meffee (1986).

Measuring genotypes in populations -- Different classes of genes

blood group antigens - proteins

restriction sites on

mitochondrial DNA, mtDNA

nuclear DNA

Electrophoresis -- measures differences in proteins that reflects genotype

Loci -- particular protein being measured

Allele -- specific type(s) of protein found at the loci, usually denoted as A and a

Each loci in an individual has 2 alleles, one from father, and one from mother. These 2 alleles (proteins) can be combined in an individual as:

AA

Aa

aa

However, a loci across the population may have more than just 2 alleles, e.g., *A*, *B*, and *C*. Then, individuals in the population may have any of the following combinations: *AA*, *AB*, *AC*, *BB*, *BC*, and *CC*.

Heterozygosity is defined as the variation in a loci, i.e., frequency of *Aa* allele pair for alleles *A* and *a*.

Suppose Probability of *A* is $\Pr(A) = p = 0.3$. Then,

$\Pr(AA) = \Pr(A)^2 = 0.3^2 = 0.09$ Homozygous
$\Pr(aa) = (1-\Pr(a))^2 = 0.7^2 = 0.49$ Homozygous
$\Pr(Aa) = \Pr(A)(1-\Pr(a))$ X 2 Heterozygous
$= 0.7$ X 0.3 X $2 = 0.42$
Total $= 1.0$

Hardy-Weinberg law implies these probabilities, i.e., random mating in a panmictic (mixed) population.

Heterozygosity of loci *j* in a population is measured as

$$\hat{h}_j = \frac{2n_j(1 - \sum x_{ij}^2)}{(2n_j - 1)}$$

where n_j is number of animals measured at loci *j*, so $2n_j$ is the number of alleles measured, and x_{ij} is frequency of allele *I* at loci *j*. The term $2n_j/(2n_j - 1)$ is a small sample bias correction.

For $\Pr(A) = 0.3$, $x_{1j} = 0.3$ and $x_{2j} = 0.7$, so $h_j = 0.42$, which is the probability of the loci containing 2 unlike alleles at loci *j*. Homozygosity is the probability of 2 like alleles at loci *j*, and thus equals $1 - h_j$.

Random mating means each animal has equal probability of breeding with any other animal in the local population, known as a deme. A deme is the unit within a population where mating is random. Two segments (e.g., north and south) of a population might each comprise a deme.

At time 0, $\Pr(A) = p = 0.3$. The number of *A*'s in the population is $2np$, i.e., 2 alleles in each member of the population. What happens after one generation in the population to the gene frequency of *A*?

Possible matings, their probability of occurring, and the probabilities of the offspring genetic frequencies are shown in the following table for $\Pr(A) = p = 0.3$ and $\Pr(a) = 1 - p = 0.7$.

Parents' Alleles	Probability of Parents Mating	Offspring's Alleles	Offspring Probability	Combined Probability
aa X *aa*	0.49^2	*aa*	1	0.49^2
aa X *aA*	0.49 X 0.42 X 2	*aa*	0.5	0.49 X 0.42
		aA	0.5	0.49 X 0.42
aa X *AA*	0.49 X 0.09 X 2	*aA*	1	0.49 X 0.09 X 2
aA X *aA*	0.42^2	*aa*	0.25	0.25 X 0.42^2
		aA	0.5	0.5 X 0.42^2
		AA	0.25	0.25 X 0.42^2
aA X *AA*	0.42 X 0.09 X 2	*aA*	0.5	0.42 X 0.09
		AA	0.5	0.42 X 0.09
AA X *AA*	0.09^2	*AA*	1	0.09^2
Totals	1.00			1.00

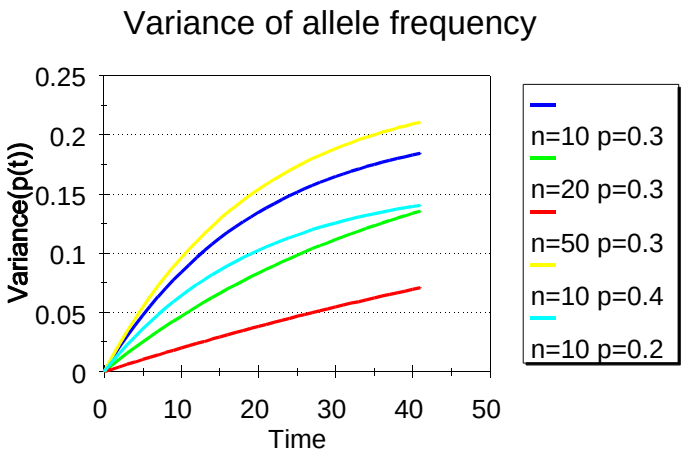
At time 1, $E(\#A) = 2np$, and $Var(\#A) = 2np(1 - p)$. The actual variation in p_1 is $Var(p_1) = Var(\#A/2n) = (1/(2n))^2 Var(\#A) = p(1 - p)/(2n)$

At time 2, $Var(p_2) = p(1 - p)(1 - (1 - 1/(2n))^2)$

In general, at time t,

$$Var(p_t) = Var(p_{t-1})\left(1 - \frac{1}{2n}\right) + \frac{p(1 - p)}{2n} .$$

Increasing n decreases the rate of increase in variance and increasing p up to 0.5 increases rate of increase of variance.



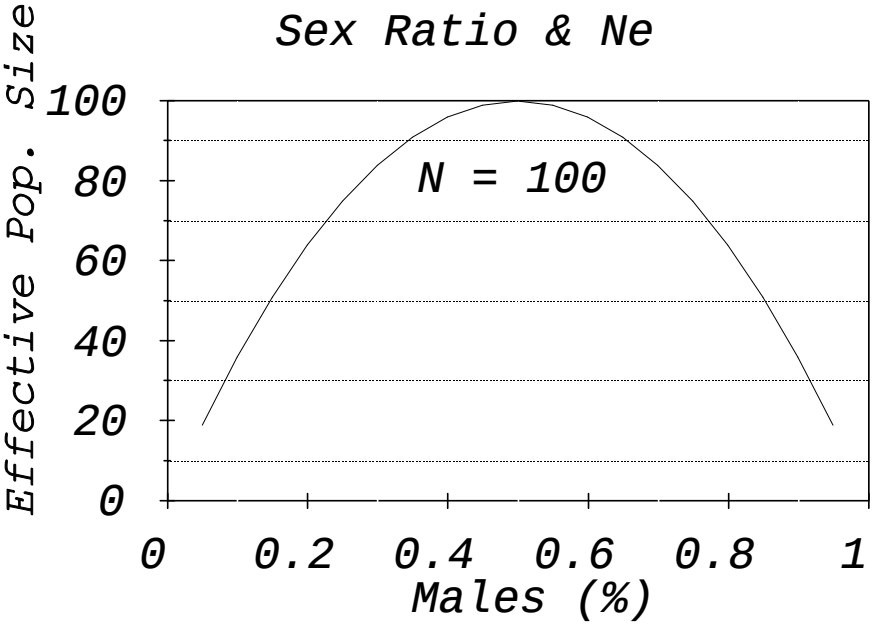
Conservation implications of this graph are important. The first implication is that the smaller the population size, the more chance of losing an allele, and hence genetic variation, or heterozygosity. Second is that the probability of losing an allele increases as allele frequency goes down. This random variation is call *random drift*.

In populations, we are concerned about the overall level of heterozygosity in the population across all loci. For L loci,

$$H = \frac{\sum_{j=1}^{L} h_j}{L}$$

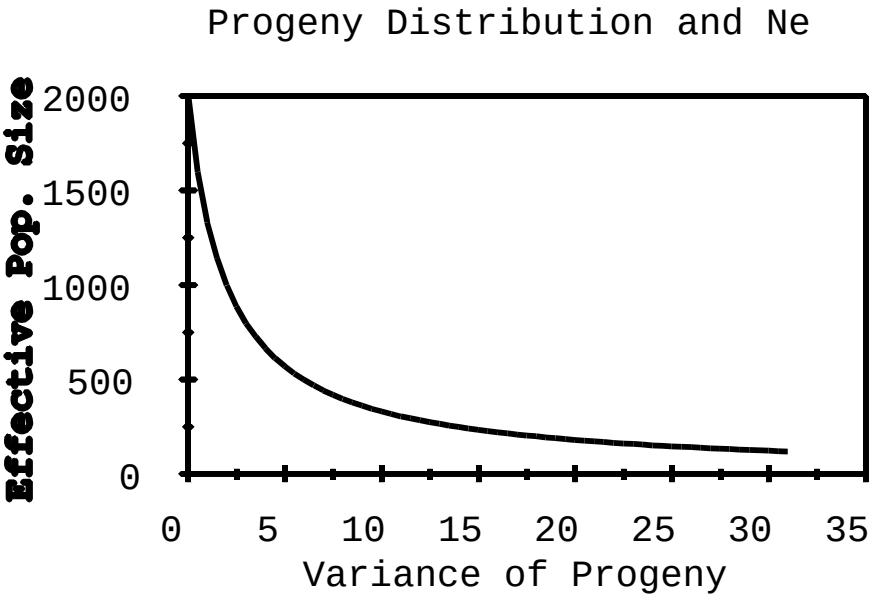
Effective population size, N_e, assumes random mating. For most of the populations we are interested in, we do not see random mating because the population consists of males and females. The effective size of the population is a function of the number of males and females.

1. Sex ratios, $N_e = (4\ N_m\ N_f)/(N_m + N_f)$



As sex ratio is biased away from 0.5, the effective population size declines drastically. This result is because only a small portion of the population is responsible for ½ of the alleles in the next generation. This has implications for male only hunting seasons.

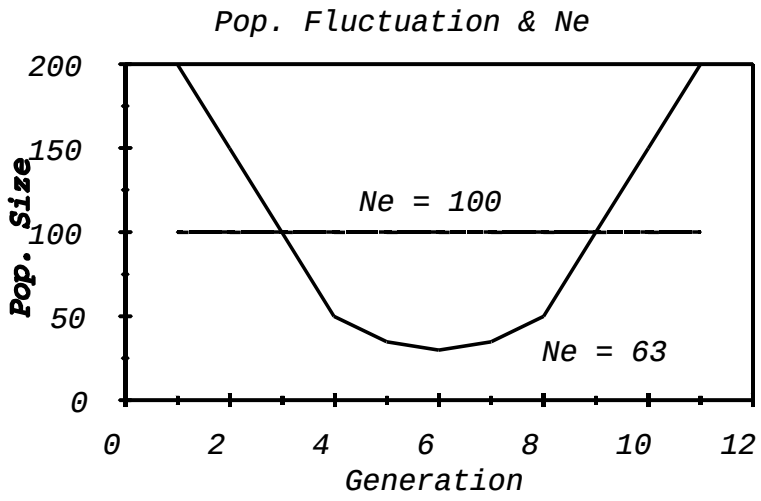
2. Distribution of progeny, $N_e = 4\,N/(2 + \sigma^2)$, where σ^2 is the variance of the progeny distribution in the population. Suppose that 1000 males mate with 1000 females and each female has on average 2 offspring that contribute to the next generation. If the distribution of offspring is Poisson distributed, then the mean equals the variance, so $\sigma^2 = 2$, and $N_e = 4$ X 2000/(2 + 2) = 2000. In contrast, if 999 females each have 1 offspring and 1 has 1001, then $\sigma^2 = 31.6$ and $N_e = 4$ X 2000/(2 + 31.6) = 238. An example of this phenomena is the reintroduction of peregrine falcons on the east coast of the US (Temple Pers. Commun.). 95% of the present birds are descendants of 5 individuals, of which 2 pairs are siblings, all from Alaska. Even though 16 subspecies were used for the introduction, including individuals from Europe, the Alaska birds provided the starting F_1 generation.



Nunney and Elam (1992) suggest underestimates of effective population size are because of a failure to account for a long maturation time, and problems with the correction for overlapping generations.

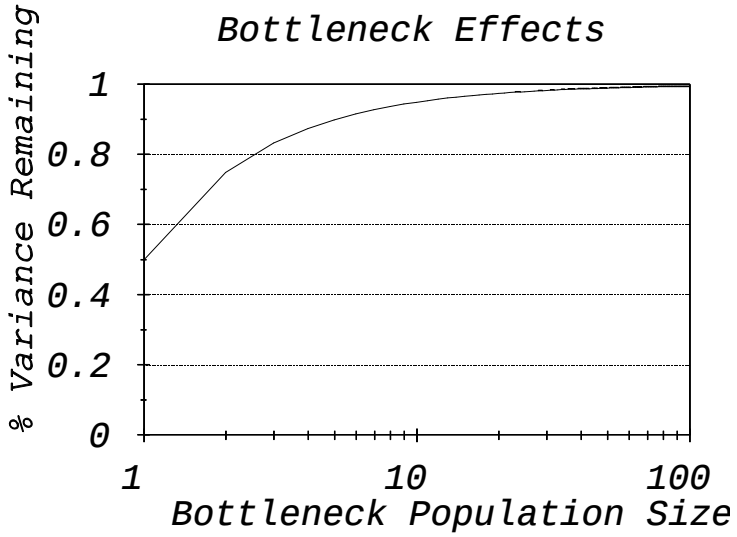
3. Population Fluctuations. When populations fluctuate through time, all the genetic variation for all future populations is contained in only a few survivors (assuming the mutation rate is zero). The harmonic mean population size represents the effect population size for the population,

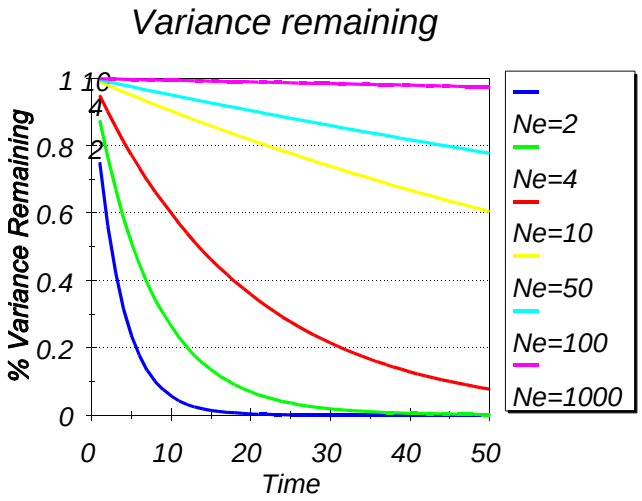
$$\frac{1}{N_e} = \frac{1}{t}\left(\frac{1}{N_1} + \frac{1}{N_2} + \cdots + \frac{1}{N_t}\right)$$



The phenomena of a population dropping to a low level and then recovering is called a *population bottleneck*. When populations go through a bottleneck, the amount of genetic variation left after the bottleneck depends on 1) the size of the population in the bottleneck, and 2) the length of time (number of generations) the population was in the bottleneck. The percent of genetic variance remaining in the population after *t* generations is

$$\left(1 - \frac{1}{2N}\right)^t$$





These graphs would suggest that severe bottlenecks are a complete disaster for genetic variation in the population. We have to examine the 2 major assumptions we are making to arrive at this conclusion.

Bottlenecks do lower genetic variation, or could the model be incorrect. Bryant et al. (1986) measured the effects of passing houseflies through population bottlenecks (Lewin 1987). Genetic effects that they saw in populations of flys that bred from 1, 4, and 16 male-female pairs in 3 separate experiments was an increase in variance, not a decrease as most mathematical models of bottlenecks would imply. There was more variation in the flys' physical characteristics (wing size and shape, 8 traits) in post-bottleneck populations than pre-bottleneck populations. They measured 8 traits on 3000 flies. From the formula for the percent of genetic variance retained, we calculate that:

1 pr. -- expect 75% of original variation
2 pr. -- expect 94% of original variation
16pr. -- expect 98% of original variation

Most variation came through the bottlenecks of intermediate sizes, i.e., 4 and 16 pairs. "The dogma of bottleneck theory has always assumed that the newly founded population is somehow at risk, because of predicted lower variance." Bryant.

Another explanation of why the observed reduction in variance is not as great as predicted by the above equation concerns frequency of alleles in the original population. Suppose 200 alleles exist at a loci in the original population. If only a single pair comes through a bottleneck, at most 4 of these 200 alleles will exist. However, in the original population, the 200 alleles are not equally frequent, and hence do not contribute equally to the next generation. Thus, the reduction from 200 to 4 is a major reduction, but not as much as it first appears.

Example of this dogma are cheetahs (O'Brien et al. 1985, 1987, Lewin 1987, Caro and Laurenson 1994). Cheetahs prior to 10,000 years ago were widely distributed. Currently the entire population is more genetically uniform than inbred laboratory mice. These animals are so genetically similar that skin graphs from one individual can be given to a completely unrelated individual. Breeding success in captivity is poor, with very low quality of the male's spermatozoa and high infant mortality. The usual explanation of these observations is a long and persistent bottleneck. Similar characteristics are observed in the Florida panther.

Caro and Laurenson (1994) suggest genetics are not the problem. They found that most cub mortality was from predation, mainly hyeanas. Reproduction in the wild was high compared to studies in captivity. Reproductive data from captivity generally is highly variable, with some zoos having good success. This result suggests that breeding in captivity is affected by animal husbandry, with inappropriate social conditions causing the low reproductive success, and not genetics.

However, Jiménez et al. (1994) experimentally demonstrate that inbreeding reduces survival, and hence fitness. But is the treatment Jiménez et al. applied a realistic manipulation, i.e., severe inbreeding is known to be deleterious, but does that imply that lower heterozygosity is necessarily as deleterious?

The second assumption we must question is the evidence for heterozygosity increasing fitness. Genetic variation (heterozygosity) varies greatly from taxon to taxon, and its evolutionary meaning is controversial. What are the *costs* and *benefits* to maintaining heterozygosity?

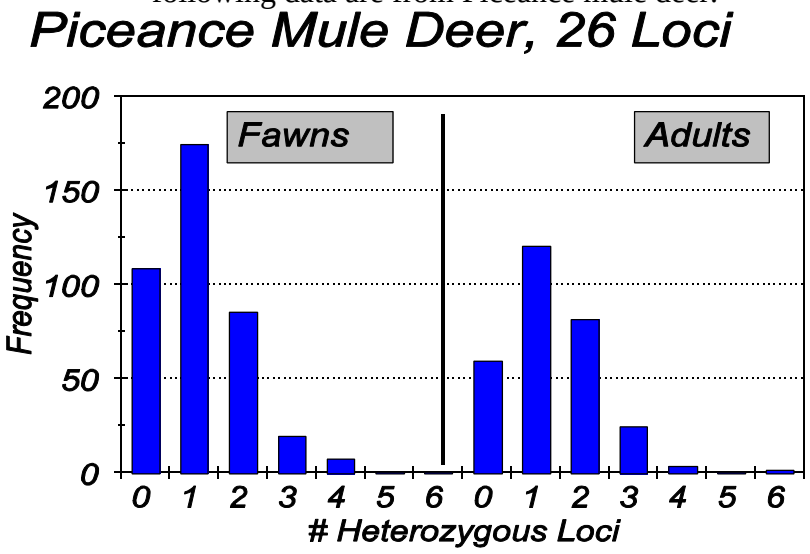
The loss of some alleles may be very beneficial, i.e., if the allele is detrimental to survival under certain conditions. Localized selection means that alleles beneficial to local situations are desirable and needed. Other alleles may be neutral or negative, so their loss may be neutral, or even positive. High heterozygosity may be detrimental to local conditions (Shields 1983). Being a jack of all trades means you are a master of none. Too much heterozygosity results in *outbreeding depression*. The opposite phenomena is *hybrid vigor*. Rhymer and Simberloff (1996) discuss the implications of increasing heterozygosity through hybridization. Either outbreeding depression or hybrid vigor may result. However, the “pure” genetics of both populations may be lost. Even if the genetics of the populations are not changed, the population dynamics may be changed because the production of no offspring, or infertile offspring, lower the recruitment rate of one or both populations. This article provides a great deal of practical advice on hybridization and *introgression* (gene flow between populations whose individuals hybridize, achieved when hybrids

backcross to one or both parental populations).

Inbreeding is often cited as evidence of the importance of heterozygosity, i.e., *inbreeding depression* is bad so outbreeding must be good. However, inbreeding really supports the case that too much heterozygosity can be bad. Inbreeding effects are often the result of 2 deleterious recessive alleles coming together. Inbreeding can be used to purge a population of these deleterious alleles. Breeding programs for domestic livestock and dogs are examples. You save the good ones, and throw the rest away. The good ones are really good, and the bad ones are really bad!

Evidence for localized selection, or at least localized differences in genotypes. Changes in allele frequency can occur over very short distances in populations. We observed differences in allele frequencies between trap sites at Little Hills, in distances <5 km. Populations are genetically structured, with genetically diverse units (Rhodes and Smith 1992). Hence, genetic variation may appear to be high across the population, but may be lower within the individuals that make up the population. This argument suggests the random mating assumptions is not realistic.

Evidence that heterozygosity improves fitness is strictly correlative. See Table 1 from Rhodes and Smith (1992). Multiple correlations are run against H, and some are bound to be found significant. Second, these correlations are looking at the results of selection, not the process itself. Frequencies of heterozygous loci suggest that too much heterozygosity is bad. The following data are from Piceance mule deer.



The frequencies represent what is left, and the possibility that these frequencies are correlated with fitness cannot be dismissed. If linear correlations are conducted against fitness, with increasing fitness up to the mode plus 1, and sharply decreasing fitness for >H, a positive correlation

of H and fitness would result. However, this correlation is spurious because we have not included the 0 fitness animals no longer present in the population. The problem with taking the sample from the population is that the low heterozygosity animals are over-represented, and hence a linear trend appears to be supported. If equal sample sizes of each level of heterozygosity were used, a quadratic relationship would likely be observed, because there is some optimum level of heterozygosity.

Implications of genetics for metapopulations. Patch extinctions mean you loose genetic diversity from the patch. Re-colonizers will effectively be coming through a bottleneck, so that patch genetic diversity is now low. As this process continues through time, the genetic diversity of the entire population becomes very low (Gilpin 1991, McCauley 1991). Hence, if the metapopulation model is valid, this loss of genetic variation cannot take place! Either the metapopulation model is incorrect, or bottleneck theory is wrong, or heterozygosity is not all that important. An example of such a population would be annual weeds. Of courss, you could argue that annual weeds are really one big population, and not a metapopulation. (The population in my garden hasn't gone extinct recently!). Implications of this idea are important to the SLOSS debate. A single large reserve would have a larger effective population size, whereas several small reserves has a lower effective population size because of the lack of a large panmictic population with random breeding.

Importance of intraspecific variation is discussed by Behnke (1992). For many trout populations, the genetic analyses suggest they are the same genetically. Yet, they breed at different times of the year, and have other important behavioral characteristics that allow their survival in unique environments. Retention of the intraspecific genetic variation is necessary to maintain biodiversity. Note that not all phenotypic characteristics are genetically based. Kroodsma and Canaby (1985) found that marsh wren (*Cistothorus palustris*) song repertoire and style of delivery was genetically controlled. In contrast, James (1983) showed that a significant proportion of red-winged black bird (*Agelaius phoeniceus*) nestling development characteristics were not genetically based. In both of these studies, birds were moved as eggs to different environments to test the hypothesis of genetic-based versus environment-based origin of the traits studied.

Conclusions about the importance of genetics in populations. Genetic models need a great deal more testing. Experiments are needed to test if heterozygosity is strongly related to fitness, i.e., cause-and-effect experiments. M. Smith (Pers. Comm.) conducted experiments with mosquito fish in wading pools. Founding pairs were selected to give low or high heterozygosity, with population persistence expected to be the greatest for high heterozygosity pools. Have not heard the results.

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