

Bio Final Review

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1 General Information

For this, see the google doc.

2 Genetics

2.1 Sex-linked genes

These are genes located on the sex chromosomes. They will show different phenotype frequencies based on gender.

- (1) Gene A is on the X chromosome. A is the wild type, and α is the diseased type.
 $X^A X^\alpha \times X^A Y$:

$$\begin{array}{c|c} X^A Y & X^\alpha Y \\ \hline X^A X^A & X^\alpha X^A \end{array}$$

2.2 Pedigrees

- = Unaffected Female
- = Affected Female
- = Unaffected Male
- = Affected Male

Connecting lines on pedigrees work just as they do on family trees. Relatively simple logic can be used to determine the genotypes of each member of the pedigree; however, some can be more difficult than others. My general method is to use the “method of staring” in the words of Mr. Letarte.

2.3 Genetic Disorders – Sickle Cell, Cystic Fibrosis, Huntington’s

Sickle Cell

- Red blood cells contain hemoglobin, which bind O_2
- Hemoglobin is made up of two α -globin and two β -globin polypeptides
- Mutation in β -globin makes it slightly less soluble
- When O_2 is low, hemoglobin without O_2 will start to clump and form long fibers that will change the shape of the red blood cell, which will then get stuck in capillaries
- If one is a heterozygote of this disease, they have an advantage against malarial
- Sickle Cell disease is recessive, because its effects are not great enough with only some of the β -globin broken.

Cystic Fibrosis

- In frame three base pair deletion in gene for CFTR
- CFTR is missing one amino acid (phenylalanine), which causes it to misfold and be destroyed
- CFTR is a channel in the epithelial cell membranes for Cl^-

- Without CFTR, there is too much extracellular Cl^- , which makes the fluid outside the cell thicker
- Mucus clogs lungs and serves as a growth substance for *Pseudomonas aeruginosa*
- The allele for Cystic Fibrosis is recessive, as with one of the two CFTR, cells still have enough paths for Cl^-

Huntington's Disease

- Mutation is dominant, but the disease does not present itself until late 30's or early 40's
- Huntingtin gene expressed in nerve cells. Its developmental role in adults is unclear
- CAG (codes for glutamine)
- Wild type 6-35 repeats
- Diseased 36+ repeats
- Diseased protein forms aggregates in neurons, which lead to cell death.

2.4 Nondisjunction

Nondisjunction Event – Failure to separate chromosomes

This is more common in meiosis I. Trisomy 21 causes Down syndrome.

2.5 Recombinant DNA – Restriction Enzymes, Ligase, Electrophoresis, GFP, PCR, Selectable Markers, Screens, Plasmids, Transformations

Recombinant DNA – Combination of two or more pieces of DNA to create an artificial construct.

Building Pieces of DNA:

1. Synthesize from scratch
2. Cut and Paste
 - Ligase is not specific and will join any two pieces of DNA.
 - Restriction enzymes originate from bacteria, where they served as a type of immune system.
 - Restriction System: methylase adds CH_3
 - Restriction Enzyme cuts DNA if not regulated.

Eco RI:

- Sticky ends of *GAATTC* (a DNA reverse palindrome)
- Eco RI cuts between the G and the A

Plasmid:

- Mini chromosome in bacteria
- Must have an ori, a selectable marker
- Plasmids can be shared between cells. They also can be picked up from the environment, when they are there for whatever reason.
- An example of plasmids which can be shared between cells is antibiotic resistance.

DNA Sequencing:

- Denature DNA into single strands
- Add primer for only one strand
- Provide DNA polymerase and the four nucleotides, with a small fraction of the nucleotides modified so that DNA polymerase cannot extend from them (remove 3' hydroxyl)

2.6 Selective Breeding – Hybridization vs Inbreeding

Selective breeding – only allowing parents with certain characteristics to breed.

Hybridization:

- Crossing two organisms (typically plants) to get the best traits from both in a hybrid
- Can be different species.

Inbreeding:

- Continued breeding of individuals with similar characteristics
- Dramatically decreases genetic variation
- Increases prominence of some traits

3 Evolution

3.1 Darwin's Observations and Background Knowledge

3.2 Evolutionary Theory – Adaptations, Fitness, Natural Selection, etc.

3.3 Evidence for Evolution

3.4 Population Genetics – Hardy-Weinberg Equation and Conditions that disrupt equilibrium, selection on polygenic traits

3.5 Speciation

3.6 Molecular Clocks

3.7 Binomial Nomenclature and Linnaean Classification

3.8 Cladograms

3.9 Domains and Kingdoms

3.10 Fossil Record

3.11 Speciation and Extinction Events

3.12 Origins of Life including Endosymbiont Theory

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