

BIOL 1005 – Concepts in Biology

Outline of topics covered for Midterm II (October 18, 2018) – *final version! posted October 16, 2018*

DISCLAIMER: This outline is meant to help you organize your lecture notes. It is not intended to be a substitute for your lecture notes! Furthermore, it is NOT EXHAUSTIVE. Just because a word or phrase does not appear on this study guide, doesn't mean you "don't have to know it." In general, you are best off studying your lecture notes and letting this outline serve as a guide to help you get your notes organized.

Overriding topic for this portion of class: what makes living things different from each other?

I. Protein synthesis

- A. Genes, proteins, and chromosomes
 1. What's the relationship between chromosomes, DNA, and proteins? What is a gene?
 2. Why are proteins important? What are they made of?
- B. DNA & RNA structure – a review
 1. Structure of the DNA double-helix: What are the four nitrogenous bases in DNA? What is complementary base-pairing? Which nucleotide forms base-pairs with which? How do the complementary base pairs hold onto each other?
 2. Structure of RNA: How is the function of RNA different from that of DNA? What are the four bases in RNA? Which nucleotide forms base-pairs with which?
- C. Two main events of protein production (see the Protein Synthesis Man handout, which uses a portion of the *CFTR* gene as an example; see also the Transcription and Translation handout)
 1. Transcription:
 - a. Where in the cell does transcription occur?
 - b. Describe the roles of DNA, RNA polymerase, mRNA, promoter, and terminator.
 2. Translation:
 - a. Where in the cell does translation occur?
 - b. Know how to use the dictionary of the genetic code! (You do not have to memorize it.)
 - c. Describe the roles of mRNA, codon, ribosome, tRNA, amino acids, and anticodon.
- D. Regulation of protein production
 1. Does every cell express every gene that it contains? Why or why not?
 2. If cells are genetically identical, how do they acquire specialized structures and functions?
 3. How does a cell control which genes to transcribe, and when? (Think epigenetics and transcription factors – what are they?)
- E. Medical applications for understanding protein synthesis: bacterial production of human proteins (why would anyone want to do that?); designing antibiotics that kill bacteria without killing us
- F. Mutations
 1. What is a mutation?
 2. How might each of the following types of mutations affect a gene (and its encoded protein), both at the site of the mutation and “downstream” from the mutation?
 - a. Deletion of one or more bases
 - b. Insertion of one or more bases
 - c. Substitution of one base
 3. How do the specific mutations associated with cystic fibrosis (deletion) and sickle cell trait (substitution) cause illness?
 4. What is the relationship between genes, mutations, and alleles?
 5. Give examples for each of the reasons that genetic mutations are important.
 6. Where might the mutations in your own DNA have come from?

II. Viruses

- A. What are the parts of a basic, no-frills virus?
- B. Similarities and differences between viruses and cells
- C. List the five stages in every viral replication cycle
 - a. Which stage is most important in determining whether a virus can infect a particular cell type?

- b. During which stage does all of that transcription and translation stuff happen? Why is that important to the virus and to the host cell?
- c. During which stage does the host cell typically (but not always) die? Why?
- D. Why do viral infections produce the symptoms they do?
- E. Prevention and treatment of viral infections
 - a. Why antibiotics don't work against viruses; why viral diseases in general are hard to cure
 - b. How vaccines work (in general)
 - c. Examples of viruses for which we do and don't have vaccines:
 - i. Cold viruses (RNA): no vaccine. Why not?
 - ii. Influenza (RNA): annual vaccine. Why do we need a new shot every year, and why might it work better some years than others?
 - iii. Genital warts/cervical cancer (DNA): one-time vaccine series. Why does immunity last so much longer than for influenza vaccine? Why is it best to receive the vaccine before reaching adulthood?
 - iv. HIV (RNA retrovirus): we have drugs that slow viral replication (e.g., AZT and protease inhibitors) but no vaccine. Why not?

III. Reproduction and inheritance

- A. Why do cells divide?
 - a. Reproduction
 - 1. Similarities and differences between asexual and sexual reproduction
 - 2. What are the roles of binary fission, mitosis, and meiosis in asexual and sexual reproduction?
 - 3. Advantages of asexual reproduction; advantages of sexual reproduction
 - 4. The importance of genetic variability in a changing environment
 - b. Growth and development
 - c. Repair of damage to the body
- B. Events of DNA replication
 - a. What does DNA polymerase do?
 - b. How does the error rate of DNA polymerase compare to that of RNA polymerase? How does this difference relate to last week's material on viral evolution and vaccines?
- C. Binary fission (prokaryotes only): function of binary fission; events in binary fission, including how the duplicate chromosomes separate
- D. Mitosis (eukaryotes only)
 - 1. Eukaryotic chromosome structure: chromosome, chromatid, and centromere
 - 2. Main events in the cell cycle: interphase → mitosis → cytokinesis → repeat ...
 - a. Interphase: what happens? Is DNA coiled into visible chromosomes or unwound? Why does it matter?
 - b. Know general events in, and be able to recognize, the stages in mitosis: prophase, metaphase, anaphase, telophase/cytokinesis [*the names of the stages are also covered in week 8's genetics lab*]
 - c. Why both cell division and controlled cell death ("apoptosis") are required for development
 - 3. Cancer as a disorder of mitotic cell division
 - a. Why tumors are harmful; difference between a benign and a malignant tumor; how metastasis occurs; the general meaning of a cancer's "stage"
 - b. How proteins control cell division; what happens when those proteins "break" or are expressed at the wrong time/place
 - c. Trace the sequence of events from mutation to cell cycle control proteins to cancer. How do the mutations occur? Is it possible for a person to reduce the risk of cancer to zero?
 - d. Why cancer is hard to treat/cure; how current cancer treatments work
- E. Meiosis (eukaryotes only)
 - 1. More on eukaryotic chromosomes
 - a. Difference between haploid and diploid cells
 - b. Homologous chromosomes (how do you know if chromosomes are homologous?)
 - c. Which chromosomes determine sex?

2. Does meiosis occur in somatic cells, germ cells, or gametes? What are the differences between those cell types? What two characteristics of gametes make them different from your body's somatic cells?
3. Know the general events in, and be able to recognize, the stages of meiosis [*note that we skipped this in lecture, but was covered in detail in week 8's genetics lab*]:
 - a. Interphase and DNA replication precede meiosis
 - b. Meiosis I: prophase I, metaphase I, anaphase I, telophase I and cytokinesis
 - c. Meiosis II: prophase II, metaphase II, anaphase II, telophase II and cytokinesis
4. What are two reasons that two people can theoretically create more than *~70 trillion* genetically different offspring?
5. Difference between identical and fraternal twins; what are conjoined twins?
6. What can go wrong in meiosis [*covered out of order on 10/16*]
 - a. Unequal crossing over: certain chromatids have missing or duplicated portions
 - b. Nondisjunction: entire chromosomes are missing or present in extra copies

V. Patterns of inheritance (see the Inheritance Man handout)

- A. Basic genetics terms: homologous chromosomes, alleles, phenotype, genotype, homozygous, heterozygous
- B. What's the relationship between the events of meiosis and a Punnett square?
- C. Inheritance and protein function
 - i. Generally speaking, what makes an allele "dominant" or "recessive"?
 - ii. Does "dominant" automatically mean "most common"?
 - iii. Is a heterozygous person more likely to pass on the dominant alleles than the recessive one? Use the events of meiosis to explain your answer.
- D. Beyond the basics: How patterns of inheritance can be more complicated than what we've seen so far; be able to use Punnett squares to predict inheritance for any gene on any chromosome
 1. X-linked traits (Why do males usually have X-linked recessive diseases more often than females? What is the role of X chromosome inactivation in calico/tortoiseshell cats? In Rett syndrome?)
 2. Codominance (ABO blood typing – why is this an example, and how does it work?)
 3. Incomplete dominance (eye color – why is this an example?)
 4. Polygenic traits (what's are some examples?)
 5. Environmental effects on gene expression (what's an example?); be able to connect this idea back to epigenetics.

VI. DNA technology

- A. How DNA profiling works
- B. How gene therapy works
- C. How genetically modified (transgenic) bacteria and plants are produced

Action Centers are always Mondays, 3:30-5:30, Housing Learning Center in Adams dorm.
Come and go as you please.

Use all of the resources on my website, where you'll find handouts, practice questions, old exams, useful videos, and more.

Also, check tutor.ou.edu for tutoring hours with Nazha, and remember that my office hours are Tues./Thurs. 8:30-10:00 am and Wed. 2:00-3:00 pm.