Topic 09
Evolution

I. Populations

A. Evolution is change over time

(change in the frequency of heritable phenotypes & the alleles that govern them)

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B. Populations are the units of evolution

I. Populations

A. Evolution is change over time

B. Populations are the units of evolution

Microevolution detected here
Allelic and phenotypic freq. change in pops.

Contrast with macroevolution
1. Speciation
2. Extinction
3. Transitions from water to land, etc.

II. Evolution

A. Forces of evolution
1. Mutation (e.g., *Drosophila* data)
- *70% deleterious*
- *30% neutral or weakly advantageous*

II. Evolution

A. Forces of evolution
1. Mutation (e.g., *Drosophila* data)
2. Nonrandom Mating

-Primarily referring to individual traits or genes:
matings may or may not be random.

-At organismal level:
-most matings restricted geographically
-mate selection (sexual selection)

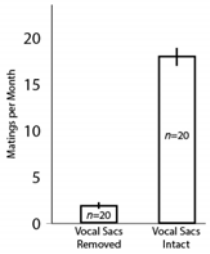
II. Evolution

Croaking used to attract mates
(by males in most species, but also females in some frogs)

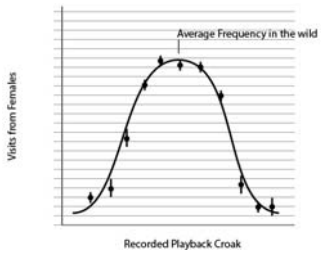
Force air through larynx,
but vocal sacs amplify.

Species-specific
Which means its selected for.

II. Evolution



II. Evolution

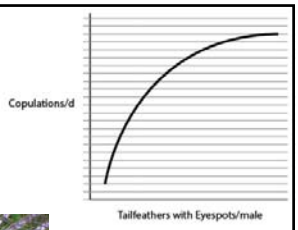


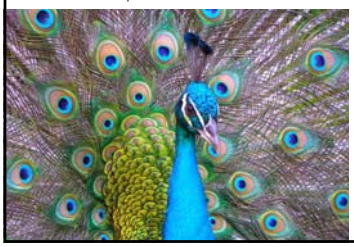
II. Evolution

Male peafowl (peacock) plumage
Displayed prominently during courtship

Fitness benefits
Attraction of peahens

Fitness Costs
Increase metabolic cost
Increased visibility & decreased flight speed in relation to predators?

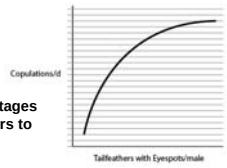


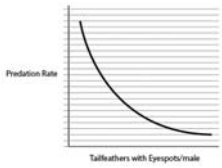


II. Evolution

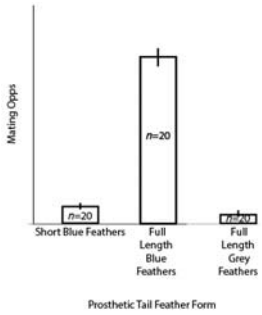
These two graphs point to two advantages peahens may have in using tailfeathers to select mate.

1.
2.





II. Evolution

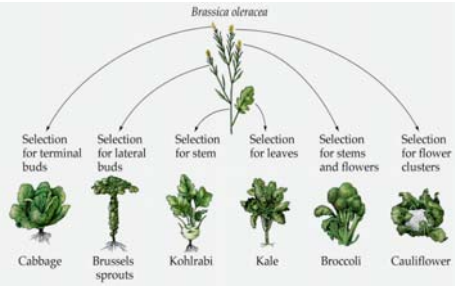


Artificial selection is human-controlled non-random mating (selective breeding)

Canis lupus

Canis lupus ssp. *familiaris*

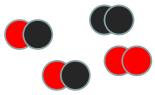
Artificial selection is human-controlled non-random mating (selective breeding)



II. Evolution

A. Forces of evolution
   1. Mutation (e.g., *Drosophila* data)
   2. Nonrandom Mating
   3. Migration (gene flow)

3. Migration

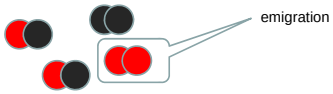


Population before

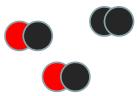
4 diploid individuals

Black allele (f = 0.50)
Red allele (f = 0.50)

3. Migration



3. Migration

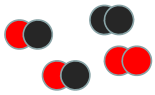


Population after

3 diploid individuals

Black allele (f = 0.67)
Red allele (f = 0.33)

3. Migration

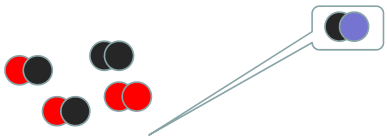


Population before

4 diploid individuals

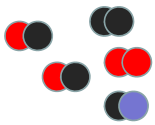
Black allele (f = 0.50)
Red allele (f = 0.50)

3. Migration



immigration

3. Migration



Population after

5 diploid individuals

Black allele (f = 0.50)
Red allele (f = 0.40)
Purple allelle (f = 0.10)

II. Evolution

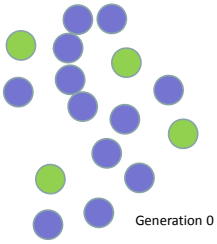
A. Forces of evolution
   1. Mutation (e.g., *Drosophila* data)
   2. Nonrandom Mating
   3. Migration
   4. Genetic drift

Chance-driven changes in trait frequencies.
Occurs more rapidly in small populations

4. Genetic drift
   a. General

Larger population

Phenotype	Count	Frequency
purple	12	0.75
green	4	0.25
	16	1.00

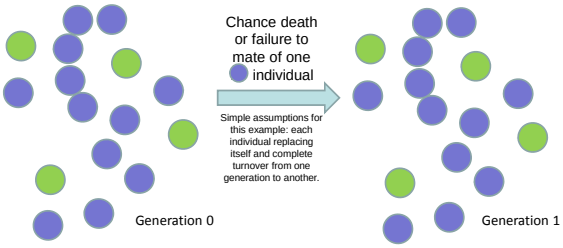


4. Genetic drift
   a. General

Larger population

Pheno	Count	Frequency
purple	12	0.75
green	4	0.25
	16	1.00

Pheno	Count	Frequency
purple	11	0.73
green	4	0.27
	15	1.00

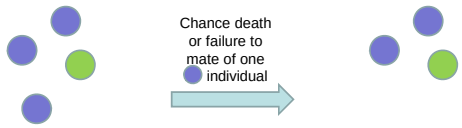


4. Genetic drift
a. General

Smaller population

Pheno	Count	Frequency
purple	3	0.75
green	1	0.25
	4	1.00

Pheno	Count	Frequency
purple	2	0.67
green	1	0.33
	3	1.00



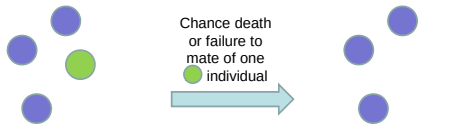
Generation 0 Generation 1

4. Genetic drift
a. General

Smaller population

Pheno	Count	Frequency
purple	3	0.75
green	1	0.25
	4	1.00

Pheno	Count	Frequency
purple	3	1.00
green	0	0.00
	3	1.00



Generation 0 Generation 1

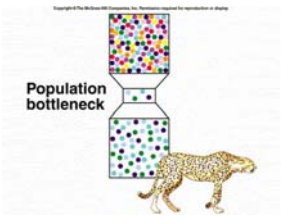
4. Genetic drift
b. Some things that produce small populations
and potentially drastic changes

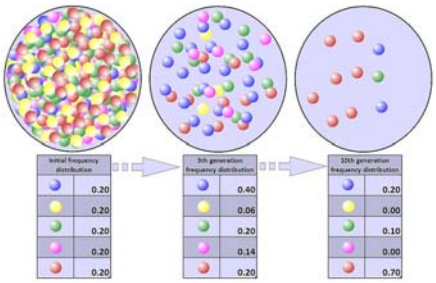
1) Founder Effect

Amish immigration – 1700s

1. Small founding population's allele freqs diff than parent population
   1 couple carried recessive allele for Ellis-van Creveld Syndrome (limb dwarfism, extra digits, etc.)
2. Interbreeding in small populations has kept freq. high.
3. Drift has influenced Amish population.

4. Genetic drift
   b. Some things that produce small populations and drastic changes
      1) Founder Effect
      2) Bottle Necks

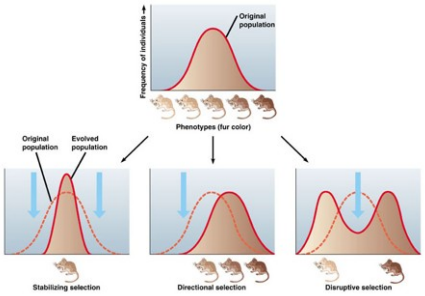




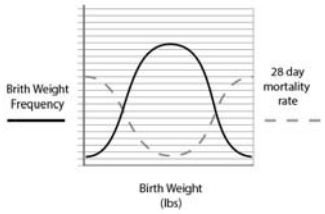
II. Evolution

A. Forces of evolution
   1. Mutation (e.g., *Drosophila* data)
   2. Nonrandom Mating
   3. Migration
   4. Genetic drift
   5. Natural selection (differential "fitness" based on phenotype)

a. Types of natural selection:
   1) Stabilizing, 2) Directional, 3) Disruptive

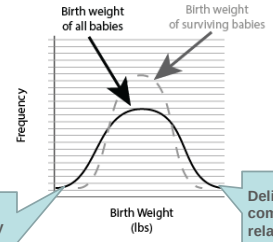


Birthweight in humans



Birthweight in humans

What type of selection here?
a. Directional
b. Stabilizing
c. Disruptive



Weakness causing difficulty eating, low fat content causing difficulty thermoregulating.

Delivery complications related to pelvic size, etc.

Pepper moths in Britain

Pepper moths in Britain

What type of selection here between 1850-1920?
a. Directional
b. Stabilizing
c. Disruptive

What type of selection here between 1920-present?
a. Directional
b. Stabilizing
c. Disruptive

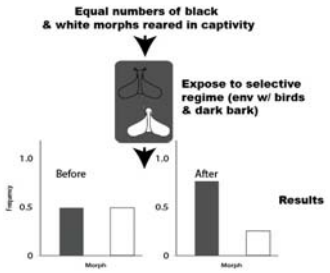
pre1850 – White-mottled morph most frequent. Camouflaged on trees with lichens.

1850 – Industrial Revolution in Britain

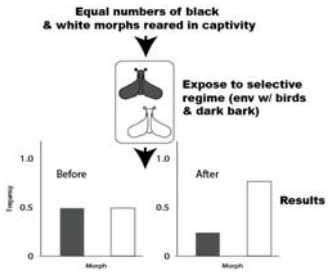
1850-1920 – Pollution and soot kill lichens, blacken trees. Black morph becomes more abundant.

1920-present – Cleaner fuel technology, emissions standards: lichens return, trees lighten, white morph becomes more abundant.

Pepper moths in Britain
Testing this hypothesis: Experiment 1



Pepper moths in Britain
Testing this hypothesis: Experiment 2



Pepper moths in Britain

Conclusions:

1) Selective Agent = predatory birds
2) Selective Regime (birds plus bark color frequencies) can and likely has influenced peppered moth evolution.

a. Types of natural selection:

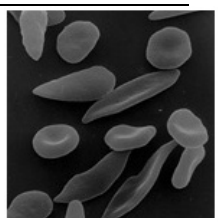
4) Balancing selection (e.g., heterozygote advantage)

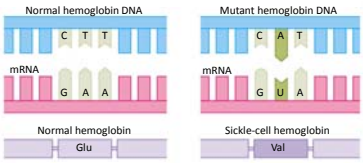
Where multiple alleles (even some "negative" ones) are actively maintained at freqs above that by which mutation alone would produce.

Balancing Selection Case Study*: Sickle-cell mutation*

Sickle-cell mutant

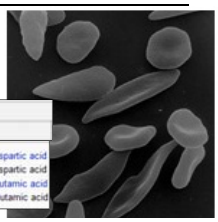
- Hemoglobin β gene on c-some11
- Missense substitution of 1 bp
  Results; GAA to GUA



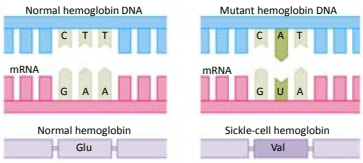


Sickle-cell mutant

[change to GAG would
have been a silent mutation]

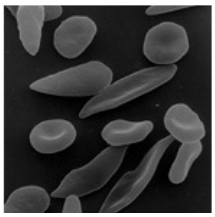


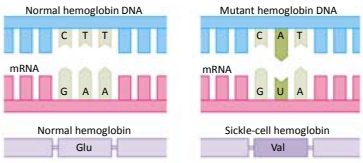
	2nd base		
	U	C	A
G	GUU (Val/V)Valine	GCU (Ala/A)Alanine	GAU (Asp/D)Aspartic acid
	GUC (Val/V)Valine	GCC (Ala/A)Alanine	GAC (Asp/D)Aspartic acid
	GUA (Val/V)Valine	GCA (Ala/A)Alanine	GAA (Glu/E)Glutamic acid
	GUG (Val/V)Valine	GCG (Ala/A)Alanine	GAG (Glu/E)Glutamic acid



Sickle-cell mutant

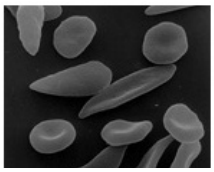
- Mutant hemoglobin aggregates into long rods.
  - deforms RBC
- Deformed RBC…
  - hinder blood flow to small capillaries.
  - Fragile & break

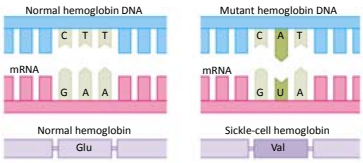




Sickle-cell Genetics

- 2 co-dominant alleles:
  - H_w
  - H_m.
- H_mH_m have sickle cell disease
  anemia,organ damage, joint pain, high mortality.
- H_wH_m are generally okay
  symptoms only w/ low O_2
  no affect on mortality.

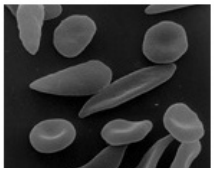


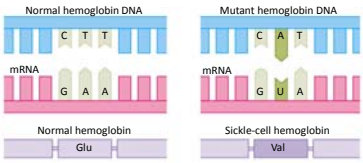


Sickle-cell Evolution

•Carrier Ratios In United States:
- •1 in 100,000 whites
- •1 in 1400 hispanics
- •1 in 12 blacks

•Carrier Ratios In Central Africa:
- •1 in 3 Africans





Sickle-cell Evolution

•Why so high in Africa?

Sickle-cell Evolution

- Why so high in Africa?
  - Malaria:
    - Plasmodium protist (infects and destroys RBC)
    - Mosquitos are vectors

Sickle-cell Evolution

- Why so high in Africa?
  - Malaria:
    - Plasmodium protist (infects and destroys RBC)
    - Mosquitos are vectors
  - Carriers (heterozygotes):
    - Plasmodium reproduction in RBC lowers O_2 levels, causing cells to sickle.
    - Sickled (infected) RBC destroyed.

Sickle-cell Evolution

- Why so high in Africa?

Parents: H_wH_m X H_wH_m

	H_w	H_m
H_w	H_wH_w (no mal. resistance)	H_wH_m (mal. resistance)
H_m	H_wH_m (mal. resistance)	H_mH_m (sickle cell disease)

Sickle-cell Evolution

•Why so high in Africa?

Parents: H_wH_m X H_wH_m

	H_w	H_m
H_w	H_wH_w (no mal. resistance)	H_wH_m (mal. resistance)
H_m	H_wH_m (mal. resistance)	H_mH_m (sickle cell disease)

Sickle-cell Evolution

•Why so high in Africa?

Parents: H_wH_w X H_wH_w

	H_w
H_w	H_wH_w (no mal. resistance)

Conclusion: H_wH_m are more fit than H_wH_w (or H_mH_m)

Sickle-cell Evolution

Why lower in African American population?

- •Carrier Ratios In United States:
  - •1 in 100,000 whites
  - •1 in 1400 hispanics
  - •1 in 12 blacks
- •Carrier Ratios In Central Africa:
  - •1 in 3 Africans

Conclusion:

Mutation Rate from Hw to Hm lower in N America?

Migration (gene flow).

Nonrandom mating?

~400 yrs of Nat Sel toward higher Hw freq.

H_wH_m (& H_mH_m) are less fit than H_wH_w

Sickle-cell Evolution

Punnett Square Demonstrates Reduced fitness of H_wH_m when selective regime doesn't include malaria

Parents: H_wH_m X H_wH_m

	H_w	H_m
H_w	H_wH_w	H_wH_m
H_m	H_wH_m	H_mH_m (sickle cell disease)