BIOL 240 GENERAL MICROBIOLOGY (Part IV) – Condensed Terms & Concepts 11/24/19

13. <u>VIRUSES</u>: DNA or RNA, double or single-stranded; protein coat, some enveloped, specific host range receptors (spikes). Helical, polyhedral; Obligate intracellular parasites – must be grown in host cells → Plaques. Identify – cytopathic effects, serology, PCR.
<u>Bacteriophage</u>: Lytic cycle: Attachment, penetration – inject DNA, biosynthesis, Maturation/ assembly, Release – lysis of host....
<u>Lysogenic cycle</u>: *prophage* genome integrated into host chromosome.
<u>Animal Viruses</u>: attachment, penetration by endocytosis or fusion, uncoating, biosynthesis of viral DNA and proteins, Maturation/ assembly, release – budding or rupture. <u>Retrovirus</u>: *reverse transcriptase*, **Provirus**; Budding of enveloped viruses. <u>PRIONS</u> --- spongiform encephalopathies, resistant proteins that convert normal cellular proteins to parasitic. <u>VIROIDS</u> – small, stable infectious RNAs in plants.

- 15. PATHOGENESIS MECHANISMS: pathogenicity, virulence. Entry-Mucous membranes, skin, parenteral. ID₅₀, LD₅₀. <u>Adherence</u> – adhesins (capsule, fimbriae, M-protein); <u>Virulence Factors – enzymes, nutrient</u> <u>acquisition, immune avoidance/camouflage, toxins</u> (coagulase, kinases, hyaluronidase, collagenase, IgA proteases, Siderophores, Antigenic variation); <u>Exotoxins</u> – A-B toxins, <u>Superantigens</u> (*TSST, enterotoxin*), Membrane-disruption (hemolysins, phospholipases, leukocidins), <u>Lysogenic conversion</u>. Endotoxin shock.
 - Virulence Factors: Promote infection, invasion, and/or survival of the pathogen inside the host organism. <u>E.g.</u>:
 - a. Adhesins
 - b. Invasins
 - c. Colonization factors
 - d. Nutrient acquisition factors
 - e. Camouflage & other Immune Avoidance factors
 - f. Toxins exotoxins, endotoxins
 - g. [Enzymes invasins, toxins, digestive, camouflage.....]
- 16. NONSPECIFIC HOST DEFENSES: Mechanical factors: Skin (epidermis, keratin), Mucous membranes, Ciliary escalator, lacrimal apparatus, saliva, urine. Chemical Factors: Sebum, low pH, Lysozyme, Gastric juice, Normal microbiota. Leukocytes: Neutrophils/PMNs, [Lymphocytes B, T], Monocytes, Eosinophils, Basophils. Phagocytosis evasion (M-protein, capsules; Leukocidins, survive phagosome). INFLAMMATION redness, heat, swelling/edema (histamine, prostaglandins), Margination and Diapedesis (emigration) of WBCs, tissue repair. Fever: hypothalamus reset higher in response to endotoxin→IL1→ shivering & fever. COMPLEMENT System: Opsonization, MAC = C6-C9 Cytolysis, chemoattraction of phagocytes (inflammation). Classical Pathway (IgG + C1 → C3, C5); Alternative pathway (B, D, P serum proteins bind cell wall). INTERFERONS: alpha & beta → stimulate anti-viral defense in neighbors; Gamma Interferon → phagocytosis of bact.
- 17. SPECIFIC HOST DEFENSES: Innate defenses; Immunity; Humoral: B-Lymphocytes, extracellular Ag's: Antigen-Antibody interactions → (agglutination, opsonization, complement activation, inflammation, neutralization, Abdependent cell-mediated cytotoxicity), Epitopes.. IgG, IgA.
 Clonal Selection, Effector cells (plasma B, or Tc), Memory cells. Cytokines. Cell-Mediated: T cells (Thymus) intracellular Ag's. M-Cells, Pyers patches. Th (CD4+) cells – interleukins → B & T cells, NK; Tc/CTL (CD8+) cells – perforin. [[Antigen Presenting Cel [APC] (macrophage, dendritic, any host cell) presents antigen-MHC complex→ IL1 → activates Th cell (IL2) → activates Tc & B cells.]] "Professional" APC's = macrophages, neutrophils /

PMNs, B-cells, dendritic cells, etc. Present phagocytized EXTRAcellular antigen on MHC-II, recognized by <u>TH-Cell</u> <u>Receptor and CD4+ on Helper T-cells</u>. All nucleated body cells can present INTRAcellular antigen via MHC-I antigen recognized by TCR on Tc cells, and MHC-I recognized by CD8+ receptor. <u>Nonspecific</u>: activated macrophages, <u>NK cells</u>.

- HIV & AIDS: Infects CD4 cells (Th, macrophages, dendritic cells).
 CD4-<u>gp120 protein</u>, endocyt., uncoating, <u>Reverse</u> <u>transcriptase</u>, and <u>HIV protease</u>. (Provirus <u>latent infection</u>, in vacuoles, fast mutation). Nucleoside RTase inhibitors, nonnucleoside RTase inhibitors, Protease inhibitors, Virus decoys.
- SKIN DISEASES: gram+. salt-tolerant normals (*staph., micrococcus, diptheroids*). <u>Staph aureus</u> leukocidin, exfoliative toxin (scalded skin, TSS (toxic shock)); <u>Strep pyogenes</u> Group A β-hemolytic; M-proteins, streptokinases, hyaluronidase, <u>Exotoxin A</u> (superantigen/TSS). <u>Pseudomonas aeruginosa</u> (gram -) –otitis, postburn. <u>Herpes</u> HHV1/2; latent in nerve ganglia
- 22. NERVOUS SYSTEM DISEASES: Meningitis in cerebrospinal fluid all meningitic bacteria= polysaccharide capsules. Haemophilus influenzae (gram-), capsule B (gram -, throat microbiota). Strep. pneumoniae Encapsulated, normal nasopharyngeal; Listeria monocytogenes: gram+, grow in fridge, foodborne, cross placenta, grow in phagocytes, actin tails/rockets for cell-cell spread. Tetanus = toxin-induced disease (no colonization of host) gram +/ spores/ anaerobe (Clostridium tetani); Tetanospasmin (Ttx) → spastic paralysis. Botulism: (C. botulinum) ingest Botox → flaccid paralysis.
- 23. BLOOD & LYMPH DISEASES: lymphatics, lymph nodes, interstitial fluid, sepsis, septic shock. <u>ANTHRAX</u> (*B. anthracis*) cutaneous (cipro antibiotic), G.I., inhalation ~100% mortalty. Soil grows and kills macrophages, release toxins, cause lesions. <u>HIV.</u>
 <u>PLAGUE:</u> Y. pestis. Survives and proliferates in phagocytes. Bubonic, Septicemia, *Pneumonic (~100% mortality)*. Reservoir = rodents, vector = rat flea; YOPs, YADs. <u>HIV/AIDS (See Ch. 19)</u>. Gp120, reverse transcriptase.
- 24. <u>RESPIRATORY DISEASES</u>: <u>Diphtheria</u> (gram +, Corynebacterium diphtheriae); Dtx kills host throat cells, damages heart and kidneys. membrane of fibrin, dead tissue, and bacteria. <u>Pertussis</u> (Bordatella pertussis, gram-) Capsule, Ptx; Tracheal cytotoxin kills ciliary cells desperate coughing. (Whooping). <u>Tuberculosis</u> (acid-fast, *Mycobacterium tuberculosis*). In alveolar macrophages; Tubercule; liquefaction → cough blood, sepsis Influenza: (8 ssRNA, env. virus) fever, headache, muscle ache only! H- & N-spikes.
- 25. GASTROINTESTINAL DISEASES: Fecal-Oral cycle; Normal microb. -mouth, large intestine (Bacteroides, E. coli,...). Infection= 12hr-2wk; Intoxication = 1-48hr. Staph. Food poisoning – Enterotoxin superantigen preformed; heat-stable! Shigellosis: Shigatoxin: invades epithelium, spreads cell-cell with lysis using actin-tail; grow in macrophages; bloody diarrhea/dysentery (Like E. coli O157:H7 = shigatoxin!). Salmonella enterica Typhimurium – Salmonellosis: replicates in epithelium, invades blood/lymph → liver, kidneys, fever, perforate intestine, sepsis; Salmonella typhi (Typhoid Fever) – systemic in phagocytes; severe sepsis, fever, shock. Viral gastroenteritis = Rotavirus, Norovirus.
- 27. ENVIRONMENTAL MICROBIOLOGY: Mycorrhizae. Biogeochemical cycles: Carbon cycle, Nitrogen cycle (decomposition, ammonification, nitrification, denitrification, nitrogen fixation). Microsymbiont, macrosymbiont. Legume-Rhizobium Symbiosis: root nodules, infection threads, colonization, N-fixation. Bioremediation; <u>Biofilms</u>.

Microbiology FINAL – Part IV (Fall 2019): Study Questions

* Possible Short Essay Topics (be prepared to draw diagrams as well!):

- 1. <u>Ch. 13</u>: Describe & diagram a basic <u>bacteriophage</u> reproductive cycle. How does this compare to infection by <u>animal viruses</u>? Describe at least <u>3 special adaptations</u>, including latent phases, that some animal viruses have to avoid the immune system?
- 2. Diagram and Describe <u>enveloped retrovirus structure</u> and its <u>reproductive cycle</u>. Compare and contrast this with the structure and life cycle of an <u>unenveloped Animal DNA virus</u>. Describe at least 3 special adaptations, including proviral stages, that retroviruses (and some other viruses) have to avoid the immune system.
- <u>Ch. 15</u>: Describe at least <u>5 types</u> of <u>Virulence Factors</u> used by bacteria to attach, locally colonize or penetrate, and systemically invade a susceptible host. What effects do these virulence mechanisms have on the host? *Provide specific <u>examples of each</u> from <u>specific species</u>.*
- 4. <u>Ch. 16</u>: Describe <u>5</u> nonspecific host defenses (innate immune factors) that protect the mucosal surfaces and the bloodstream from pathogenic invasion. Occasionally, how might some of these defenses contribute to damage of the host?
- 5. Compare the responses by serum <u>complement</u> (alternative pathway; <u>3</u> responses) to <u>antibody</u>mediated responses (<u>5</u> responses) to bacteria in the bloodstream. What host cells are involved in each response, and how are bacteria or other antigens destroyed or effectively removed?
- 6. <u>Ch. 17</u>: Compare and contrast <u>5</u> differences between the <u>humoral</u> and <u>cell-mediated</u> responses of the <u>Acquired Immune System</u> to pathogen invasion. What cells and protein/chemical factors are involved in each during a successful response? How is long-term immunity produced? What <u>cells</u> and <u>chemical factors</u> are involved in both responses?
- 7. List <u>5 molecules and cell types</u> that are shared and interconnect the <u>Innate</u> defenses of the body with the <u>Acquired/Adaptive Immunity</u> pathways. Define the defensive functions of each shared component and explain how they connect the immune and innate pathways.
- 8. Chs. 21-25: Compare in detail <u>2</u> microbial diseases of each of the following organ systems: <u>skin</u>, <u>nervous system</u>, and <u>blood/lymphatic systems</u>. How does a successful pathogen gain entry, invade host tissues (if applicable), and damage the host? What host defenses and medical treatments are a patient's best hopes? Are disease symptoms caused by growth of the pathogen within the host, or by virulence factors (eg: toxins) released by the microbe, or both?
- 9. Compare in detail <u>3</u> microbial diseases of each of the following organ systems: <u>gastrointestinal</u> <u>system</u>, and <u>respiratory system</u>. How does a successful pathogen gain entry, invade host tissues (if applicable), and damage the host? What host defenses and medical treatments are a patient's best hopes? Are disease symptoms caused by growth of the pathogen within the host, or by virulence factors (eg: toxins) released by the microbe, or both?
- 10. Ch. 27: Describe three environmental (ecological) processes that require microbial metabolism in order to proceed successfully and efficiently. What specific biochemical activities by the microbes contribute to each of these processes? What would happen to the environment or ecosystem if the participating microorganisms were removed (be specific!)?
- 11. Compare the <u>symbiotic</u> interactions between the organisms involved in forming Lichens, Mycorrhizae, and Legume-*Rhizobia* symbioses. What special adaptations and benefits do these associations provide for the organisms involved, and what effects are produced on the ecosystem?

!!! GOOD LUCK on <u>Tuesday., 12/10/2019</u> (<u>11:10 AM Sharp</u>!) !!!

BIOL 240 (GENERAL MICROBIOLOGY) - CUMULATIVE Terms & Concepts 11/24/19

<u>Tortora et al. Chapter:</u>

- Atoms, molecules; nucleus, protons, neutrons; electrons. Chemical bonds: covalent, ionic (cation, anion), hydrogen bond. Energy: endergonic reactions, exergonic reactions; synthesis/ anabolism (condensation/ dehydtration reactions), Decomposition/ catabolism (hydrolysis reactions).
- Water polar, solvent, H-bonds, *Hydrophilic, Hydrophobic.* dissociates (ionization/dissociation = H+, -OH) = pH. Acid, Base. pH Buffers
- CHNOPS; Carbon Skeleton, functional groups (hydroxyl, amino, sulfhydryl, carboxyl, phosphate). <u>Carbohydrates</u> (CH₂O) = mono/di/poly-saccharides, Isomers; Lipids = hydrophobic, mostly CH, tryglycerides (saturated fat, unsaturated oil); pigments, steroids/cholesterol, phospholipids. <u>Proteins</u> = amino acids; enzymes, transporters, toxins, movement, hormones. **Primary, secondary (** α -helix, β -pleated sheet) tertiary, quaternary protein structure. –SH, disulfide bridges (cysteine). <u>Nucleic Acids</u> = DNA, RNA. Ribose/deoxyribose, nucleotides, purines (A, G), pyrimidines (C, T, U), double helix, hydrogen bonds (A=T, G=C), phosphate-sugar-base; ATP.
- Prokaryote: Eukaryote comparison. Glycocalyx capsule, slime layer. Flagella runs and tumbles; counterclockwise and clockwise rotation ("corkscrew"). Axial filaments/ endoflagella, Fimbriae, Pili.
- CELL WALL: peptidoglycan (NAG-NAM)n; Polypeptide crosslinks; D-amino acids; Gram + vs. Gram- Cell Walls. Techoic acids (wall & lipo-); Outer membrane, O-antigens and Lipid A = Lipopolysaccharide outer leaflet. Gram stain Mechanism. Plasma Membrane *Fluid Mosaic Model* ("proteins afloat in a sea of phospholipids"; integral & peripheral membrane proteins). Chromatophores, thylakoids, Simple Diffusion, Facilitated Diffusion, Active transport. Isotonic, Hypotonic, Hypertonic solutions. Osmosis/ osmotic pressure. Nucleoid, cytoplasm; Endospores sporulation, germination, vegetative cell.
- **Eukaryotic Cells**: Flagella & Cilia (9+2 microtubules); Euk. Cell walls = cellulose, chitin, glucan/mannan; Cytosol, Cytoskeleton (actin microfilaments, tubulin microtubules, keratin/lamin intermediate filaments – very dynamic structure and movement, cytoplasmic streaming; **Endocytosis** (Phagocytosis, Pinocytosis) & Exocytosis, Lysosomes. Nucleus, Lysosome, Vacuole, Mitochondria, Chloroplasts. *Endosymbiosis Theory.*
- Metabolism, enzymes, cofactors, regulating enzyme activity; coenzymes, feedback inhibition, Catalysis, Activation Energy. reaction rate. Denaturation, ribozymes, Redox reactions; Reduction, Oxidation. ATP. Ribozymes. Reduction, Oxidation; Carbohydrate catabolism: <u>Glycolysis</u> (invest 2 ATP, harvest 4 = 2 net; <u>6C</u> <u>glucose → 6CO₂ + H₂O + Energy.</u> (<u>6 NADH, 2FADH₂, 4CO₂, 2 ATP per glucose in TCA</u> <u>cycle</u>), <u>Electron Transport Chain</u> (ETC; Proton Motive force, Chemiosmosis, Ubiquinone/Q, Cytochrome C, Cytochrome C Oxidase, O₂; <u>ATP Synthase</u>).
 <u>2ATP/FADH₂, 3ATP/NADH</u>. Prokaryotes harvest 38

ATP/glc, Eukaryotes harvest 36 ATP/glc. <u>Anaerobic</u>

<u>Respiration:</u> Nitrate, Sulfate, or Carbonate electron acceptors – none yields as much energy as O₂. <u>Fermentation</u> = only 2 net ATP/glc; organic electron acceptor with lots of energy left over – regenerates NAD+ for glycolysis. *Anabolic and catabolic pathways are tied together (feed off of each other for Carbon and Energy)*!!

- 6. MICROBIAL GROWTH: Physical Requirements (Temp = psychro-, meso-, thermo-philes; <u>pH</u> = acido-, neutero-, alkalo-philes; <u>Osmotic pressure</u> = halophiles/ "saltlovers"); <u>Chemical Requirements</u> <u>C, N, S, P & Trace</u> <u>elements</u>; <u>O2</u> (toxic = Singlet, Superoxide, Peroxide anion, Hydroxyl radical): *obligate aerobes, facultative aerobes, obligate an-aerobes, aerotolerant anaerobes, microaerophiles*. – Superoxide Dismutase, Catalase, Peroxidase. <u>Culture Media</u>: Chemically Defined, Differential, Selective. Binary Fission. Bacterial growth curve (lag, log, stationary, death phases).
- MICROB. GENETICS: Genetics, Gene, Genome, Genotype, Phenotype. E. coli as Model Organism. <u>Central Dogma</u>: DNA (replicates) → (transcription) RNA → (translation) Proteins; <u>DNA</u>: A=T, G≡C. Semi-Conservative & Bidirectional <u>Replication</u>, Antiparallel Strands, Origin of Replication, 5'→3' synthesis, DNA Polymerase (III), RNA Primase, RNA Primers, template strands, DNA Ligase. Continuous leading strand, discontinuous Lagging Strand Synthesis.
- ★ <u>Transcription</u>: RNA Polymerase; mRNA, tRNA, rRNA, 5'→3' from PROMOTER on DNA template, unidirectional synthesis! Codons/Genetic Code stop (UAA, UAG, UGA) and start (AUG) codons. <u>Translation</u>: mRNA + tRNA with Amino Acid attached (and AntiCodon) + Small Ribosomal subunit + Large Ribosomal subunit. Ribosome moves 5'→3' along mRNA template, Peptide synthesized Amino (N) Terminus → Carboxyl (C) Terminus according to mRNA codons starting with AUG/methionine. Constitutive Enzymes, Repressible Enzymes.
- OPERONS: = promoter + operator + structural genes, regulated by a Repressor protein. Lac Operon (catabolic) = ON if Lactose present (repressor OFF when BOUND by lact. signal) AND glucose absent; Trp **Operon (anabolic/ biosynthetic)** = ON if Trp end product NOT in excess and bound to Trp Repressor (repressor OFF when UNBOUND by Trp signal). MUTATIONS: chemical and radiation mutagens. Missense, Nonsense, Frameshift mutations. (& "Silent" mutations). Horizontal Genetic Transfer: **Transformation** (Griffiths: $R \rightarrow S$ pneumococcus), recombination/crossover into chromosome ("homologous gene replacement"), Conjugation (F factor/ Plasmid, sex pilus, Hfr Cells; F+ donor cells, F- recipient cells). Bacteriophage Transduction. R-Factors (MDR), Transposons.
- Ch. 11-12: Know Major differences Between: Bacteria, Archaea, Protozoans, Algae, Fungi, and Helminths.

Microbiology FINAL – Cumulative Portion (Fall 2019): Study Questions * <u>Possible Short Essay Topics</u> (be prepared to draw diagrams as well!):

- 1. ** Using simple <u>diagrams</u>, compare and contrast the general chemical structures and shapes of <u>each of the</u> <u>four major types of macromolecule</u>. Use <u>specific examples</u> to briefly <u>explain</u> how these structures directly contribute to the cellular functions of each type of molecule.
- 2. Compare and contrast <u>10 *structural*</u> and <u>genetic</u> characteristics that distinguish between "<u>Prokaryotic</u>" and <u>Eukaryotic</u> cells. Be sure to define/describe the significance of the characteristics that you mention.
- 3. <u>Describe</u>, <u>diagram</u> and give specific <u>examples</u> of each level of protein structure. Include the types of molecular bonding and interactions that are important at each level.
- 4. Diagram and describe the prevailing model on the <u>structure of biological membranes</u>. Include a brief description each of <u>4</u> major types of membrane transport and <u>2</u> types of transporter molecules.
- 5. Describe and give examples of each level of protein structure. Include the types of molecular bonding/ interactions that are important at each level.
- 6. Describe <u>6 properties</u> of DNA structure and DNA replication that are essential to its successful synthesis, stability, and function. (Hint: consider comparisons to RNA structure and function!)
- 7. Diagram & compare the biochemical structures and general properties of the gram positive and gram negative cell walls. How does the Gram stain distinguish between the two?
- 8. Diagram and describe how enzymes speed up chemical reactions, and explain how they affect the energy and equilibrium of a reaction. Describe <u>6</u> different physical and chemical factors that can regulate enzyme activity. How do enzymes affect metabolism and growth??
- 9. <u>DIAGRAM</u> and briefly <u>explain</u> how and <u>where</u> energy from high energy <u>electron carriers is converted to ATP</u> during respiration in mitochondria and aerobic bacteria. Label each process involved in energy conversions, and label two of the proteins involved in the final steps of making ATP. (compare this to ATP production during *Photosynthesis*)
- 10. Describe & diagram when and how <u>six carbons</u> in glucose are all transferred and released, & in what form (molecule), from glycolysis through the Krebs (TCA) cycle. What else happens each time carbons are released?
- 11. Compare and contrast the <u>energy inputs and outputs</u> of aerobic respiration vs. fermentation. Include all phosphorylated compounds and high energy electron carriers, and briefly explain how these are produced.
- 12. Explain how the <u>Central Dogma</u> of molecular genetics illustrates the process by which hereditary Genotype becomes hereditary Phenotype in an organism (include 3 processes involved in Heredity and "Gene Expression").
- Distinguish between the starting sequences and ending sequences and enzymes used to initiate, polymerize (elongate), and terminate <u>Replication</u>, <u>Transcription</u>, and <u>Translation</u>. *Define each process, including directions of synthesis, and the type of molecule produced.*
- 14. Compare and contrast regulation of the <u>LAC Operon</u> and the <u>TRP Operon</u>. When is each turned ON or OFF? <u>Draw</u> each operon in the PRESENCE of its own ligand (signal molecule). What controls the activity of the regulatory proteins involved? Explain <u>how each type of regulation is appropriate</u> for an operon encoding catabolic or anabolic enzymes
- 15. ** Diagram and describe at least THREE examples of <u>allosteric control</u> of protein activity in microbial <u>metabolism</u> and genetics/ <u>gene regulation</u>. Define *allostery*, and explain why it is such an efficient method of controlling cellular processes.
- 16. Compare and contrast the <u>cellular</u>, <u>nutritional</u>, <u>and life cycle (sexual/asexual</u>, <u>haploid/diploid</u>) characteristics of <u>protozoan</u>, <u>algal</u>, and <u>fungal</u> microbes and <u>helminths</u>. Give an example of each that is important to humans or to the environment.

** Once again: concentrate on the <u>study questions first</u>, and see how the detailed terms fit into these most important concepts. Then, concentrate on the <u>bolded and italicized terms</u>! ** ** **Pay particular attention to Chapters 2, 4, 5 & 8,** **

Terminology from Ch's 1-5 should already be part of your personal biological vocabulary!! *** Also, Make sure you are still familiar with the <u>major differences</u> between the Prokaryotic and Eukaryotic Microbes & Viruses in <u>Chapters 11, 12 & 13!!</u> ***

NOTE: The Cumulative Portion is 33% (50pts.) of the Final Exam. So, take it seriously, review and practice the essays, but try not to spend <u>too</u> much time on the older material!!