Biology 6 Test 3 Study Guide

Chapter 10 – Classification

A. Classification Systems
   a. Taxonomy – classification of organisms by physical traits.
      i. Hierarchy gives idea of relationship (Fig. 10.5)
      iii. Dichotomous keys use traits for identification. (Fig. 10.8)
   b. Systematics – uses phylogeny (evolutionary history). Also uses more modern technology.
      i. Lines on phylogenic trees (cladograms) represent evolutionary distance (time).
      ii. Branch points represent divergence.
   c. History of the classification
      i. 1735 Carolus Linnaeus introduce Plant and Animal Kingdoms
      ii. 1969 Robert Whittaker founded 5 Kingdom System.
         1. 5 Kingdoms: Monera, Protista, Fungi, Plantae, Animalia
      iii. 1990 Carl Woese found the Domain System (Fig. 10.1)
         1. 3 Domains: Bacteria, Archaea, Eukarya (Tab. 10.1)
      iv. 2000 – present Ford Doolittle introduces the “shrub of life”
         1. Evolution is not always linear. There is lateral transfer. E.g. endosymbiosis, gene transfer.

B. Methods of Classification
   a. Morphology – gross shape/size
      i. Colonies – color, texture, size (e.g. S. aureus gives smooth, round, yellow colonies)
      ii. Cells – size, shape, motility (e.g. S. aureus are small staphylococcus, non-motile)
   b. Metabolism – test for waste products and ingredient usage
      i. Uses differential and selective media.
      ii. Enterotube and API are multiple tests in one package (Fig. 10.9)
   c. Serology – use of antibodies (will cover later)
   d. Phage typing – use viruses to infect bacteria to identify (Fig. 10.13)
   e. Molecular Biology
      i. Sequencing – determines composition and order of DNA, RNA, or protein
      ii. Hybridization – when two strands of nucleic acid match more closely, the better the base pairing (Fig. 10.15)
      iii. DNA chip – contains DNA from known organisms spotted on a glass plate (chip). Unknown sample of DNA will hybridize only to known spots. (Fig. 10.17)
      iv. Gel electrophoresis – fingerprinting (Fig. 10.14)

Chapter 13 – Viruses et al.

A. General characteristics
   a. Definition
      i. Acellular particle
      ii. Uses host cell for reproduction
         1. Replication and gene expression comes from host cell
         2. Specificity
a. Viruses may be very specific for different hosts or have a broad range of hosts.
b. Viruses may have cell specificity (e.g. HIV infects only certain immune cells in humans)

b. Structure
   i. Components (Fig. 13.3)
      1. Nucleic acid – can be single or double stranded RNA or DNA
      2. Capsid – protein coat. Made from capsomere subunits
      3. Optional components
         a. Some have envelopes – uses host membrane with virus proteins (spikes) embedded. These spikes are used for attachment or can be enzymes. (Fig. 13.3)
         b. Complex components – bacteriophages have other structures for injection of DNA (Fig. 13.5)
   ii. Size – varied, but in nanometers. (Fig. 13.1)
   iii. Shape – varied. Some helical, round, polyhedral, long.

d. Classification – based on type of nucleic acid, components, hosts, and shape (Tab 13.2)
   i. Nucleic acids: double/single stranded RNA, DNA
   ii. Single stranded RNA
      1. Sense (+): can be directly translated
      2. Antisense (-): need to make the complement before translation

B. Cultivation
   a. Non-animal viruses – phages grow on bacterial lawns (Fig. 13.6)
   b. Animal viruses
      i. Use whole animals – mice are widely used.
      ii. Eggs – a fertilized (embryonated) egg can be injected (Fig. 13.7)
      iii. Cell culture – easiest and most efficient. Use variety of cell lines. Many cell types show a visible difference called a cytopathic effect (Fig. 13.9)

C. Life Cycles
   a. General stages of virus life cycles
      i. Attachment (adsorption) – virus attaches to host cell by specific binding
      ii. Penetration – genome enters the cell
      iii. Synthesis – replication and gene expression of viral components for next generation
      iv. Maturation – processing and assembly of viral components
      v. Release – exit of newborn viruses from cell
   b. Bacteriophages
      i. Lytic – phage makes particles and kills host (Fig. 13.11)
         1. Attachment – uses tail fibers to attach to cell.
         2. Penetration – DNA is injected into cell
         3. Synthesis – replication, transcription, and translation
         4. Maturation – components assembled
         5. Release – lyses the cell.
      ii. Lysogenic – some bacteriophages can alternate between lytic and lysogenic (latent) cycles (Fig. 13.12)
         1. Integration – DNA is integrated into host DNA and can be carried on indefinitely as a prophage.
         2. Excision – under bacterial stress, virus may re-enter lytic cycle by first excising the DNA and continuing with the synthesis stage.
   c. Animal viruses
i. DNA viruses – stages similar to general life cycle. Uncoating is breakdown of capsid after penetration. DNA needs to enter nucleus (Fig. 13.15)

ii. RNA viruses (Fig. 13.17)
   1. ssRNA viruses – RNA has to be transcribed into complementary strand for replication. Only the sense (+) strand can be translated into viral proteins.
   2. Retroviruses – ssRNA is reverse-transcribed into DNA, than made double-stranded. dsDNA integrates into chromosome and directs transcription as a prophage. Exits by budding (13.19)

D. Viruses and Cancer
a. Cancer is uncontrolled cell division and invasive growth. (13.8)
   b. Integrative viruses can land next to a gene and cause it to over or underexpress.
      i. Overexpressed genes that cause cancer are called oncogenes. The normal proto-oncogene is usually involved in activating cell division
      ii. Underexpressed ones are tumor suppressors. These are normally inhibitors of cell division.
   c. Types of cancer causing viruses
      i. DNA – e.g. HPV (human papilloma virus) gives cervical cancer
      ii. Retroviruses – e.g. HTLV (human T cell leukemia virus) causes leukemia.

E. Virus-like Particles
a. Viroids – “naked RNA”
   i. Found in plants and causes deformation, lesions, stunted growth. (Fig. 13.23)
   ii. Circular ssRNA, 200-400 bp long.
   iii. Replicates in nucleus using host machinery.
   iv. Viroids bind plant mRNA preventing translation. May also bind up plant proteins.
   b. Prions – “proteins gone wild”
      i. Causes “mad cow disease” and Creutzfeldt-Jakob (CJD). Neurological degeneration (dementia, loss of motor control, wasting). Post mortem plaques and lesions in brain found.
      ii. Protein alone is infectious agent. PrP<sup>C</sup> is a normal cell-surface protein involved in neuronal function. It can normally be destroyed when not needed by proteases.
      iii. PrP<sup>Sc</sup> is a rare conformational form of PrP<sup>C</sup> that is resistant to proteases. It also converts normal PrP<sup>C</sup> into PrP<sup>Sc</sup>. (Fig. 13.22)
      iv. Buildup of PrP<sup>Sc</sup> causes cell death and plaques forming in the brain.

Chapter 11 – Prokaryotes

A. Classification
a. Problems
   i. Too many unidentified species
   ii. Too little known about identified species
   iii. Lateral transfer makes phylogenic history difficult to discover
b. American Type Culture Collection (ATCC) – collects organisms, collects information, and distributes organisms.
c. Bergey’s Manuals
   i. Internationally recognized for classification of bacteria
   ii. Undergoes dramatic changes from one edition to the next

B. Bacteria
a. Proteobacteria
   i. Alpha – can grow in low nutrient conditions
1. *Rickettsia* – coccobacillus that is an obligate parasite of mammals. Transmitted by ticks. Causes fever and rashes (Fig. 11.1)

ii. Beta – can grow on gaseous byproducts

1. *Neisseria* – diplococcus that inhibits mucous membranes. Pathogenic species cause gonorrhea and meningococcal meningitis (Fig. 11.6)

iii. Gamma – largest and most diverse class

1. Pseudomonadales
   a. *Pseudomonas* – bacillus, motile, aerobic. Some species cause infections, wounds, burns. Found in hospitals. (Fig. 11.7)

2. Enterics
   a. *Escherichia* – bacillus found in gut. Used in research. Some strains can cause diarrhea and urinary tract infections (UTI).

iv. Delta – prey on other bacteria

1. *Myxococcus* – bacillus that moves by gliding. Will lyse and digest other bacteria. Produces spores under low nutrient conditions (Fig. 11.11)

v. Epsilon – twisted shape

1. *Helicobacter* – vibrio with multiple flagella. Some species can survive stomach acid and cause peptic ulcers and stomach cancer. (Fig. 11.12)

b. Nonproteo Gram- - photosynthetic

i. Cyanobacteria – oxygenic photosynthesis (produce oxygen)

1. *Anabaena* – pleiomorphic, colonial, filamentous. Has special cell type called heterocysts to fix nitrogen (Fig. 11.13a)

ii. Purple – anoxygenic photosynthesis

1. *Chromatium* – pleiomorphic. Electron acceptor is hydrogen sulfide instead of water. Sulfur accumulates then stored in granules which give off color. (Fig. 11.14)

c. Gram + Low G+C (Firmicutes)

i. *Bacillus* – the anthrax species produces tough endospores. Can be used in biological warfare.

ii. *Staphylococcus* – common cause of food poisoning. Survives in low moisture, high osmotic conditions. Found on mucous membranes and skin and causes infections during surgery due to breakage of skin. (Fig. 11.18)

d. Gram + High G+C (Actinobacteria)

i. *Mycobacterium* – bacillus with filamentous growth. Have mycolic acids replacing LPS which makes them resistant to drying out and antimicrobial drugs. Some species cause tuberculosis and leprosy. (Fig. 11.20)

e. Other

i. *Chlamydiae* – Coccus causing chlamydial infections. Transmitted through sex. Life cycle includes infection by elementary bodies, conversion to reticulate bodies, division, and conversion back to elementary bodies (Fig. 11.23)

C. Archaea

a. Halophiles – like high salt. E.g. *Halobacterium*

b. Thermophiles – like high heat. E.g. *Pyrodictium*

c. Methanogens – produce methane from hydrogen gas and carbon dioxide. E.g. *Methanobacterium*

Chapter 12 – Eukaryotes

A. Parasitology

a. Types of hosts

i. Intermediate host – harbors asexual (juvenile) stage
ii. Definitive host – harbors sexual (adult) stage
b. Some ways parasites can evade the immune system
   i. Encystment – formation of an outer shell that is resistant to environment. Much like a spore
   ii. High mutation rate of surface antigens
   iii. Antigen decoys – molecules released to elicit immune response. These are not present on the parasite and are meant to distract immune system.
   iv. Hide inside of cells

B. Protists
   a. Algae – “plant-like”
      i. General characteristics
         1. Photosynthesis
         2. Asexual and sexual
         3. Must be in water
      ii. E.g. Dinoflagellates (Fig. 12.13)
         1. Have flagella and very hard cell wall plates.
         2. Comprise plankton but can be dangerous in high numbers (red tide)
         3. Many produce neurotoxins that concentrate in fish and shellfish.

   b. Protozoa – “animal-like”
      i. General characteristics
         1. Chemoheterotrophs
         2. Mostly asexual, some sexual
      ii. Archaezoans
         1. Have flagella
         2. Most lack mitochondria and are digestive tract symbionts.
         3. E.g. *Giardia* can infect the intestines and cause “backpacker’s diarrhea” (Fig. 12.16)
      iii. Amoebas
         1. Have amorphic shape and move by pseudopods.
         2. E.g. *Entamoeba* causes amoebic dysentery. (Fig. 12.17)
      iv. Apicomplexans
         1. Immobile
         2. Enzymes at apex of cell are used to digest host membranes for entry.
         3. E.g. *Plasmodium* (causes malaria) life cycle (Fig. 12.18)
            b. Sporozoites produce merozoites that reproduce in red blood cells.
            c. Merozoites produce gametocytes that get picked up by mosquito.
            d. Male and female gametocytes fuse to form sporozoites in mosquito.
      v. Ciliates – have cilia. E.g. *Paramecium* (not a parasite) (Fig. 12.19)
   c. Slime Molds – “fungal-like” and amoeboid
      i. General characteristics
         1. Chemoheterotrophic, absorbs food.
         2. Asexual and sexual
         3. Movements similar to amoeba
      ii. Cellular slime mold (Fig. 12.21)
         1. Amoeboids congregate upon release of cAMP signal.
         2. Slug (pseudoplasmodium) is formed and fruiting body is produced.
         3. Spores released (asexual)
      iii. Plasmodial slime molds (Fig. 12.22)
1. Zygote will form a multinucleate mass called a plasmodium.
2. Cytoplasmic streaming allows for movement.
3. Sexual spores are released producing gametes.

C. Fungi
   a. General features
      ii. Body is called mycelium composed of threadlike hyphae. Reproductive portion is
           fruiting body. Yeasts are unicellular.
      iii. Reproduce asexually and sexually.
           1. Spore formation can be asexual or sexual.
           2. Fertilization can occur in two steps: plasmogamy is when the cells fuse
              without fusion of nuclei. Karyogamy is when nuclei fuse.
      iv. Many cause opportunistic infections
   b. Zygomycete (bread mold) life cycle (Fig. 12.6)
      i. E.g. Rhizopus.
      ii. Asexual: haploid hyphae produce sporangium that release haploid spores.
      iii. Sexual: haploids of opposite mating type can fuse to form zygospore (diploid).
           Zygospore undergoes meiosis and produces sexual spores.
   c. Ascomycete (sac fungus) life cycle (Fig. 11.7)
      i. E.g. Candida (causes “yeast infections”) 
      ii. Asexual: haploid hyphae produce conidia that release haploid spores.
      iii. Sexual: mating produces an ascus that releases sexual ascospores.
   d. Basidiomycete (club fungus) life cycle (Fig. 11.8)
      i. E.g. Agaricus (button mushroom)
      ii. Asexual: no spores produced, just vegetative growth.
      iii. Sexual: mating produces fruiting body (mushroom) that contains basidiospores.

D. Animals
   a. Flatworms (Platyhelminths)
      i. Flukes (Trematodes) – life cycle (Fig. 12.25)
         2. Larvae attach to snails (intermediate).
         4. Adults mature in human
      ii. Tapeworms (Cestodes) (Fig. 12.26, 12.27)
         1. Structures: scolex is head. Proglottids are rest of segments (sexual and
            produce eggs)
         2. Life cycle:
            a. Eggs released from intestine of definitive host
            b. Intermediate host eats eggs, eggs hatch and encyst.
            c. Definitive host eats cysts, the uncyst and scolex attaches to
               intestine and forms proglottids.
   b. Roundworms (Nematodes)
      i. E.g. Ascaris life cycle (dimorphic – separate males and females)
         1. Eggs eaten
         2. Larvae hatch in small intestine and burrow into the bloodstream and
            carried to lungs.
         3. Juveniles develop in lung, get coughed up and swallowed.
         4. Adults mature and mate in the small intestine.
      ii. E.g. Trichonella life cycle
         1. Encysted larvae eaten (raw pork)
         2. Adults develop in small intestine, reproduce.
3. Eggs hatch and larvae enter bloodstream/lymph and spread throughout body. Larvae encyst in muscles.

c. Arthropods – common vectors (12.30-12.32)
   i. Arachnids
      1. Two body regions – cephalothorax and abdomen
      2. Eight legs
      3. E.g. spiders, ticks. Ticks can spread Rickettsia
   ii. Insects
      1. Three body regions – head, thorax, and abdomen
      2. Six legs
      3. E.g. Mosquitoes spread Plasmodium, Tsetse fly spreads Trypanosoma
   iii. Crustaceans
      1. Aquatic with shells
      2. E.g. Crayfish, crabs, lobsters may carry encysted flukes.

Chapter 7 and 20 – Antimicrobial Agents

A. Testing Antimicrobial Agents for Effectiveness
   a. Testing Specific Agents
      i. Disk Diffusion Method (Fig. 7.6, 20.17)
         1. Plate a lawn of bacteria
         2. Soak paper disks with agent and look for zones of inhibition on plate
         3. Can estimate minimum inhibitory concentration (MIC) of agent (Fig. 20.18).
      ii. Dilution Method (Fig. 20.19)
         1. Make dilutions of agent and find lowest concentration that kills culture.
         2. More quantitative than disk method because of known concentrations in each well. Can calculate MIC.

B. Nonspecific Agents
   a. Physical Agents
      i. Temperature and Drying
         1. Heat
            a. Dry heat can reach hotter temperatures and is necessary when moisture cannot be used (sterilizing equipment that could rust)
            b. Moist heat is more effective because it is more penetrating. The autoclave is a moist heat sterilizer. Uses high pressure to help increase temperature (Fig. 7.2)
            c. Pasteurization – heat at a lower temperature to prevent proteins from denaturing, but kills most microbes (e.g. milk)
         2. Cold
            a. Freezing, refrigeration
            b. Freeze-drying – freeze quickly in dry ice/nitrogen under a vacuum to remove water.
      ii. Filtration – trap organisms on a filter (Fig. 7.4)
      iii. Osmotic Pressure – high solute (e.g. salt) will plasmolyze most organisms.
      iv. Radiation
         1. Ionizing radiation – damages molecules by charging them and making them highly reactive
         2. UV light causes DNA mutations
         3. Microwaves generate heat by vibrating molecules
      v. Sonication – sounds vibrations disrupt organelles/membranes
b. Chemicals
   i. Phenols – disrupt membranes and denature proteins. E.g. phenol, cresol
   ii. Halogens and metals – act as oxidizing agents (addition of oxygen and sulfur-containing groups) or replace existing chemical groups. E.g. iodine, nickel (Fig. 7.8)
   iii. Detergents and alcohols – act as surfactants to dissolve membranes. E.g. soap, ethanol.
   iv. Acids/Bases – will break hydrogen bonds, covalent bonds, and ionize many molecules. E.g. bleach, acetic acid.
   v. Peroxygens – free radical oxidizing agents. E.g. hydrogen peroxide
   vi. Alkylating agents – add methyl groups to molecules. E.g. ethylene oxide

C. Specific Agents
   a. Mechanisms of action (Fig. 20.2)
      i. Inhibition of cell wall synthesis
         1. Enzymes that crosslink peptidoglycans are inhibited.
         2. E.g. penicillin. Discovered by Alexander Fleming by accident in 1928
      ii. Membrane disruption
         1. Bind membrane of specific type of organism
         2. E.g. polymyxins distort outer membrane of Gram negative bacteria
      iii. Inhibition of protein synthesis
         1. Bind to bacterial ribosomes. Bacterial ribosomes are smaller and have a different shape than eukaryotes.
      iv. Inhibition of nucleic acid synthesis
         1. Transcription can be disrupted by targeting RNA polymerase
      v. Antimetabolites
         1. Mimic building blocks of macromolecules
         2. E.g. Sulfa drugs resemble PABA, a precursor to folic acid. Acts as an enzyme inhibitor. Discovered by Domagk and Fourneau in 1936

b. Side effects
   i. Toxicity – sometimes, there may be an effect on our own molecules. E.g. polymyxins can disrupt our own membranes. Kidney failure and respiratory arrest are possible.
   ii. Allergy – immune reaction to drug or breakdown products. E.g. penicillin
   iii. Disruption of normal flora – our “good” bacteria can be killed off and this may allow opportunistic infections. Products are available to replace normal flora.

c. Resistance
   i. Acquired by mutation, transformation, transduction, conjugation
   ii. Mechanisms
      1. New enzymes. Penicillin can be broken down by β-lactamase.
      2. Membrane permeability is blocked. Import mechanisms are change that makes the drug no longer transported across membrane.
      3. Target is altered. E.g. ribosomes changed shape so that penicillin no longer binds
      4. Ejection of drug. New mechanism that exports the drug rapidly before it can harm the cell.
   iii. Selective pressures that cause resistance
      1. Unnecessary prescriptions – about 50% of all antibiotic prescriptions are unnecessary.
      2. Cross-resistance – acquired resistance to one drug can also give resistance to other related drugs.
3. Premature termination of treatment results in greater chance of having surviving resistant organisms