LECTURE 13

A. Genetic Drift

1. Random fluctuations in gene frequencies: best considered as small population effects arising from non-random (chance) reproduction of genotypes.

2. Main (genetic) effects of genetic drift are:

   a) Changes in gene (allele) and genotype frequencies. Under many situations where genetic drift is occurring, there also will be an increase in homozygosity and a concomitant decrease in heterozygosity. This actually is *inbreeding*, not *drift*, but is a consequence of small *effective* population size.

   b) One can explore the possibilities by considering a single locus with two alleles and intermediate gene frequency, i.e., $p = 0.5$, $q = 0.5$, and starting with a small population.

      (i) Note the tendency to go to fixation and the associated increase in homozygosity

      (ii) Note also the difference in potential for "random" allele-frequency change in a large versus a small population, e.g., one with 10^6 individuals versus one with 10 individuals

3. Under genetic drift (random chance events), one can predict the probable *magnitude* of gene frequency change but not the *direction.* The *magnitude* of gene frequency change will be an inverse function of the effective population size (N_e). Because the effect is random, one cannot predict the direction of gene frequency change.

4. Consider the difference between N_e (the effective population size) and N (the census size).

   a) N_e is the size of the ideal population that will result in the same amount of genetic drift as the actual population being considered

   b) Consider species that are harem brooders, where a single, alpha male can inseminate 10-15 females

5. Notable examples of genetic drift include founder effects and bottleneck effects:

   Founder effects: ABO blood groups frequencies in Dunkers and the US Caucasian population

Blood group	USA	Dunkers (Europe)	Dunkers (USA)
AB	.04	.05	.02
A	.40	.44	.60*
B	.11	.10	.03*
O	.45	.41	.35

Bottleneck effects: Flush/crash cycles (essentially same as founder effects = genetic drift)

6. Because the "key" parameter underlying genetic drift is N_e, one would predict that any biological or other parameter leading to small N_e should promote genetic drift and potentially lead to rapid genetic and evolutionary change

    a) There are numerous examples indicating this is the case: one good example is chromosomal and genetic change in populations of wild horses

7. Hypothesis of "mutational meltdown"

8. A major (historical) question in population genetics regards the following:

    Surveys of genetic variations (electrophoresis of proteins, direct DNA sequences, etc.) demonstrate amply that fairly extensive heterozygosity is maintained in natural populations, even in situations where genetic drift should be fairly intense. How can this be?

    a) One possibility is overdominant selection. This is possible but one must deal effectively with the segregational load "problem" noted previously. Another possibility is frequency-dependent selection, although this is somewhat difficult to demonstrate unequivocally in natural populations.

    b) A third possibility is…

B. <u>Migration</u>

1. Basic definition is movement of genes from one population to another that results in a change in gene frequency

    a) Note the "genetic migration" implies movement of genes from one population to another, and that this may or may not be reflected in physical movement of individuals

2. Main genetic effects of migration are twofold:

    a) Maintenance of genetic variability (heterozygosity

    b) Homogenization of diverging subpopulations, i.e., retards genetic divergence

3. Migration is very difficult to assess in natural populations but theory predicts that very few migrants are needed to maintain similar allele distributions in natural populations at least for nearly-neutral alleles

Quantitative Genetics [Multiple Gene Inheritance)

A. History begins with studies of the genetics of plant size in the tobacco plant, *Nicotiana tabacum*

1. Investigators carried out a standard Mendelian approach

P_1	tall x dwarf	(just like Mendel)
F_1	intermediate	(not like Mendel)
F_2	continuous gradation	(also not like Mendel)

2. Results differed from those of Mendel in the garden pea (*Pisum*) where a 3:1 (tall:dwarf) ratio was found in the F_2, and results were attributed to a single locus with two alleles and simple dominance

3. It should be noted that some (but not much) variation around the two phenotypic classes was found in Mendel's experiments; overall, however, there was a clear discontinuity between the phenotypic classes – this was *not* the case in the studies in tobacco

B. The difficulty was how to explain the differences between…

1. *Continuous variation*: continuous gradation, with no distinct phenotypic classes, and

2. *Discontinuous variation*: distinct phenotypic classes

a) It seemed obvious given what was known about "particulate inheritance" that the observation of continuous variation was incompatible with Mendelian laws; thus, during the early 1900s, most geneticists believed there were two kinds of genes.

(i) those that showed discontinuous variation: Mendelian traits
(ii) those that showed continuous variation: quantitative traits

b) A few individuals, however, proposed that both types of traits (Mendelian and quantitative) shared a common Mendelian basis, but that quantitative traits were caused by Mendelian genes whose effects on phenotype were small and cumulative (additive), and where there was no dominance between alleles at genes and no epistasis among genes

c) This is the multiple gene (*polygene*) hypothesis and is commonly observed for most metric traits, i.e., traits (characters) that are measured and generally distributed "normally" in natural populations