

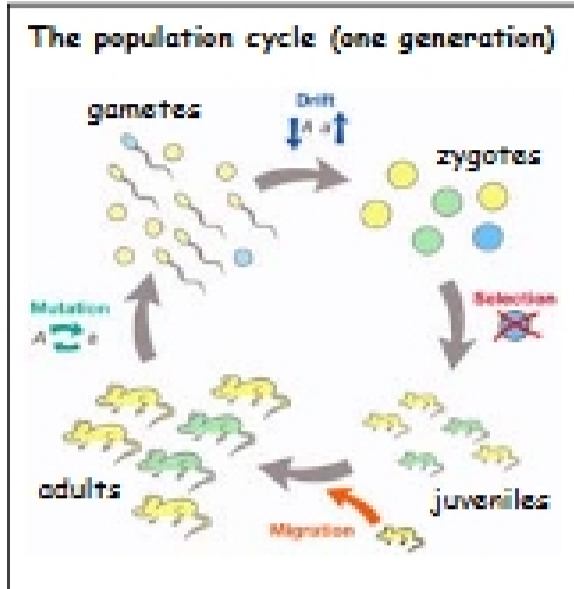
Evolution is *allele-frequency change in populations ... and four basic processes or "forces" govern such change.*

**Mutation**  
(creates new, untested alleles from random damages and errors of DNA replication)

**Drift**  
(randomly samples alleles already present in the population's "gene pool")

**Selection**  
(tests those alleles for their relative abilities to survive and reproduce under current environmental conditions)

**Migration**  
(introduces old, pre-tested alleles from other populations or closely related species)



Sci 2410, 2 February 2009

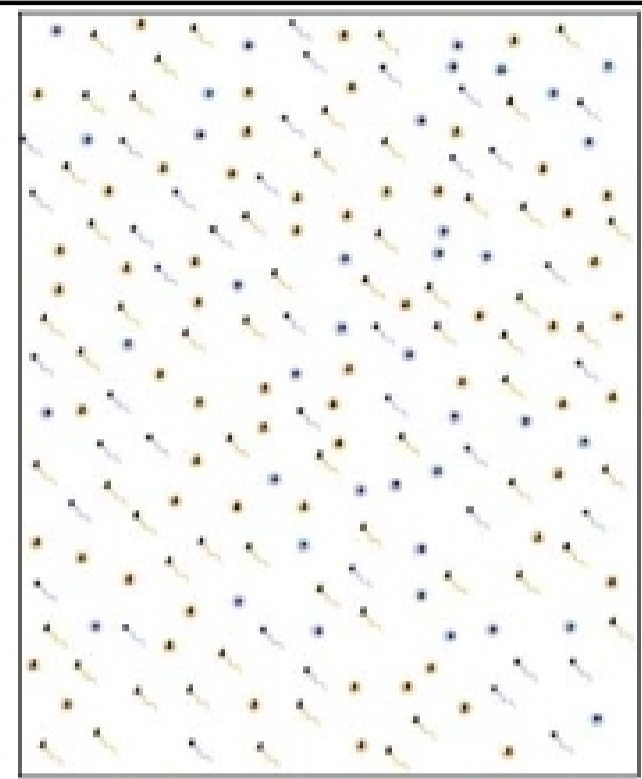
A gene pool with allele frequencies (proportions)  $p=0.6$  (A) and  $q=0.4$  (a)

100 eggs in all  
60 are A  
40 are a  
 $p(A) = 60/100 = 0.6$   
 $q(a) = 40/100 = 0.4 = 1 - p(A)$

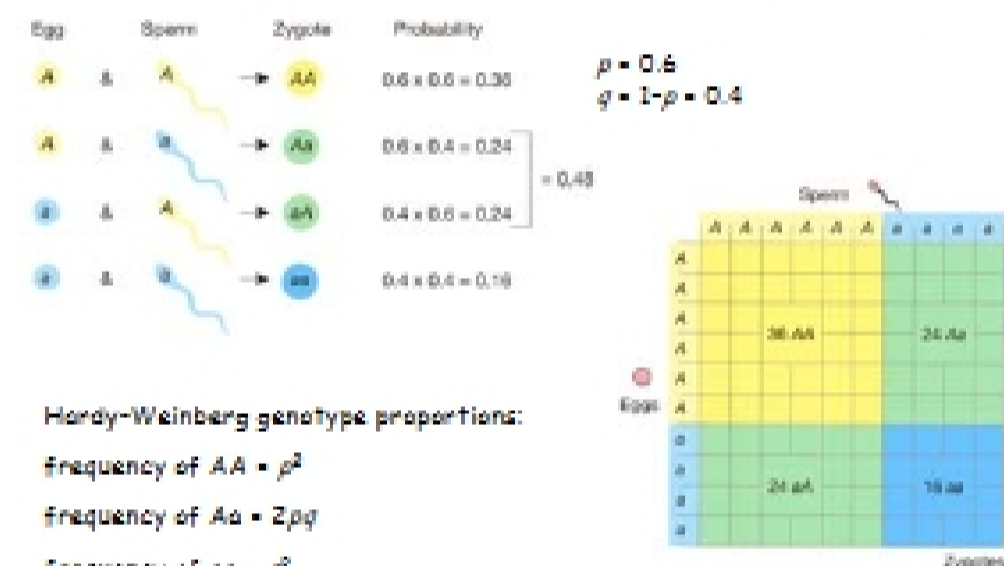
100 sperm in all  
60 are A  
40 are a  
 $p(A) = 60/100 = 0.6$   
 $q(a) = 40/100 = 0.4 = 1 - p(A)$

If we reach in and take an egg or sperm at random:  
What's the probability it carries A? a?

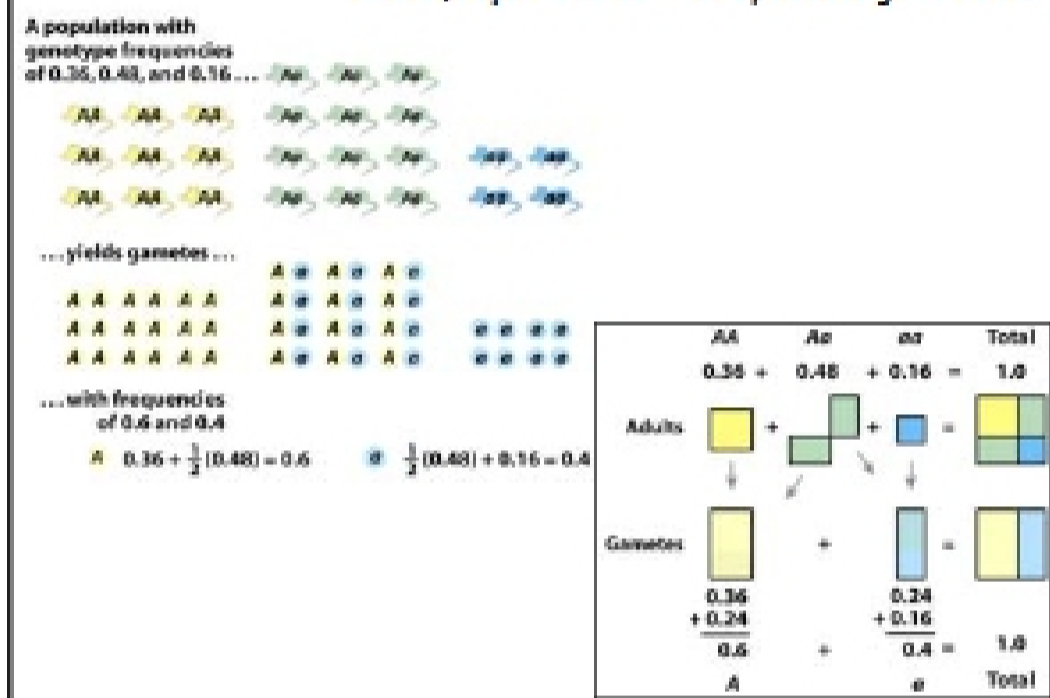
What happens when we combine (pair) these eggs and sperms at random?



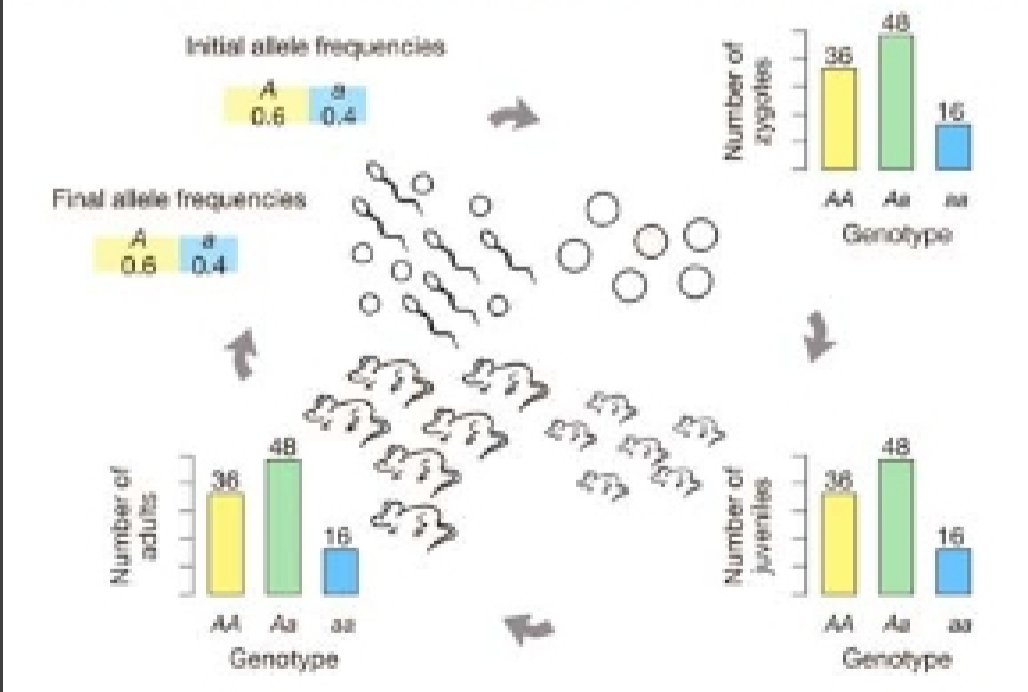
"Random" mating causes *predictable* unions of eggs and sperm.



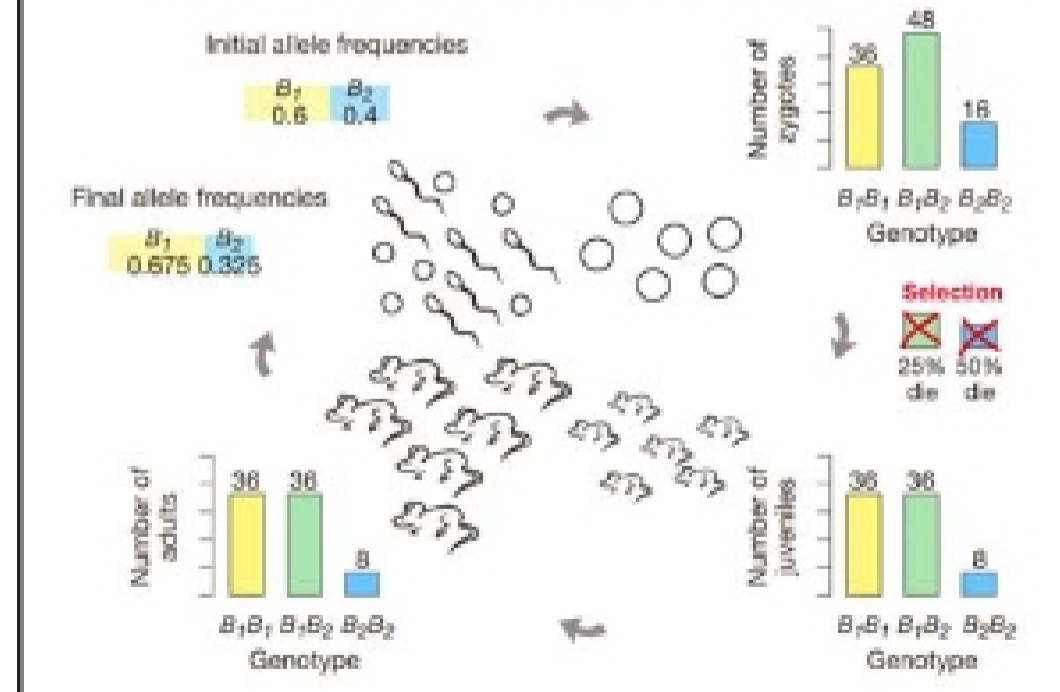
Next generation, gametogenesis makes a gene pool with the *same* allele frequencies as in the previous generation!



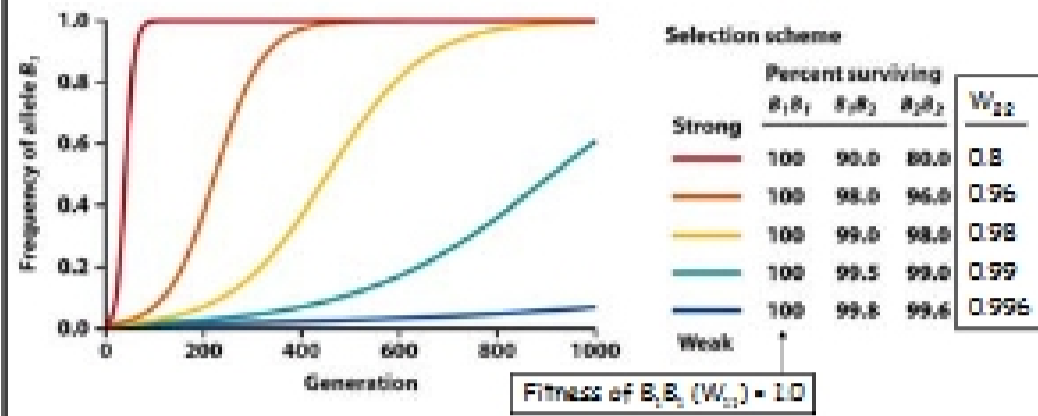
Thus dominance, recessiveness, random mating and reproduction cause *no evolution* in the absence of mutation, drift, selection and migration.



Selection changes allele frequencies in predictable directions by systematically affecting survival and/or reproduction.



**And small fitness differences can have large effects.**



Selection is closely related to population growth.  
 One "population" of alleles increases at a faster rate than the other.  
 The principle of "compound interest" applies, and this is why the increase of a rare allele looks exponential (blue curves, above).

**The changes can easily be modeled quantitatively.**

Let  $p$  be the frequency of allele  $A_1$  in the *present* generation [ $q = 1 - p = f(A_2)$ ].  
 Let  $p'$  be its frequency *next* generation.

Let  $W_1$  be the average allelic or "marginal" fitness of  $A_1$  alleles (that is, the fitnesses of  $A_1A_1$  homozygotes and  $A_1A_2$  heterozygotes, weighted by the total numbers of  $A_1$  alleles that they carry).

Similarly, let  $W_2$  be the average (marginal) fitness of  $A_2$  alleles.

Then the mean fitness of *all* alleles at the A locus is  $W = pW_1 + qW_2$ . (This is just the average fitness of all *individuals* in the population.)

From these definitions and just a little algebra (as shown in the handout), it follows that:

$$p' = p[W_1 / W]$$

I.e., the frequency of  $A_1$  *next* generation is just its frequency *this* generation, times the ratio of its fitness to that of all the alleles at the A locus.

This is equivalent to Freeman and Herron's derivation in Box 6.3 (page 186), but probably easier to understand.

**An important special case: lethal recessive alleles**

$$W(A_1A_1) = W(A_1A_2) = 1$$

$$W(A_2A_2) = 0$$

$$W_1 = 1$$

$$W_2 = [(1) \frac{1}{2} 2pq + (0)q^2] / [pq + q^2] = p$$

$$W = p(1) + q(p) = p(1+q) = p(2-p)$$

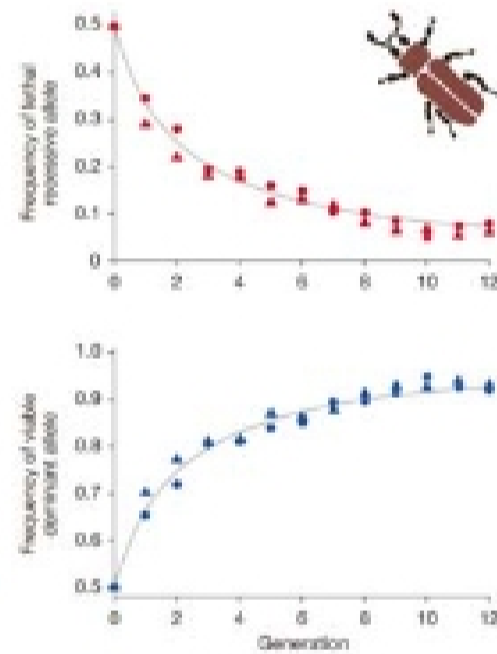
$$p' = p[W_1 / W] = p[1 / p(2-p)] = 1 / (2-p)$$

E.g., suppose  $p = 0.5$

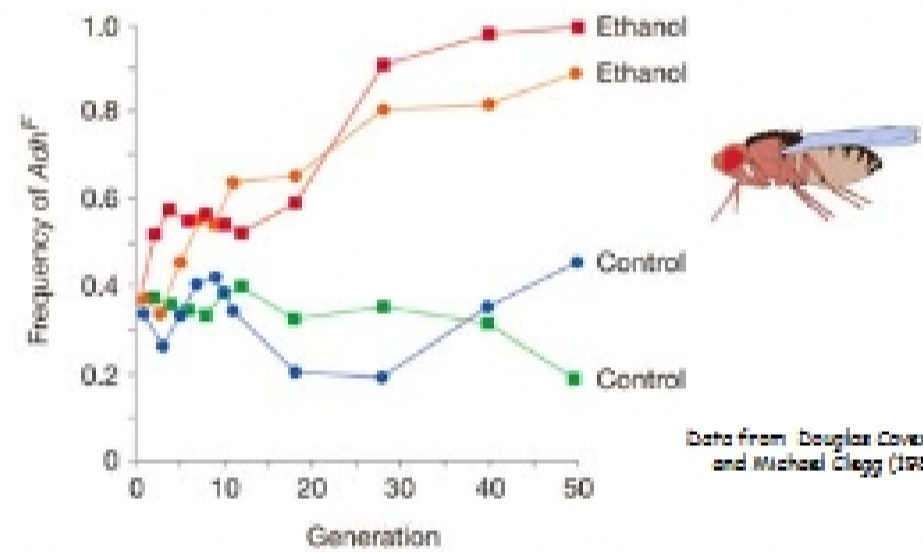
$$\text{Then } p' = 1 / (2 - 0.5) = 1 / 1.5 = 0.67$$

Peter Dawson's (1970) data from an experiment with flour beetles is shown on the right (red and blue symbols), with the theoretical prediction graphed as continuous gray lines. Amazing!

(See Freeman & Herron, pp. 195-197.)



**$Adh^F$  beats  $Adh^S$  on laboratory fly food soaked in EtOH**



Homework problem: Let FF homozygotes have a fitness of 1.0 ( $W_{FF} = 1$ ), and assume F/S heterozygotes have *intermediate* fitness. Very roughly, what is the fitness of the SS homozygotes, in the ethanol-soaked environment?