SCIENCE: BIOLOGY
UNIT #3: EVOLUTION MECHANISMS (5 WEEKS)

SYNOPSIS: Students are introduced to the four major mechanisms of evolution and how each affects changes in allele frequencies for various genes represented in populations over time. Students begin to see why a species can change so much that it becomes quite different from the ancestral species, or a part of the population can branch off and become a new species. Students explore the fact that natural selection and genetic drift cannot operate unless some individuals are genetically different from others. Students create a fictitious organism and explain how its population evolves because of the adaptive value of inherited variations in response to environmental changes and the impact of the four mechanisms of evolutionary change.

Enablers- Elementary**:** Evolution concepts include the relationship between organisms and the environment, parent and offspring, and an introduction to the fossil record and extinction. Middle school- Concepts include biodiversity (as part of biomes) and speciation, further exploration of the fossil record and Earth history, changing environmental conditions (abiotic factors), natural selection and biological evolution.

STANDARDS
II. EVOLUTION
  A. Mechanisms
    1. Natural selection is the process by which traits become more and less common in a population.
        a. it is due to consistent environmental effects upon the survival or reproduction of the individual with the trait (survival of the fittest)
        b. it works on the phenotype - - different phenotypes result from new combinations of existing genes or from mutations of genes in reproductive cells
        c. [combine grade 8 knowledge with] it explains the internal structure and function of chromosomes
        d. heritable characteristics influence how likely an organism is to survive and reproduce in a particular environment; when an environment changes, the survival value of inherited characteristics may change; this may or may not cause a change in species that inhabit the environment
    2. Mutations are described in the Content Elaboration for Heredity D.
        a. mutations are changes in a genomic sequence; i.e., the DNA sequence of a cell's genome or the DNA or RNA sequence of a virus
        b. they are defined as sudden and spontaneous changes in the cell
        c. mutations are caused by radiation, viruses, transposons and mutagenic chemicals, as well as errors that occur during meiosis or DNA replication
    3. Genetic drift involves mutation over time.
        a. mutation and genetic drift are evident in real-world examples (e.g., African cheetahs exist in small populations; and smaller populations have less variation and, therefore, a lower ability to respond favorably to changing conditions)
        b. descent with modification occurs in a population over time
    4. Gene flow involves both *immigration* and *emigration.*
        a. examples in the real-world include:
            (1) in the 1960s and1970s, U.S. soldiers had children in Southeast Asia with Vietnamese women; this altered the gene pool frequencies of the Vietnamese population.
            (2) if all red-haired people were to leave Scotland, the next generation there would likely have very few people with this trait; the Scottish population would have evolved - - as would the populations into which the red haired people migrated
            (3) segments of DNA may be transferred from one species to another by viruses as they invade the cells of animals or plants (rare, but documented in some species of insects, fish, reptiles, mammals, and especially microorganisms; not conclusively demonstrated for humans)
        b. explanations for gene flow are revised based on gene flow between species rather than within them
    5. Sexual selection is a type of natural selection that affects the traits that influence an individual's ability to attain or choose a mate, instead of the traits that influence an individual's ability to survive.
        a. different phenotypes result from new combinations of existing genes or mutations in reproductive cells
        b. explanations for sexual selection can be formulated and revised (e.g., many elaborate morphological features, such as long plumes in birds, courtship displays, and bright colors)

LITERACY STANDARDS

RST.2   Determine the central ideas or conclusions of a text; trace the text's explanation or depiction of a complex process, phenomenon, or concept; provide an accurate summary of the text.

RST. 4   Determine the meaning of symbols, key terms, and other domain-specific words and phrases as they are used in a specific scientific or technical context relevant to *grades 9–10 texts and topics*.

MOTIVATION	TEACHER NOTES
1. Teacher introduces evolution unit and relates it to their understanding of adaptations; students do **Build a Beast activity (attached**) which demonstrates how adaptations are important to evolution. 2. Teacher explains that there are many ideas about how evolution works; small groups pick a written statement out of fishbowl about evolution and reflect on what they know about it. The statements are misconceptions of evolution which helps the teacher find out the students' previous knowledge. Teacher records on chart paper the ideas presented for each statement. website - http://evolution.berkeley.edu/evosite/misconceps/IHowitworks.shtml (copy statements from heading on each page) or http://evolution.berkeley.edu/evolibrary/misconceptions_faq.php (click on statement for explanation) 3. Teacher previews for students what the end of the unit authentic assessment will be and what students will be expected to do. 4. Students write personal and academic goals	1. **Build a Beast activity attached, pages 6-7**

TEACHING-LEARNING	TEACHER NOTES
1. Teacher introduces evolution using website visuals; students record notes about the definition of evolution, why the definition for mutation is descent with modification rather than change over time and how the process produces a pattern of relationships between species. **(II A.1d)** http://evolution.berkeley.edu/evolibrary/article/evo_02 **(**click on left side for topics to use as intro) 2. Teacher introduces the mechanisms of Natural Selection using video PPT – Recipe for Evolution -Variation, Selection and Time which shows how molecular genetics is shedding light on the process of natural selection; students discuss how variation, selection and time provide the three ingredients for the evolution recipe. Teacher shows video about the evolution of the eye; students record video examples how natural selection has generated a diverse collection of eyes, from simple to complex, that help animals sense their surroundings. **(II A.1 a,b,c,d)** Video PPT - http://learn.genetics.utah.edu/content/variations/recipe 3. Teacher explains how groups will conduct simulation to study how mutations in the DNA of an organism can lead directly to the evolution of the population it is a part of; students control the mutations and environment of a population of rabbits. Groups create four hypotheses and design an experiment to test each one. An interactive can be done to see how camouflage protects peppered moths through the eyes of a predator. **(II A. 1a,b,c,d)** Natural Selection Bunny Simulation- http://phet.colorado.edu/en/simulation/natural-selection **OR** Peppered Moths interactive -http://www.techapps.net/interactive/pepperMoths.swf	

TEACHING-LEARNING	TEACHER NOTES
4. Teacher identifies the fundamental assumptions of **Darwin's perspective of evolution**; students view a short segment of Carl Sagan's Cosmos video "*One Voice in the Cosmic Fugue*" in which Sagan follows the story of the Japanese Heike crab, whose form gradually changed as people selected which crabs should live and which should die. Students use evidence of the Heike crabs to explain how humans influenced the gradual change in the design of the crabs. **(II A. 1a,b,c,d)**	4. **Darwin's Explanation of Natural Selection attached, page 8**
5. Teacher shows **PPT: Genetic Variations (show slides 1-12)** to show how variations are simply differences in genetic sequence and can be seen at every genetic level; students take notes to compare variations and the mutations they cause in terms whether or not they affect our ability to survive or adapt. In small groups students read an assigned part of an article to find out about favorable mutations; groups share out the information they discovered. **(II A.2 a,b,c)**	5. **PPT: Genetic Variations** 5. **Article – Are There Favorable Mutations? attached, pages 9-10** 6. **Article: A Mutation Story attached page 11**
6. Teacher introduces sickle cell anemia; students receive copy of the video background information and highlight/underline parts that teacher indicates will relate to what they see in the video. Students watch video - **A Mutation Story** (4:50) which tells the story of a genetic mutation affecting the population of West Africa; class discusses video. **(II A.2 a,b,c)**	7. **Article - A Case Study of the Effects of Mutation: Sickle Cell Anemia attached, page 12-13**
7. Teacher projects article on board and explains that what happens at the DNA level continues up to the level of the complete organism; students take notes. Students read the **student notes – Genetics of Sickle Cell Anemia**; teacher discusses article with class. Students complete activity based on information in the article. **(II A.2 a,b,c)**	7. **Genetics of Sickle Cell Anemia – students notes, student activity, teacher's answer key attached, pages 14-17**
8. Teacher introduces transposons or "jumping genes using website information and clicks on animated visual to make a comparison between the two methods of transposition; students draw and label both diagrams in visual and write an explanation why the word transposon offers an accurate description of these kinds of genes. Students observe animation and answer quiz questions at the end. **Teacher notes with additional information are attached**. **(II A.2 a,b,c)** Website - http://www.broadinstitute.org/education/glossary/transposable-elements Video animation - Transposons: Shifting Segments of the Genome (Google – McGraw Hill animation of Transposons)	8. **Teacher Notes: Transposons and Mutations (attached, pages 18-19)**
9. Teacher introduces genetic drift and explains that through sampling error, genetic drift can cause populations to lose genetic variation; students view website pages and write how the examples given relate to changes in the population. **(II A.3 a,b)** wwebsite - http://evolution.berkeley.edu/evosite/evo101/IIIDGeneticdrift.shtml (view pages - genetic drift, sampling error and evolution, effects of genetic drift 1 & 2)	
10. Teacher introduces genetic drift simulation: students examine a simple model of a population of fictional organisms to visualize the evolution of color in the population. Students color a **Worm Worksheet** with the simulation or if using the manual version included at the site. This simulation focuses on drift. (*Note: pay attention to the brackets; especially in simulation panels 3 and 4 for Random Genetic Drift section.*) **(II A.3 a,b)** simulation - http://www.biology.arizona.edu/evolution/act/drift/about.html	10. **Worm Worksheet attached, page 20**
11. Teacher explains the effect of gene flow on populations referring to several examples of gene flow included in the standards (II A.4a.1,2); students take notes. **(II A.4 a,b)** (*optional*) Teacher introduces gene flow activity; students gather data from **Breeding**	11. **Activity: Breeding Bunnies attached, pages 21-25**

TEACHING-LEARNING	TEACHER NOTES
Bunnies activity, graph results and answer discussion questions (attached). Class discusses results of the activity. **(II A.4 b)**	11. **Breeding Bunnies Discussion Questions – attached, page 26**
12. Students read article: **Gene flow from India to Australia (attached**); class discusses how this migration affected the human gene pool. **Students read article about the first humans who ventured out of Africa** some 60,000 years ago and left genetic footprints still visible today. These great migrations eventually led the descendants of a small group of Africans to occupy even the farthest reaches of the Earth and have left a dramatic impact on the human gene pool. A map of the migrations is shown at the website. The class examines the migration routes (see website) to determine when humans left Africa, where they migrated first, what routes they took as they spread around the world, and how long it took them to reach different places. **(II A.4 b)** website - http://genographic.nationalgeographic.com/human-journey/	12. **Article - Gene Flow From India to Australia attached, page 27** 12. **Article – The Human Journey: Migration Routes attached, page 28**
13. (*Optional*) Teacher divides students into small "detective teams" and imagine they are researchers working on the Genographic Project; students brainstorm how they would solve the migration mystery and refer to questions written on the board *Where would you start? Where would you go? What clues would you look for? What technologies and scientific techniques would you use?* Groups share their ideas, and list their solutions on the board. Teacher reminds them of the name of the project—Genographic—is an important clue(if students do not mention genes or DNA as one possible clue). **(II A.4 b)**	
14. Teacher lectures about sexual selection and how it affects the gene pool (**notes attached**); students take notes and read article (attached). Student complete literacy analysis (attached) of article by completing questions about the article they have read. **(II A.5 a,b) (RST-2, RST-4)**	14. **Sexual Selection – Teacher Notes and Article: Sexual Selection attached, pages 29-31**
15. Students conduct two interactive activities about sexual selection and **summarize** what they learned from each activity – **(RST.2; RST.4)** The Mating Game This mating game interactive involves choosing the right partner which helps you pass your genes on to the next generation, but choosing the wrong one can send you straight down an evolutionary dead end. Sex and the Single Guppy Play in our streams and see how exhibitionism has an evolutionary payoff in this simulation of Endler's experiment. **(II A.5 a,b)** Activities website: http://www.pbs.org/wgbh/evolution/sex/mating	14. **Literacy Analysis of Article attached, pages 32-34**

TEACHER CLASSROOM ASSESSMENT	TEACHER NOTES
1. 2 pt. and 4 pt. response questions 2. Science Notebooks – includes students work on labs, activities, literacy standards 3. Quizzes 4. Out of class work – research	

TEACHER CLASSROOM ASSESSMENT	TEACHER NOTES
1. Unit Test	

AUTHENTIC ASSESSMENT	TEACHER NOTES

1. Students design a fictitious organism with realistic traits (not nonsense features) and tell its evolutionary story. Students will:
    - Build a model or draw a picture of the organism.
    - Create a visual (e.g., chart, poster, ppt) to show the environment where it lives compared to the environment of it's ancestors.
    - Describe 5 inherited variations which helped the organism to survive when its environment changed and describe how each variation provided significant adaptive value.
    - Explain how each of Darwin's 4 mechanisms of evolution could influence the evolution of a population of organisms like this one.
 	
2. Students evaluate their goals.	

MOTIVATION #1: BUILD A BEAST EVOLUTION ACTIVITY

This activity demonstrates an important part of evolution: adaptation. Adaptation is "an alteration or adjustment in structure or habits, often hereditary, by which a species or individual improves its condition in relationship to its environment."[1] In order to survive in an environment, an animal needs to adapt, to develop traits that fit it to where it lives. For example, mountain goats have padded hooves to grip the rocks on which they climb, giraffes are tall to reach the leaves of tall trees, and frogs have long, sticky tongues to catch insects to eat, and are colored brown or green to match their environments so that predators do not notice them. In fact, nearly everything about an animal is an adaptation of one kind or another. Think of a trait that an animal has, and then think of how that trait helps it to survive or reproduce in its environment.

Plants also adapt to their environments in order to survive. For example, cacti are able to store water because they live in dry places, and they have spines to discourage animals from eating them. The flowers of flowering plants are an adaptation for attracting bees and butterflies, which enable the plant to reproduce.

This activity is about creating animals with useful adaptations. The animal's environment is given, because that is how Darwin's theory of evolution and adaptation work: the individual organisms that are best adapted to their particular environments survive, so the adaptations gradually appear in more and more of the population. Different adaptations are helpful for different environments: For a rabbit living in the Arctic, white fur would be helpful to avoid being seen by predators. For a rabbit living in the woods, being white would make it more conspicuous, but being brown would be helpful.

To "build a beast," first roll a die for each category to determine the conditions under which the animal lives. Then make up, write about, and draw an animal which has adaptations to be able to live well in that kind of environment.

A) WHERE DOES IT LIVE?
1 - mountains
2 - flatlands
3 - rocky, harsh
4 - small island
5 - near a volcano
6 - in a cave

B) HOW MUCH WATER IS THERE?
1 - almost none; dry and barren
2 - water part of the year, drought the rest
3 - lots of precipitation all year
4 - near a coastline
5 - in a swamp
6 - in the ocean

C) WHAT IS THE CLIMATE/WEATHER LIKE?
1 - hot and humid
2 - hot and dry
3 - moderate
4 - cold, rainy, and windy
5 - seasons change from hot to cold
6 - sub-zero temperatures

D) WHAT DOES IT EAT?
1 - leaves from tall plants
2 - fungus growing under rocks
3 - berries, plants, and small animals
4 - water animals

5 - swift running deer-like animals
6 - flying insects

E) WHAT EATS IT?
1 - stompsuckers squash it flat
2 - vampire butterflies land on it and suck it dry
3 - buzzbugs lay eggs that burrow into its skin
4 - web devils set gooey traps to catch it
5 - ratrax packs are wolf-like and chase it

TEACHING-LEARNING # 4   Darwin's EXPLANATION OF NATURAL SELECTION

<u>Darwin's Model of Natural Selection</u>:

- Populations are made up of individual organisms whose traits (structural, behavioral, biochemical) exhibit variation from one individual to the next
- Many trait variations are inherited
- Some trait variations may be advantageous while others may not
- Whether or not a variation is advantageous depends on the environment within which a population lives
- The frequency of particular trait variations in a population may change over time due to differential survival and reproduction
- Evolutionary changes occur in populations or species, not individuals
- Offspring are not exactly like their parents-they also vary, but the variations of their traits are likely to be similar to those of their parents
- Variability in a population can be represented graphically
- Organisms produce more offspring than can survive
- There is competition between organisms for resources

_____

TEACHING-LEARNING # 5   Article: *Are There Favorable Mutations?*

Excepts taken from Article: Are Mutations Harmful? by **Richard Harter** , Copyright © 1999-2003

People often ask questions such as "Doesn't evolution depend on mutations, and aren't most mutations harmful?" and "Are there favorable mutations?". In this FAQ we try to answer these questions. Briefly:

- Mutations happen.
- They happen with great regularity.
- Almost all mutations are neutral.
- Of the remainder, benefit/harm depends on circumstances

Q: Are there favorable mutations?
A: There are, but it can be hard to tell.

For a number of reasons it is not simple to give examples of favorable mutations. First of all, as we have seen, traits may be favorable or unfavorable, depending upon the environment. Secondly it is not usually known to what extent a trait is genetically fixed and to what extent it reflects a reaction to the environment. Thirdly we don't usually know what genes effect which traits. Moreover a mutation may be favorable in the sense that it permits survival in an unfavorable environment and yet be unfavorable in a better environment.

However there are a number of good examples**:**

1. ***Antibiotic resistance in bacteria***
    In modern times antibiotics, drugs that target specific features of bacteria, have become very popular. Bacteria evolve very quickly so it is not surprising that they have evolved resistance to antibiotics. As a general thing this involves changing the features that antibiotics target.

    Commonly, but not always, these mutations decrease the fitness of the bacteria, i.e., in environments where there are not antibiotics present, they don't reproduce as quickly as bacteria without the mutation. This is not always true; some of these mutations do not involve any loss of fitness. What is more, there are often secondary mutations that restore fitness.

    Bacteria are easy to study. This is an advantage in evolutionary studies because we can see evolution happening in the laboratory. There is a standard experiment in which the experimenter begins with a single bacterium and lets it reproduce in a controlled environment. Since bacteria reproduce asexually all of its descendants are clones. Since reproduction is not perfect mutations happen. The experimenter can set the environment so that mutations for a particular attribute are selected. The experimenter knows both that the mutation was not present originally and, hence, when it occurred.

    In the wild it is usually impossible to determine when a mutation occurred. Usually all we know (and often we do not even know that) is the current distribution of particular traits.

    The situation with insects and pesticides is similar to that of bacteria and antibiotics. Pesticides are widely used to kill insects. In turn the insects quickly evolve in ways to become immune to the pesticides.

2. ***Bacteria that eat nylon***
    Well, no, they don't actually eat nylon; they eat short molecules (nylon oligomers) found in the waste waters of plants that produce nylon. They metabolize short nylon oligomers, breaking the nylon linkages with a couple of related enzymes. Since the bonds involved aren't found in natural products, the enzymes must have arisen since the time nylon was invented (around the 1940s). It would appear this happened by new mutations in that time period.

    These enzymes which break down the nylon oligomers appear to have arisen by frameshift mutation from some other gene which codes for a functionally unrelated enzyme. This adaptation has been experimentally duplicated. In the experiments, non-nylon-metabolizing strains of Pseudomonas were grown in media with nylon oligomers available as the primary food source. Within a relatively small number of generations, they developed these enzyme activities. This would appear to be an example of documented occurrence of beneficial mutations in the lab.

3. *Sickle cell resistance to malaria*

   The sickle cell allele causes the normally round blood cell to have a sickle shape. The effect of this allele depends on whether a person has one or two copies of the allele. It is generally fatal if a person has two copies. If they have one they have sickle shaped blood cells.

   In general this is an undesirable mutation because the sickle cells are less efficient than normal cells. In areas where malaria is prevalent it turns out to be favorable because people with sickle shaped blood cells are less likely to get malaria from mosquitoes.

   This is an example where a mutation decreases the normal efficiency of the body (its fitness in one sense) but none-the-less provides a relative advantage.

4. *Lactose tolerance*

   Lactose intolerance in adult mammals has a clear evolutionary explanation; the onset of lactose intolerance makes it easy to wean the young. Human beings, however, have taken up the habit of eating milk products. This is not universal; it is something that originated in cultures that kept cattle and goats. In these cultures lactose tolerance had a strong selective value. In the modern world there is a strong correlation between lactose tolerance and having ancestors who lived in cultures that exploited milk as a food.

   It should be understood that it was a matter of chance that the lactose tolerance mutation appeared in a group where it was advantageous. It might have been established first by genetic drift within a group which then discovered that they could use milk. [9]

5. *Resistance to atherosclerosis*

   Atherosclerosis is principally a disease of the modern age, one produced by modern diets and modern life-styles. There is a community in Italy near Milan (see Appendices II and III for biological details) whose residents don't get atherosclerosis because of a fortunate mutation in one of their forebearers. This mutation is particularly interesting because the person who had the original mutation has been identified.

   Note that this is a mutation that is favorable in modern times because (a) people live longer and (b) people have diets and life-styles that are not like those of our ancestors. In prehistoric times this would not have been a favorable mutation. Even today we cannot be certain that this mutation is reproductively favorable, i.e., that people with this mutation will have more than the average number of descendents. It is clear, however, that the mutation is personally advantageous to the individuals having it.

6. *Immunity to HIV*

   HIV infects a number of cell types including T-lymphocytes, macrophages, dendritic cells and neurons. AIDS occurs when lymphocytes, particularly CD4+ T cells are killed off, leaving the patient unable to fight off opportunistic infections. The HIV virus has to attach to molecules that are expressed on the surface of the T-cells. One of these molecules is called CD4 (or CD4 receptor); another is C-C chemokine receptor 5, known variously as CCR5, CCCKR5 and CKR5. Some people carry a mutant allele of the CCR5 gene that results in lack of expression of this protein on the surface of T-cells. Homozygous individuals are resistant to HIV infection and AIDS. The frequency of the mutant allele is quite high in some populations that have never been exposed to AIDS so it seems likely that there was prior selection for this allele.

TEACHING-LEARNING #6   Article – *A Mutation Story*   (background information for PBS video)

A gene known as HbS was the center of a medical and evolutionary detective story that began in the middle 1940s in Africa. Doctors noticed that patients who had sickle cell anemia, a serious hereditary blood disease, were more likely to survive malaria, a disease which kills some 1.2 million people every year. What was puzzling was why sickle cell anemia was so prevalent in some African populations.

How could a "bad" gene -- the mutation that causes the sometimes lethal sickle cell disease -- also be beneficial? On the other hand, if it didn't provide some survival advantage, why had the sickle gene persisted in such a high frequency in the populations that had it?

The sickle cell mutation is a like a typographical error in the DNA code of the gene that tells the body how to make a form of hemoglobin (Hb), the oxygen-carrying molecule in our blood. Every person has two copies of the hemoglobin gene. Usually, both genes make a normal hemoglobin protein. When someone inherits two mutant copies of the hemoglobin gene, the abnormal form of the hemoglobin protein causes the red blood cells to lose oxygen and warp into a sickle shape during periods of high activity. These sickled cells become stuck in small blood vessels, causing a "crisis" of pain, fever, swelling, and tissue damage that can lead to death. This is sickle cell anemia.

But it takes two copies of the mutant gene, one from each parent, to give someone the full-blown disease. Many people have just one copy, the other being normal. Those who carry the sickle cell trait do not suffer nearly as severely from the disease.

Researchers found that the sickle cell gene is especially prevalent in areas of Africa hard-hit by malaria. In some regions, as much as 40 percent of the population carries at least one HbS gene.

It turns out that, in these areas, HbS carriers have been naturally selected, because the trait confers some resistance to malaria. Their red blood cells, containing some abnormal hemoglobin, tend to sickle when they are infected by the malaria parasite. Those infected cells flow through the spleen, which culls them out because of their sickle shape -- and the parasite is eliminated along with them.

Scientists believe the sickle cell gene appeared and disappeared in the population several times, but became permanently established after a particularly vicious form of malaria jumped from animals to humans in Asia, the Middle East, and Africa.

In areas where the sickle cell gene is common, the immunity conferred has become a selective advantage. Unfortunately, it is also a disadvantage because the chances of being born with sickle cell anemia are relatively high.

For parents who each carry the sickle cell trait, the chance that their child will also have the trait -- and be immune to malaria -- is 50 percent. There is a 25 percent chance that the child will have neither sickle cell anemia nor the trait which enables immunity to malaria. Finally, the chances that their child will have two copies of the gene, and therefore sickle cell anemia, is also 25 percent. This situation is a stark example of genetic compromise, or an evolutionary "trade-off."

TEACHING-LEARNING # 6: Article -*A Case Study of the Effects of Mutation: Sickle Cell Anemia*

Sickle cell anemia is a genetic disease with severe symptoms, including pain and anemia. The disease is caused by a mutated version of the gene that helps make hemoglobin — a protein that carries oxygen in red blood cells. People with two copies of the sickle cell gene have the disease. People who carry only one copy of the sickle cell gene do not have the disease, but may pass the gene on to their children.

The mutations that cause sickle cell anemia have been extensively studied and demonstrate how the effects of mutations can be traced from the DNA level up to the level of the whole organism. Consider someone carrying only one copy of the gene. She does not have the disease, but the gene that she carries still affects her, her cells, and her proteins:

1. There are effects at the DNA level
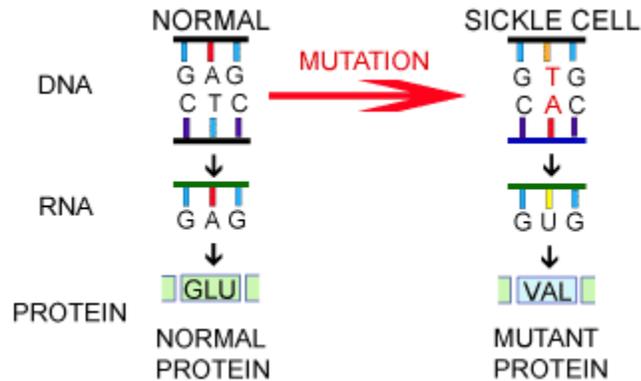
2. There are effects at the protein level
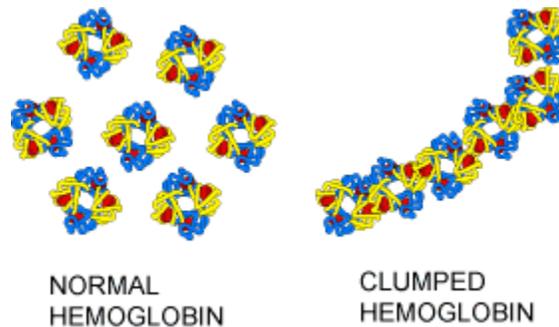
Normal hemoglobin (left) and hemoglobin in sickled red blood cells (right) look different; the mutation in the DNA slightly changes the shape of the hemoglobin molecule, allowing it to clump together.
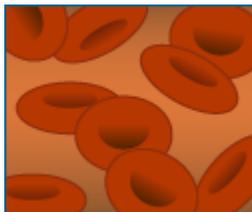
Normal red blood cells   Sickle cells

3. There are effects at the cellular level
When red blood cells carrying mutant hemoglobin are deprived of oxygen, they become "sickle-shaped" instead of the usual

round shape (see picture). This shape can sometimes interrupt blood flow.

4. There are negative effects at the whole organism level
Under conditions such as high elevation and intense exercise, a carrier of the sickle cell allele may occasionally show symptoms such as pain and fatigue.

5. There are positive effects at the whole organism level
Carriers of the sickle cell allele are resistant to malaria, because the parasites that cause this disease are killed inside sickle-shaped blood cells.

This is a chain of causation. What happens at the DNA level propagates up to the level of the complete organism. This example illustrates how a single mutation can have a large effect, in this case, both a positive and a negative one. But in many cases, evolutionary change is based on the accumulation of many mutations, each having a small effect. Whether the mutations are large or small, however, the same chain of causation applies: changes at the DNA level propagate up to the phenotype.

TEACHING-LEARNING # 7   GENETICS OF SICKLE-CELL ANEMIA

STUDENT SHEETS – GENETICS OF SICKLE CELL ANEMIA

What is sickle cell disease?

Sickle cell anemia was the first genetic disease to be described in terms of a gene mutation. The mutation changes the molecule hemoglobin, causing the red blood cells (RBC's) to become stiff and sometimes sickle-shaped when they release oxygen to the body tissues. The sickled cells tend to get stuck in narrow blood vessels, blocking the flow of blood. As a result, those with sickle cell disease suffer painful "crises" in their joints and bones. They may suffer strokes, blindness, or damage to the lungs, kidneys, or heart. They must often be hospitalized for blood transfusions and are at risk for a life-threatening complication called acute chest syndrome. Although many sufferers of sickle cell disease die before the age of 20, modern medical treatments can sometimes prolong these individuals' lives into their 40's and 50's.

How is sickle cell disease inherited?

There are two versions or **alleles** of the gene important for the inheritance of sickle cell anemia: A and S. Individuals with two A alleles (AA) have normal hemoglobin, and therefore normal RBCs. Those with two S alleles (SS) develop sickle cell anemia. Those who are heterozygous for the sickle cell allele (AS) produce both normal and abnormal hemoglobin. Heterozygous individuals are usually healthy, but they may suffer some symptoms of sickle cell anemia under conditions of low blood oxygen, such as high altitudes or during exercise. Heterozygous (AS) individuals are said to be 'carriers' of the sickle cell gene. Both forms of hemoglobin are made in the AS heterozygotes (this is different from the situation with the more common dominant and recessive genes where either the dominant *or* the recessive characteristic is expressed). Heterozygotes are said to have 'sickle cell trait'.

Sickle cell anemia and natural selection

In the United States, about 1 in 500 African-Americans develops sickle cell anemia. In Africa, about 1 in 100 individuals develops the disease. Why is the frequency of such a serious disease so much higher in Africa? The answer is to do with natural selection and malaria.

In the late 1940s, studies of diseases in populations suggested a connection between African populations, malaria and sickle cell disease. This is one of many examples where clinical observations led to further investigations, advancing other areas of biology such as molecular biology, genetics and physiology. The geneticist J. B. S. Haldane suggested a theory of how genetic diseases could evolve by natural selection; if the heterozygotes (AS) are protected from malaria, and the negative effects are only present in the small proportion of people who are homozygous for the affected allele (SS) the affected allele could become more common. In 1949 the molecular biologist Linus Pauling described differences between the structures of normal and sickle cell hemoglobin. It wasn't until the 1950s, when Watson and Crick proposed their model of DNA structure, that the links between genetic material (DNA), mutations, genetic diseases and proteins in cells were understood. Later studies supported

Haldane's theory, showing that African children who are heterozygous for the sickle cell allele have a ten-fold reduction in their risk of getting malaria. The exact position of the sickle cell allele is now known, and gene therapy for sickle cell disease in mice has been successfully carried out early in the 21st century by treating the stem cells which give rise to red blood cells.

Malaria is characterized by chills and fever, vomiting, and severe headaches. Anemia and death may result. Malaria is caused by a protozoan parasite *(Plasmodium)* that is transmitted to humans by mosquitoes. When malarial parasites invade the bloodstream of a person with the S allele, the red cells that contain defective hemoglobin become sickled and die, trapping the parasites inside them and reducing infection.

Compared to AS heterozygotes, people with the AA genotype (normal hemoglobin) have a greater risk of dying from malaria. AA homozygotes that die young may not pass on their genes to the next generation. Individuals with the AS genotype do not develop sickle cell anemia and also have less chance of getting malaria. They are able to survive and reproduce in malaria-infected regions. AS heterozygotes pass on A and S alleles to the next generation, so both the A and S alleles of these people remain in the population. SS homozygotes have sickle cell anemia, which usually results in early death. S alleles are removed from the population's 'gene pool' when SS people die. In a region where malaria is common, the S allele gives a survival advantage to people who have one copy of the allele, and the otherwise harmful S allele is kept in the population at a relatively high frequency.

STUDENT ACTIVITY – GENETICS OF SICKLE CELL ANEMIA

Complete the table and answer questions.
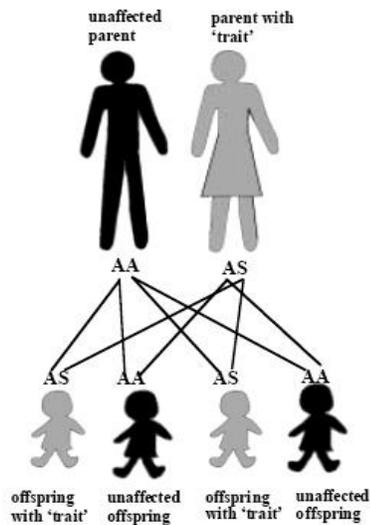
1. The diagram shows the alleles of parents and offspring where one of the parents is a carrier for sickle cell anemia. Draw a similar diagram where one parent is unaffected and the other is homozygous for the sickle cell allele i.e. SS.

What proportion of the offspring from the second cross

(a) are unaffected

(b) have sickle cell trait (carriers)

(c) have sickle cell anemia?

2. Use the information in the text to complete the table below:

Genetic make-up of individual	Characteristic of individual	Likely outcome with no malarial infection	Likely outcome with malarial infection
AA			dies young
AS	sickle cell trait	survives	
SS			

3. Explain why the frequency of sickle cell allele is so much lower in Northern Europe than in Africa.

TEACHER'S ANSWER KEY - GENETICS OF SICKLE CELL ANEMIA
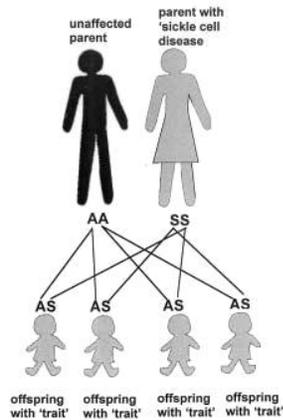
What percentage of the offspring have the sickle cell disease?

(a) none

(b) 100% /all of them

(c) <u>none</u>   (all of them are carriers of the disease gene)

2. Complete the following chart

Genetic make-up of individual	Characteristic of individual	Likely outcome with no malarial infection	Likely outcome with malarial infection
AA	unaffected	survives	dies young
AS	sickle cell trait	survives	survives
SS	sickle cell disease	dies young	dies young

3. The sickle cell allele only becomes common in areas where it gives an advantage to people carrying the allele.

In countries, such as Africa where there is malaria, carriers of the sickle cell allele (AS) are less likely to die of malaria than unaffected people (AA). The A allele becomes less common as AA people die young of malaria, and the S allele becomes more common because heterozygotes (AS) survive. In Northern Europe, the S allele gives no advantage, so remains rare.

_____

TEACHING-LEARNING # 8: Teacher Notes: Transposons and Mutations

Introduction

Transposons are short DNA sequences that have the ability to move from one location in DNA to another location. Transposons encode an enzyme, transposase, that acts to splice the transposon into new locations in a genome (see schematic, below left, of a transposon) Transposons were discovered by Barbara McClintock and she won a Nobel prize for her work. The human genome contains many inactivated copies of transposons that have lost their ability to move or 'jump' to new locations. About 50% of the human genome is composed of 'dead' transposons.

The movement of active transposons can lead to mutations, altering the activity of genes. A visible example of transposon movement (called transposition), is the coloration of the kernels in Indian corn (see below right). The transposons that are active in humans are thought to be involved in human disease, including cancer.
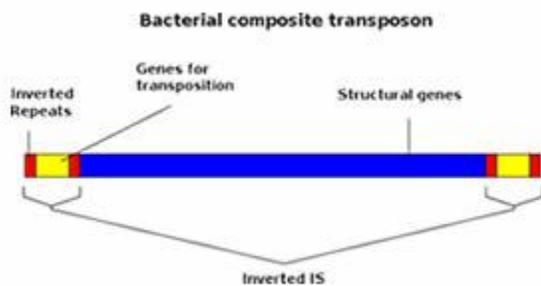
How can transposons cause evolutionary changes?

Transposons are segments of the genome that have the ability to both replicate themselves and to insert themselves elsewhere in the genome. The function of transposons is merely to replicate themselves. However they can potentially play an important role in evolution by copying and transferring other genetic material. There is evidence that transposons and their host genomes have co-evolved over long periods and that transposon activity may increase in response to environmental factors. The movement of transposons is a source that increases genetic variability. In particular, transposons contain regulatory genes that control transposon activity. It is thought that these regulatory genes may evolve to control other genes as well.

Transposons: Mobile DNA

Transposons are segments of DNA that can move around to different positions in the genome of a single cell. In the process, they may

- cause mutations
- increase (or decrease) the amount of DNA in the genome of the cell, and if the cell is the precursor of a gamete, in the genomes of any descendants.

These mobile segments of DNA are sometimes called **"jumping genes"**.

There are two distinct types:

- **Class II transposons**. These consist of DNA that moves directly from place to place.
- **Class I transposons**. These are **retrotransposons** that first transcribe the DNA into RNA and then use reverse transcriptase to make a DNA copy of the RNA to insert in a new location.

CLASS II TRANSPOSONS: Class II transposons move by a "**cut and paste**" process: the transposon is cut out of its location (like **command/control-X** on your computer) and inserted into a new location (**command/control-V**).

This process requires an enzyme — a **transposase** — that is encoded within some of these transposons.

Transposase binds to:

- both ends of the transposon, which consist of **inverted repeats**; that is, identical sequences reading in opposite directions.

- a sequence of DNA that makes up the target site. Some transposases require a specific sequence as their target site; others can insert the transposon anywhere in the genome.

The DNA at the target site is cut in an offset manner (like the "sticky ends" produced by some restriction enzymes [Examples]).

After the transposon is ligated to the host DNA, the gaps are filled in by Watson-Crick base pairing. This creates identical **direct repeats** at each end of the transposon.

Often transposons lose their gene for transposase. But as long as somewhere in the cell there is a transposon that can synthesize the enzyme, their inverted repeats are recognized and they, too, can be moved to a new location.

CLASS I TRANSPOSONS (OR RETROTRANSPOSONS)

Retrotransposons also move by a "**copy and paste**" mechanism but in contrast to the transposons described above, the copy is made of RNA, not DNA.

The RNA copies are then transcribed back into DNA — using a reverse transcriptase — and these are inserted into new locations in the genome.

Many retrotransposons have **long terminal repeats** (**LTRs**) at their ends that may contain over 1000 base pairs in each.

Like DNA transposons, retrotransposons generate direct repeats at their new sites of insertion. In fact, it is the presence of these direct repeats that often is the clue that the intervening stretch of DNA arrived there by retrotransposition.

Some 50% of the entire human genome consists of retrotransposons.

Generations of Driftworms

Name(s): _____

Parent #

0	1	2	3	4	5	6	7	8	9
1									
2									
3									
4									
5									

10	11	12	13	14	15	16	17	18	19
1									
2									
3									
4									
5									

20	21	22	23	24	25	26	27	28	29
1									
2									
3									
4									
5									

30	31	32	33	34	35	36	37	38	39
1									
2									
3									
4									
5									

40	41	42	43	44	45	46	47	48	49
1									
2									
3									
4									
5									

Name_____

Breeding Bunnies

Background Information: Sometimes the frequency of alleles changes in a population over a period of time. This means that how often you will see a particular trait will change also.

Breeders of rabbits have long been familiar with a variety of genetic traits that affect the ability of rabbits to survive in the wild, as well as in breeding populations. One such trait is the trait for furless rabbits (naked bunnies). This trait was first discovered in England by W.E. Castle in 1933. The furless rabbit is rarely found in the wild because the cold English winters are too harsh for the rabbits; if the rabbits cannot survive the cold, they cannot survive to reproduce.

Vocabulary to know:

Allele: One of the alternative forms of a gene. For example, if a gene determines the seed color of peas, one allele of that gene may produce green seeds and another allele produce yellow seeds. In a diploid cell there are usually two alleles of any one gene (one from each parent).

Dominant: Describes a trait that covers over, or dominates, another form of that trait. An allele that is almost always expressed, even if only one copy is present.

Recessive: describes a trait that is covered over, or dominated, by another form of that trait and seems to disappear. An allele that is expressed only when two copies are present.

Homozygous: Having identical alleles for a particular trait.

Heterozygous: Having two different alleles for a particular trait.

Genotype: The set of two genes possessed by an individual at a given spot on a chromosome.

Gene frequency: The frequency in the population of a particular gene relative to other genes. . Expressed as a proportion (between 0 and 1) or percentage (between 0 and 100 percent).

Purpose: To model the changes in gene frequency over several generations.

Materials:

50 orange beads	50 purple beads	1 paper bag
3 cups		

Procedure:

   Note: In this lab, the dominant allele for normal fur is represented by *F(purple bead)* and the recessive allele for no fur is represented by *f (orange bead).* Bunnies that inherit two *F* alleles or one *F* and one *f* allele have fur, while bunnies that inherit two fs have no fur.

1. Label one cup *FF* for the homozygous dominant genotype.
2. Label a second dish *Ff* for the heterozygous condition.
3. Label the third dish *ff* for those rabbits with the homozygous recessive genotype.
4. Place the 50 orange and 50 purple beans (alleles) in the container and shake up (mate) the rabbits.
5. Without looking at the beans, select two at a time, and record the results on the data form next to "Generation 1." For instance, if you draw one purple and one orange bead, place a mark in the chart under "Number of *Ff* individuals." Continue drawing pairs of beads and recording the results in your chart until all beans have been selected and sorted. Place the "rabbits" into the appropriate dish: *FF, Ff,* or *ff.* (Please note that the total number of individuals will be half the total number of beans because each rabbit requires two alleles.)
6. The *ff* bunnies are born furless. The cold weather kills them before they reach reproductive age, so they can't pass on their genes. Place the beads from the *ff* container aside before beginning the next round.
7. Count the *F* and *f* alleles (beads) that were placed in each of the "furred rabbit" dishes in the first round and record the number in the chart in the columns labeled "Number of *F* Alleles" and "Number of *f* Alleles." (This time you are really counting each bead, but don't count the alleles of the *ff* bunnies because they are dead.) Total the number of *F* alleles and *f* alleles for the first generation and record this number in the column labeled "Total Number of Alleles."
8. Place the alleles of the surviving rabbits (which have grown, survived and reached reproductive age) back into the container and mate them again to get the next generation.
9. Repeat steps five through nine to obtain generations two through ten.
10. Determine the gene frequency of *F* and *f* for each generation and record them in the chart in the columns labeled "Gene Frequency *F*" and "Gene Frequency *f*." To find the gene frequency of *F*, divide the number of *F* by the total, and to find the gene frequency of *f*, divide the number of *f* by the total. Express results in decimal form. The sum of the frequency of *F* and *f* should equal one for each generation.
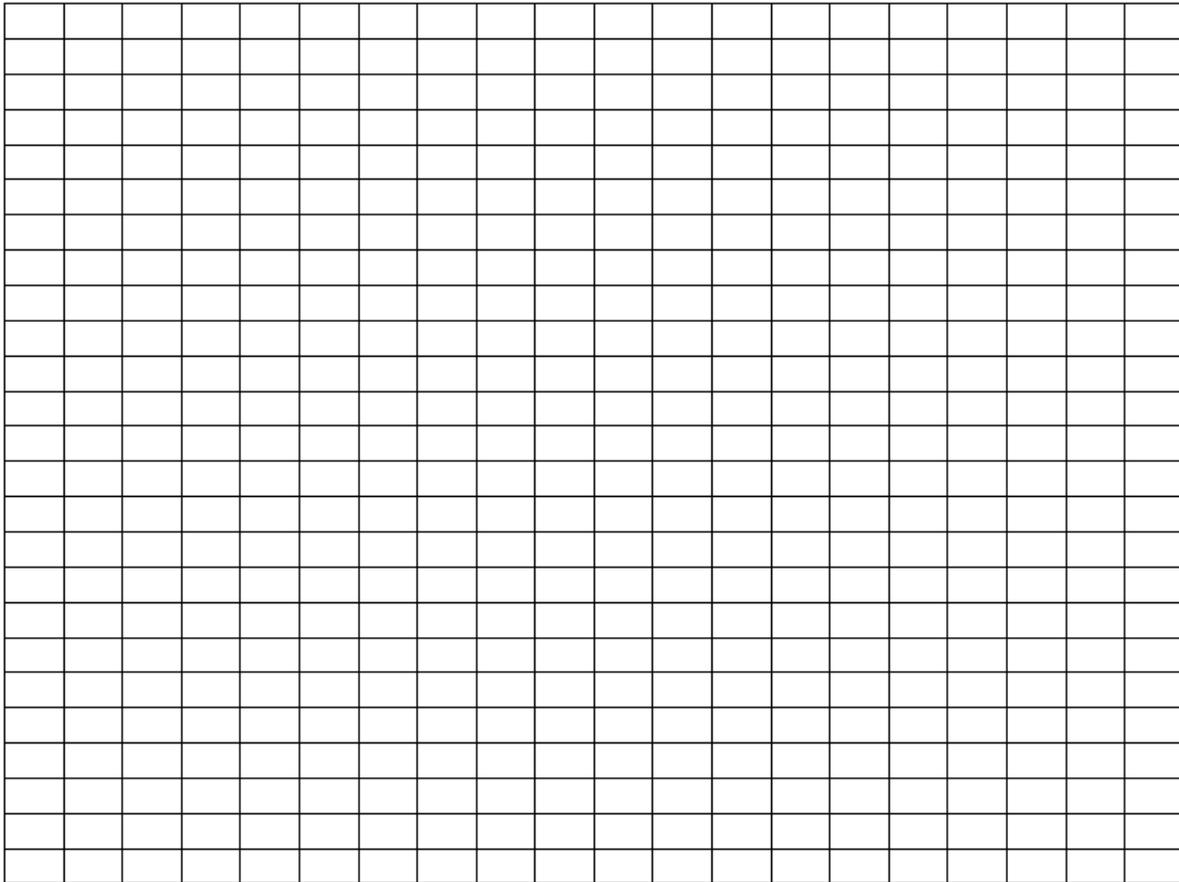
Data:

Generation	# of FF individuals	# of Ff individuals	# of ff individuals	# of F alleles	# of f alleles	Total # of alleles	Gene frequency of F	Gene frequency of f
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								

What is the best type of graph to show change? _____

What is the independent variable in this investigation? _____

What is the dependent variable in this investigation? _____

Graph your data. Remember TAILS and DRY MIX.

Conclusions:

1. Compare the number of alleles for the dominant characteristic with the number of alleles for the recessive characteristic.

2. Compare the frequencies of the dominant allele to the frequencies of the recessive allele.

3. In a real rabbit habitat new animals often come into the habitat (immigrate), and others leave the area (emigrate). How might emigration and immigration affect the gene frequency of *F* and *f* in this population of rabbits? How might you simulate this effect if you were to repeat this activity?

4. How do your results compare with the class data? If significantly different, why are they different?

5. What are some limitations of this type of model?

TEACHING-LEARNING #11   BREEDING BUNNIES: DISCUSSION QUESTIONS

1. What was your original hypothesis?

2. Based on your lab data, do you need to change your hypothesis? Explain.

3. Compare the number of alleles for the dominant characteristic with the number of alleles for the recessive characteristic.

4. Compare the frequencies of the dominant allele to the frequencies of the recessive allele.

5. In a real rabbit habitat new animals often come into the habitat (immigrate), and others leave the area (emigrate). How might emigration and immigration affect the gene frequency of F and f in this population of rabbits?

   How might you simulate this effect if you were to repeat this activity?

6. How do your results compare with the class data? If significantly different, why are they different?

7. How are the results of this simulation an example of evolution?

Breeding Bunnies Activity modified from: http://www.pbs.org/wgbh/evolution/educators/lessons/lesson4/act1.html

Gene flow from India to Australia about 4,000 years ago
Tuesday, January 14, 2013 Science Daily

4,000 years ago, Australia was no longer connected to the mainland as it had been during the ice age.

The immigrants thus crossed the ocean, arriving by boat. Credit: Gunter Senft/MPI for Psycholinguistics

Australia is thought to have remained largely isolated between its initial colonization around 40,000 years ago and the arrival of Europeans in the late 1800s. A study led by researchers of the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany, now finds evidence of substantial **gene flow** between Indian populations and Australia about 4,000 years ago. In addition, the researchers found a common origin for Australian, New Guinean and the Philippine Mamanwa populations. These populations followed an early southern **migration route** out of Africa, while other populations settled in the region only at a later date.

Australia holds some of the earliest archaeological evidence for the presence of modern humans outside Africa, with the earliest sites dated to at least 45,000 years ago, making Australian aboriginals one of the oldest continuous populations outside Africa. It is commonly assumed that following the initial dispersal of people into Sahul (joint Australia-New Guinea landmass) and until the arrival of the Europeans late in the 18th Century, there was no contact between Australia and the rest of the world.

Researcher Irina Pugach and colleagues now analysed genetic variation from across the genome from aboriginal Australians, New Guineans, island Southeast Asians, and Indians. Their findings suggest substantial gene flow from India to Australia 4,230 years ago, i.e. during the Holocene and well before European contact. "Interestingly," says Pugach, "this date also coincides with many changes in the archaeological record of Australia, which include a sudden change in plant processing and stone tool technologies, with microliths appearing for the first time, and the first appearance of the dingo in the fossil record. Since we **detect inflow of genes from India into Australia** at around the same time, **it is likely that these changes were related to this migration**."

TEACHING-LEARNING #12: ARTICLE - - *THE HUMAN JOURNEY: MIGRATION ROUTES*

taken from the National Geographic website – The Genographic Project

When humans first ventured out of Africa some 60,000 years ago, they left genetic footprints still visible today. By mapping the appearance and frequency of genetic markers in modern peoples, we create a picture of when and where ancient humans moved around the world. These great **migrations** eventually led the descendants of a small group of Africans to occupy even the farthest reaches of the Earth.

Our species is an African one: Africa is where we first evolved, and where we have spent the majority of our time on Earth. The earliest fossils of recognizably modern *Homo sapiens* appear in the fossil record at Omo Kibish in Ethiopia, around 200,000 years ago. Although earlier **fossils** may be found over the coming years, this is our best understanding of when and approximately where we originated.

According to the genetic and paleontological record, we only started to leave Africa between 60,000 and 70,000 years ago. What set this in motion is uncertain, but we think it has something to do with major climatic shifts that were happening around that time—a sudden cooling in the Earth's climate driven by the onset of one of the worst parts of the last Ice Age. This cold snap would have made life difficult for our African ancestors, and the genetic evidence points to a sharp reduction in population size around this time. In fact, the human population likely dropped to fewer than 10,000. We were holding on by a thread.

Once the climate started to improve, after 70,000 years ago, we came back from this near-extinction event. The population expanded, and some intrepid explorers ventured beyond Africa. The earliest people to colonize the Eurasian landmass likely did so across the Bab-al-Mandab Strait separating present-day Yemen from Djibouti. These early beachcombers expanded rapidly along the coast to India, and reached Southeast Asia and Australia by 50,000 years ago. The first great foray of our species beyond Africa had led us all the way across the globe.

Slightly later, a little after 50,000 years ago, a second group appears to have set out on an inland trek, leaving behind the certainties of life in the tropics to head out into the Middle East and southern Central Asia. From these base camps, they were poised to colonize the northern latitudes of Asia, Europe, and beyond.

Around 20,000 years ago a small group of these Asian hunters headed into the face of the storm, entering the East Asian Arctic during the Last Glacial Maximum. At this time the great ice sheets covering the far north had literally sucked up much of the Earth's moisture in their vast expanses of white wasteland, dropping sea levels by more than 300 feet. This exposed a land bridge that connected the Old World to the New, joining Asia to the Americas. In crossing it, the hunters had made the final great leap of the human journey. By 15,000 years ago they had penetrated the land south of the ice, and within 1,000 years they had made it all the way to the tip of South America. Some may have even made the journey by sea.

The story doesn't end there, of course. The rise of agriculture around 10,000 years ago—and the population explosion it created—has left a dramatic impact on the **human gene pool**. The rise of empires, the astounding oceangoing voyages of the Polynesians, even the extraordinary increase in global migration over the past 500 years could all leave traces in our **DNA**. There are many human journey questions waiting to be asked and answered. What stories are waiting to be told in your own DNA?

The **Genographic Project** is an ambitious attempt to answer fundamental questions about where we originated and how we came to populate the Earth. Through your participation, you can play an active role in this historic endeavor. Details of the project are found at http://genographic.nationalgeographic.com/about/

TEACHING –LEARNING #14:   SEXUAL SELECTION – Teacher Notes

1. Non-reproductive characteristics that differ between males and females of the same species are called **secondary sexual characteristics**, and are the cause of **sexual dimorphism** (differences between males and females).

Why do female peacocks, for example, prefer males with particular secondary sexual characteristics, like long tail?

2. What is sexual selection, and how does it lead to differences between males and females?

Vocabulary:
Secondary sexual characteristics - physical feature characteristic of the individual's sex; a characteristic that develops at puberty but is not directly concerned with reproduction, e.g. a woman's breasts or a man's facial hair

Sexual dimorphism- is the distinction between males and females on the basis of secondary sexual characteristics.

SEXUAL SELECTION -

- is the selection for traits that may not be adaptive to the environment but do enhance reproductive success.
- such traits may increase an individual's success in competing for or attracting mate
- intrasexual selection- individual's success in competing for mate
- intersexual selection - individual's success in attracting a mate
- intersexual selection- may be based on showy traits that reflect the general health of the male and thus fitness of his alleles.

This picture shows an example of a peacock's showy traits to attract mates.

(Fernandez) http://upload.wikimedia.org/wikipedia/commons/thumb/1/1c/Pfau_imponierend.jpg/260px-Pfau_imponierend.jpg

TEACHING-LEARNING #14: ARTICLE: *SEXUAL SELECTION*

Sexual selection is another recognized type of selection that is a potent force in nature. It involves the competition of one gender, usually the male, for the preference or favor of another, usually the female. Ultimately the goal of such competition, often in the form of attractive appearance, beneficial habits, or courtship behavior, is the "right" to reproduce with the selected mate. In one sense, sexual selection is "natural selective breeding," since it ensures that the most attractive (usually the most fit) members of the species are reproductively active. However, this is not the basis or justification of sexual selection, and it does not always act in this way.

Sexual selection's most common form is the competition among males for the favors of the females of a species. Such competition may involve the male's display of his virility, strength, desirability, etc., or may take the form of attraction by way of physical or chemical characteristics. For example, in some bird species such as bower birds, the male builds a nest or bower and the female comes to inspect it. If she "likes" the bower, she will become the male's mate; if she does not, she simply flies away, leaving the male to try again. This is a simple example of attraction by way of display - in this case, the males' display of their ability to design a nest to care for future young. An example of the chemical method is the release of pheromones, common in insect species.

Sexual selection obviously affects the frequency of alleles in a given gene pool. If females consistently choose males with certain characteristics, then the genes for these characteristics will become more common, because these males will reproduce more often. Also, the genes that caused the females to choose the way they did in the first place will become more frequent, since they will outnumber their counterparts to begin with and will pass along their genes. If this process achieves total or near-total saturation of the gene pool with one allele, selection usually focuses on another characteristic.

Sexual selection often leads to an increase in the overall fitness of the members of a species, since the females' criterion is often strength, fitness, or some suitable display thereof. However, the preference for a display of strength can also cause sexual selection to offset or counterbalance the force of pure natural selection. For example, suppose that a species of bird exists in which females prefer feathers on the crest of the head to heads without feathers. The crested males will come to out-reproduce their uncrested counterparts, and crests will typically become the norm over many generations. However, the females' preference may continue to tend toward more and more elaborate feathers - perhaps the ability to maintain attractive feathers is a good proxy measure for a bird's overall health. Suppose further that overly elaborate feathers conferred a disadvantage in survival because the feathers impeded the bird's sight, or tended to attract fleas, or any other disadvantage. The opposing forces of natural and sexual selection generally eventually find a "happy medium" between the large feathers preferred by females and the smaller feathers that confer a survival advantage.

- **Notes: Definition of Evolution**

How often have you heard executives report that "the idea evolved into a successful project" or popular science show narrators describe how a star "has been evolving for millions of years"? *Evolution* is no longer strictly a biological term since every academic field and nonacademic industry uses it. Such uses of the verb "evolve" reveal its meaning in its simplest form—to evolve means to change. You should remember the biological definition of evolution: *descent with modification*. Don't let the general uses of the word mislead you; a key part of this definition is *descent*, which can happen only when one group of organisms gives rise to another. When you see the word evolution, think of something that happens in populations, not in individuals.

- More specifically, evolution describes change in allele frequencies in populations over time. When one generation of organisms (whether algae or giraffes or ferns) reproduces and creates the next, the frequencies of the alleles for the various genes represented in the population may be different from what they were in the parent generation. Frequencies can change so much that certain alleles are lost or others become fixed—all individuals have the same allele for that character. Over many

generations, the species can change so much that it becomes quite different from the ancestral species, or a part of the population can branch off and become a new species (**speciation**). Why do we see this change in allele frequencies with time?

- Allele frequencies may change because of random factors or by natural selection. Let's consider chance events first. Imagine a population of fish in a large pond that exhibits two alleles for fin length (short and long) and is isolated from other populations of the same species. One day a tornado kills 50 percent of the fish population. Completely by chance, most of the fish killed possess the long-fin allele, very few of these individuals are left in the population. In the next generation, there are many fewer fish with long fins because fewer long finned fish were left to reproduce; that allele is much more poorly represented in the pond than it was in the original parent generation before the catastrophe. This is an example of **genetic drift**: a change in allele frequencies that is due to chance events. When drift dramatically reduces population size, we call it a **bottleneck**.

- Now imagine that the same pond becomes connected to another pond by a small stream. The two populations mix, and by chance, all the long-finned fish migrate to the other pond, and no long-finned fish migrate in. Again, which individuals migrated was random in this example; thus, there will be a change in the allele frequencies in the next generation. This is an example of **gene flow**, or the change in alleles frequencies as genes from one population are incorporated into another.

- Gene flow (also more loosely known as *migration* when the individuals are actively relocating) is random with respect to which organisms succeed, but keep in mind that we could think of situations in which migration is not random. For example, if only the short-finned fish could fit in the stream connecting the two ponds, the alleles represented in the subsequent generation would not be random with respect to that allele. We also have not stated that the short-finned fish have an advantage by swimming to the other pond—if they did, this would be an example of natural selection, which we'll discuss below.

- Finally, let's consider **mutation**, the third random event that can cause changes in allele frequencies. Mutation is *always* random with respect to which genes are affected, although the changes in allele frequencies that occur as a result of the mutation may not be. Let's say that a mutation occurs in the offspring of a fish in our hypothetical pond. The mutation creates a new allele. As a result, the allele frequencies in the offspring generation has changed, simply because we have added a new allele (remember that allele frequencies for a given gene always add up to one). As you can imagine, one mutation on its own does not have the potential to dramatically alter the allele frequencies in a population, unless this is a *really* small pond! But mutation is extremely important because it is the basis of the variation we see in the first place and it is a very strong force when it is paired with natural selection.

The four major modes of evolution are

1. Genetic drift
2. Gene flow (also called *migration*)
3. Mutation
4. Natural selection.

KEY IDEA

- Remember that the first three factors act randomly with respect to the alleles in the population—which alleles increase and which decrease in frequency are determined by chance events, not because some alleles are inherently better than others are. We'll now turn to the fourth mode or process of evolution, natural selection, where the modification that occurs with descent is *non*random.

Reading Standards for Literacy in Science, 6–12

Grades 10-12 - - RST 2 and RST 4

Grade 9-10 Students
2. Determine the central ideas or conclusions of a text; trace the text's explanation or depiction of a complex process, phenomenon, or concept; provide an accurate summary of the text.

Sample Activities - - Basic Analysis

Teacher uses familiar [fairly simple] texts to model **gr. 6-12** (a) identify central ideas (b) identify conclusions (c) summarize the text (distinct from prior knowledge or opinions) **gr. 9-10** (d) trace the complex process or concept through the text **gr 11, 12** (e) summarize the processes and ideas of a text by paraphrasing them in simpler (but accurate) terms Teacher places results in an analysis template (attached)	Students replicate the analysis with a NEW [fairly simple] document **gr. 6-12** (a) identify central ideas (b) identify conclusions (c) summarize the text (distinct from prior knowledge or opinions) **gr. 9-10** (d) trace the complex process or concept through the text **gr 11, 12** (e) summarize the processes and ideas of a text by paraphrasing them in simpler (but accurate) terms Students place results in an analysis template (attached)

Student _____ **Class** _____ **Date** _____

Texts: Primary _____ **Secondary** _____

Science and Technical Text

Grades 6-12	Grades 9,10
(a) central ideas _____ **(b) conclusions** _____ _____ _____ _____ **(c) summarize the text** (distinct from prior knowledge or opinions) _____ _____ _____ _____ _____ _____	**Use a highlighter and pen to "trace" the process / concept right on the copy of the text.**

Grade 9-10 Students
4. Determine the meaning of symbols, key terms, and other domain-specific words and phrases as they are used in a specific scientific or technical context relevant to *grades 9– 10 texts and topics*.

Sample Activities - - Symbols, Key Terms, Domain-specific Words	
Teacher uses familiar [fairly simple] texts to model - -	Students replicate the analysis with a NEW [fairly simple] document
symbols	symbols
key terms	key terms
domain-specific words	domain-specific words