Hannah’s Study Guide Exam 3

Section X – Infection and Immunology

1. Immune system overview
   1. Important components of the immune system
      1. *Barrier tissue* – tissue that doesn’t have loose junctions and doesn’t allow bacteria and other foreign objects to pass through
         1. Non-specific immune system
         2. Ex: epithelial tissue in the gut, skin
      2. *Chemical secretions* – involved in attacking pathogens directly or stimulating other parts of our body to do it effectively
         1. Specific or innate immune system
         2. Ex = proteins
      3. *Leukocytes* – cells involved in the immune system (WBCs)
         1. Specific or innate immune system
      4. *Lymph tissue* – allow WBCs to migrate through the body
         1. Ex = thymus, spleen, lymph nodes
   2. Cells of the immune system
      1. *Key terminology*
         1. **Pathogen-associated molecular patterns (PAMPs) =** unique microbial signatures that form from specific sequences within proteins, polysaccharides, lipids, nucleic acids, etc.

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| Cell | Function |
| Leukocytes | * Innate and adaptive immunity * **Pattern recognition receptors = PRRs =** receptors that bind to PAMPs; are soluble and membrane bound (e.g. acute-phase proteins, MBP) * Membrane bound PRRs on phagocytic cells allow 2 things (1) distinguish btwn harmful microbes and host molecule and (2) respond by ingesting/degrading the PAMP source |
| Mast cells = bone marrow-derived cells that differentiate in connective tissue | * Cytoplasm filled with granules * Nonphagocytic * release granule content to extracellular environment when stimulated (degranulation) * Have high-affinity receptors for type of antibody associated with allergic response |
| Granulocytes | * Granules in cytoplasm contain reactive things that kill microbes and enhance inflammation (3 types) * *Basophils* * *Eosinophils* * *Neutrophils* (polymorphonuclear neutrophils = PMNs): phagocytic cells. Circulate in blood to migrate to tissue damage when it happens (have 2 kinds of granules) * Primary: contain peroxidase, lysozyme, defensins * Sencondary: collagenase, lactoferrin, lysozyme |
| Monocytes | * Migrate into tissues and mature into macrophages or dendritic cells |
| Macrophages | * Have a plasma membrane covered with microvilli * Bridge innate and adaptive immunity * Binding of their PRRs to PAMPs induces phagocytosis (2 kinds of PRR) * *NOD-like receptors:* soluble and remains in cytoplasm of phagocyte * Sense endogenous metabolites and they regulate apoptosis * Detect cytosolic PAMPs and DAMPs (damage associated molecular patterns) – e.g. heat shock proteins * *Toll-like receptors:* recognize and bind unique PAMPs (LPS, peptidoglycan, flagellin, etc.) and communicate by binding to host cell nucleus to initiate appropriate gene expression and host response * Migrate as activated cells to lymphoid tissues to present antigens to lymphocytes |
| Dendritic cells | * Heterogeneous group of cells * Tissue bound to bridge innate and adaptive immunity * Detect and phagocytose foreign things * Capture foreign stuff with cell surface receptors |

* 1. Phagocytosis
     1. *Key terminology*
        1. **Phagocytosis =** a process in which cells recognize, ingest, and kill cellular microbes
        2. **Lysosome =** an organelle that fuses with the phagosome
     2. *2 mechanisms*
        1. Opsonin-independent recognition: molecular patterns are recognized to activate phagocytes through PRR
        2. Opsonin-dependent recognition: complement activation
     3. *Intracellular digestion removal processes (2)*
        1. imports extraceullar particles via cell membrane extension to get an internal phagosome to initiate phagocytosis
           1. *Lysosome contains 4 things*

Hydrolytic enzymes

Cationic antimicrobial peptides/defensins

ROS

RNS

* + - 1. beings with intracellular microbes and coats them with ubiquitin to label them for capture by a phagophore
    1. *2 things after phagocytosis*
       1. exocytosis = process in which the cell expels the microbial fragments (neutrophils)
       2. antigen presentation

1. Innate Immune System
   1. Physical and mechanical barriers

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| Barrier | How they kill microbes |
| Skin | * Skin cells shed a lot to get rid of microorganisms * pH 5-6 due to sebum and organic acids made by staphylococci |
| Mucous membranes | * stratified epithelium and mucus form covering that prevents penetration; traps microbes * covered in specific antimicrobial secretions (3 examples) * **lysozyme =** enzymes that lyses bacteria by hydrolyzing te bond connecting NAM and NAG * **lactoferrin =** released by activated macrophages and polymorphonuclear leukocytes (PMNs) to sequester iron from the blood plasma to reduce the amt available to invading microbes * **lactoperoxidase =** enzyme that catalyzes production of superoxide radicals |
| Respiratory system | * microbes are trapped by hairs and cilia in the nasal cavity and mucous * coughing and sneezing clear the system * salivation rides them |
| GI tract | * gastric juice (HCl, proteolytic enzymes, mucus) is acidic (pH 2-3) * peristalsis and shedding of columnar epithelial cells purge intestinal microbes |
| GI tract | * Urine is acidic * Kidney is hypertonic |

* 1. Chemical mediators

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| Chemical mediator | How they kill microbes |
| Antimicrobial peptides | * Electrostatically attracted to microbial cell surfaces due to positive charge * 2 major types * *Cationic peptides:* damage bacterial plasma membranes (e.g. defensins) * *Bacteriocins:* peptides that give their producers an adaptive advantage against other bacteria |
| Cytokines | * **Cytokine =** any soluble, low weight protein or glycoprotein that is released by one cell population that acts as an intercellular mediator or signaling molecule * **Colony-stimulating factors =** cytokines that stimulate the growth and differentiation of immature leukocytes in the bone marrow * **Monokines =** cytokines released from mononuclear phagocytes * **Lymphokines =** cytokines released from T lymphocytes * **Chemokines =** cytokines that stimulate chemotaxis and chemokinesis * **Interleukins =** cytokines made by 1 leukocyte acting on another leukocyte * **Interferons =** cytokines made by eukaryotic cells in reponse to viral infection |
| Acute-phase proteins | * **Acute-phase proteins =** mediators that assist in the prevention of blood loss and prepare the host for microbial invasion |

* 1. Gut
     1. *Key terminology*
        1. **GALT** = gut associated lymph tissue
        2. **M cell =** cells in the gut that lack the brush border of microvilli found on adjacent columnar epithelial cells; reside above large epithelial pockets that contain B and T cells, and macrophages
        3. **Macrophage =** resides in pocket that come from monocytes
     2. *Process of foreign particle invasion:* foreign particle comes to mucosal surface > contacts M cell > pathogen is endocytosed > released into pocket > macrophages engulf particle and try to degrade it > B cells in follicle rxt to pathogen and mature into Ab-producing plasma cells > plasma cells leave follicle > plasma cell secretes mucous membrane-associated Ab > Ab transported to lumen of the gut > Ab interacts w the particles that made the pathogen
     3. *Phagocytic cells in GALT:* monocytes, macrophages, dendritic cells
     4. *5 mechanisms a pathogen can take to avoid phagocytosis (aided by losing PAMPs and taking over actin)*
        1. invade epithelial cells
        2. divide within M cells or phagocytic cells
        3. invade neighboring cells from M cells
        4. resist phagocytosis
        5. resist lysis inside phagocytic cells
  2. Complement
     1. *Key terminology*
        1. **Complement =** 30 interacting proteins that are in the gut mucus and GALT and common in blood; produced by liver. Proteins cascade to lyse cell membranes, augment phagocytosis, and make inflammatory peptides
        2. **Opsonization** = signal from the immune system that increases the likelihood of phagocytosis of the object; the process in which microorganisms or inanimate particles are coated by serum components to prepare them for recognition and ingestion by phagocytic cells (complement proteins are opsonins and are a connector btwn microbe and phagocyte)
     2. *4 functions*
        1. lysis of bacteria by a membrane attack complex to cause bacteria to lose cell integrity
        2. opsonization
        3. bridges innate and adaptive immune systems via synergistic activity with antibodies
        4. disposes of waste
     3. *3 pathways of complement activation*

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| Complement activation pathway | Description |
| Alternative complement pathway | * Activation triggered in response to bacterial and fungal molecules with repetitive structures (e.g. lipopolysaccharide LPS) * LPS activated complement C protein 3 (C3) > cleaves into fragments C3a and C3b > C3b binds to LPS and becomes stable > C3b acts as an opsonin and helps catch bacteria for phagocytes that have receptors for C3b > blood Factor B adsorbs to bound C3b > factor D cleaved factor B and C3b into 2 fragments > active enzyme C3bBb is formed (C3 convertase) > cleaves more C3 to C3a and C3b |
| MB-lectin pathway | * C3 convertase is formed > lectin protein initiates proteolytic cascade (e.g. **mannose-binding protein MBP =** protein made when macrophages ingest viruses, bacteria, etc.; binds mannose and enhances phagocytosis thus it’s an opsonin > forms complex with blood protein MASP > MBP binds to mannose on pathogens > MASP activated > MASP cleaves C4 and C2 complement proteins > c3 convertase is formed |
| Classical complement pathway | * Occurs in response to only some microbial products; usually initiated by interaction of antibodies with antigen * Antibody binds to antigen > c1 complement activated > c1q binds to antigen-bound antibody > c1s is activated > c1s attacks and cleaves c4 and c2 > binding of each molecule to antigen-antibody complement complex |

* + 1. Membrane attack complex = MAC = creates a pore in the plasma/outer membrane
       1. *if target is eukaryotic:* Na+ and H2O enter and cell lyses
       2. *if target is gram-neg bacterium:* lysozyme passes through pores to digest peptidoglycan and weaken cell wall > lysis
       3. *if target is gram-pos bacterium or fungi:* resist action bc they don’t have exposed outer membrane

1. Acquired Immune System
   1. Key terminology
      1. **antigen presenting cell = APC =** a macrophage/dendritic cell/B cell that takes in the antigen or pathogen by receptor-mediated endocytosis or phagocytosis and produces antigen fragments via digestion in the phagolysosome
   2. 4 characteristics that distinguish from innate: discrimination btwn self and nonself, diversity, specificity, memory
   3. 2 kinds of acquired immunity
      1. *Naturally acquired immunity =* acquired through the normal life experiences of a human and is not induced through medical mean (2 kinds)
         1. Active when there is exposure of the host to antigen (e.g. sickness)
         2. Passive when one person is receiving preformed immunity made by another person (e.g. from mom to baby through placenta or breast-feeding)
      2. *Artificially acquired immunity (2 kinds):*
         1. Active when there is exposure to intentionally introduced weakened antigen (e.g. vaccine)
            1. “boosters” are given to “re-awaken” memory cells
         2. Passive when antibodies being transferred from 1 host to another artificially (e.g. therapeutic medicine)
   4. Lymphocytes (3 types)
      1. *T cells*: leave the bone marrow and mature in the thymus gland
      2. *B cells*: originate and mature in the bone marrow
      3. *Natural Killer cells*: non-phagocytic granular lymphocytes that attack and destroy cells (recognize targets in 2 ways)
         1. Look over somatic cells for MHC I complexes and if they come across one with altered/missing, cell is destroyed
         2. Find target cells that are coated with antibodies and attack them via perforin proteins and granzymes
   5. Major histocompatibility complex = MHC (3 classes)
      1. *class I molecules:* found on all nucleated cells to identify them as self
         1. stimulate immune response when cells from 1 host are introduced into another host bc they have diff class I molecules (thus we have MHC typing)
      2. *class II molecules:* produced only by activated macrophages, dendritic cells, mature B cells, some T cells
         1. required for T-cell communication with macrophages, dendritic cells, and B cells
         2. contains antigen binding pocket where a non self-peptide can be captured for presentation to other immunocytes
         3. fragments combine with preformed class II MHCs > delivered to cell surface > peptide within antigen binding pocket recognized by CD4+ T helper cells
      3. *class III molecules:* include various secreted proteins that have immune functions
   6. T cells
      1. *Key terminology*
         1. **Naïve =** state of the inactive T or B cells until they are activated
         2. **Antigens =** substances that illicit an immune response; can be self or non-self
         3. **Epitopes =** unique molecular signatures defined by their primary, secondary, and tertiary structure that is bound by specific immune cells
         4. **Valence =** the number of epitopes on the surface of an antigen that determines the number of antibody molecules can combine with it (multivalent antigens elicit stronger immune response)
         5. **T-cell receptor = TCR =** complexes on plasma membrane surface in which T cells can respond to antigen fragments presented in MHC molecules
         6. **Effector cells =** t-helper cells (TH), cytotoxic lymphocytes (CTLs), NK T cells, or T-regulatory cells that respond to diff Ags by making and secreting cytokines
      2. *2 types*

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| T-cell type | Description |
| **T-helper (TH) cells** = TH = CD4+ T cells | * 4 important types * TH1 cells: promote cytotoxic T lymphocyte activity, activate macrophages, and mediate inflammation, produce cytokines to activate macrophages and CTLs * TH2 cells: stimulate antibody responses and defend against helminth parasites, make cytokines to activate specific B-cells * TH9 cells: inflammation and autoimmune disease * TH17 cells: found in skin and intestinal epithelia; response to bacterial invasion, make defensins, recruit neutrophils, mediate inflammation in skin and intestinal epithelium |
| **Cytotoxic lymphocytes** = TC = CD8+ | * Destroy host cells that have been infected by intracellular pathogens (e.g. virus) or have altered MHC surface proteins * To be activated, they interact with APCs through class II MHCs * Any host cell that presents the same antigen is targeted for apoptosis * Kill target cells in 2 ways (1) cytolytic pathway (2) apoptotic pathway |

* + 1. *T –cell activation*
       1. Overall: antigen presentation > binding of MHC antigen presenting cell and T cell > binding causes 3D change in TCR > stimulates cd3 proteins to send signals to t-cell nucleus
       2. CD4+ cells: 2 signals
          1. *Signal 1:* antigen fragment is presented in an MHC II molecule of APC and fills TCR
          2. *Signal 2:* antigens are presented and become effector and memory cells
       3. CD8+ cells: 2 signals
          1. *Signal 1:* antigen fragment is presented in an MHC I molecule of APC and fills TCR
          2. *Signal 2:* antigens are presented
  1. B cells
     1. *Key terminology*
        1. **Immunoglobins =** receptors for antigens
        2. **Isotype =** class of antibody
        3. **T-dependent antigens =** antigens that elicit a response with the help of T-helper cells
     2. *2 roles:* (1) proliferate and differentiate into memory cells and plasma cells that respond to antigens by making antibodies (2) act as APCs to stimulate response in T cells
     3. *antibody (immunoglobulin) structure on surface:*
        1. 2 light and heavy chains (2 regions within each)
           1. *Constant region:* have AA sequences that don’t vary between antibodies of the same class (5 types)
           2. *Variable region:* have diff AA sequences that fold together to form the antigen binding sites
        2. Crystallizable fragment = Fc = the stalk that can bind to a host cell by interacting with the cell-surface Fc receptor (made of constant regions)
        3. Antigen-binding fragments = Fab = bind with compatible epitopes
     4. *5 classes of antibodies*

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| Immunoglobin class | Description |
| IgG | * major Ig in human serum, blood plasma, and tissue fluids * acts against bacteria and viruses by opsonizing the invaders and neutralizing toxins and viruses * activate complement by the classical pathway * able to cross placenta |
| IgM | * first Ig made during B-cell maturation * individual IgM monomers expressed on b cells * serve as antibody component of the BCR * secreted during primary antibody response * agglutinates bacteria in the bloodstream * activates complement by classical pathway * enhances ingestion of pathogens by phagocytic cells * always comes first and then they switch to IgG/A/E |
| IgA | * mucous secretions * protects surface tissues against infection * found in the lumen of the intestine and plasma cell * can switch to IgG |
| IgD | * part of the b-cell receptor complex * signals the b cell to start antibody production when antigen binds * only able to respond to one epitope |
| IgE | * involved in activating mast cells * involved in inflammatory response * gut fluidity to aid in purging of helminths |

* + 1. *5 functions of antibodies*
       1. opsonization – bacteria are coated by antibodies marking them for phagocytosis
       2. complement fixation – complement is activated that causes lysis
       3. agglutination – antigens are clumped together so that macrophage can phagocytose easier
       4. neutralization – reducing the SA by coating the Ag in Abs to block out any secretion systems, prevents adhesion, interacts with complement
       5. precipitation – Abs target antigens that are soluble and if enough of them come together they ppt it out
    2. *Process of utilizing Igs:* B cell 1 > can produce IgD-1 or IgM-1 > then it can switch to IgG-1/IgA-1/IgE-1 OR B Cell 2 > makes IgD-2 or IgM-2 > then it switches to IgG-2/IgA-2/IgE-2
    3. *B-cell activation (2 signals):* (1) antigens binding (2) cytokines from active TH2 cell binding the same antigen on TCR > differentiates into plasma (effector) cells or memory cells
       1. **Plasma cell =** a mature and activated B cell that secrete large quantities of Abs; IgM > IgG in blood and lymph > IgA in mucus
       2. **Memory cells =** lymphocytes in a state of limited activity bc they remember the pathogen that activated them
    4. *2 different exposures*

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| 1st exposure to antigen – primary | 2nd exposure to antigen – secondary |
| 10 days for IgM to be secreted | 5 days for IgM to be secreted |
| 20 days for IgG to be secreted | 10 days for IgG to be secreted |
| Ab total is lower | Ab total is higher |
| Latent period is 10 days | Latent period is 2 days |

* 1. How pathogens survive – virulence factors help them do 4 things
     1. Evade complement – with proteases and resistant forms of LPS
     2. Resist phagocytosis – with capsule
     3. Survive within phagocytes
     4. Evade Abs – by changing surface and with proteases

1. Antimicrobial chemotherapy
   1. 4 functions of antibiotics

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| Function | How they do it | Examples |
| Cell wall synthesis inhibition | Inhibition of peptidoglycan synthesis | * penicillins and cephalosporins: inhibit transpeptidation enzymes that help cross-link the polysaccharide chains in peptidoglycan * vancomycin: prevents transpeptidation of peptidoglycan subunits |
| Protein synthesis inhibition | Bind to ribosomes | * aminoglycosides and tetracyclines: interfere with protein synthesis by inhibiting it and causing misreading of mRNA * macrolides and chloramphenicol: inhibits peptide chain elongation during protein synthesis |
| Antimetabolites | Disrupt folic acid and nucleic acid synthesis | * sulfonamides and trimethoprim |
| Nucleic acid synthesis inhibition | Bind to DNA or RNA polymerase | * quinolones: inhibit DNA gyrase to block DNA replication |

* 1. drugs resistance to virulence factors (4)
     1. bind to the antibiotic before it makes contact with the cell and degrades it enzymatically
     2. alters the antibiotic in the cell enzymatically
     3. pumps the antibiotic out really quickly with an efflux pump
     4. make an altered antibiotic target for it to bind to and then get it out

Bacteria of Humans: Diversity and Diseases (Section X)

1. Bacteria that affect the GI tract
   1. *Vibrio cholera* – Class *proteobacteria*
      1. Cholera – Explosive and blood diarrheal disease. John Snow traced it to a water supply to disprove miasma
      2. Spread through contaminated water or food
      3. Pathogenesis involved binding to mucus and secretion of toxin
   2. *Campylobacter jejuni* – Class -*proteobacteria*
      1. Gastroenteritis, food poisoning
      2. Invades intestinal epithelial cells and produces toxins
   3. *Helicobacter pylori* – Class -*proteobacteria*
      1. Peptic ulcers, gastritis, cancer
      2. Duodenum and gastric juice of stomach
      3. Eat away epithelial tissues – neutrophils and mast cells accumulate in the burrow sites and kill parts of the epithelial tissue > tissue necrosis
      4. Produces urease to create more alkaline microevmt to survive
   4. *Bacteroides thetoiotaomicron* – Class *Bacteroides*
      1. Aids in the breakdown of complex polymers, shed epithelial cells, and mucous
   5. Phylum *Firmicutes*
      1. *The Gram (+) cell wall*
         1. More peptidoglycan than (-) to make up for lack of plasma membrane
         2. Peptide interbridges in the cross-link – ID characteristic bc some have diff AAs
         3. *2 acids* (provide cell w negative charge, serve as PAMPs, stimulate phagocytosis bc of toll-like receptors)
            1. *Lipoteichoic acid =* acid that is anchored in the plasma membrane
            2. *Teichoic acid =* anchored in the peptidoglycan or plasma membrane
         4. Simple basal body
         5. *3 protein secretions*
            1. Sec system
            2. ABC system
            3. Tat system
         6. Conjugation performed without a pilus
         7. Low GC
      2. *Endospores =* dormant state of cells with no metabolic activity that are formed intracellularly during adverse growth conditions
         1. 5 factors that contribute to resistance
            1. Spore coat
            2. Inner membrane
            3. Peptidoglycan cortex
            4. Core containing high calcium, dipicolinic acid, proteins that protect and repair DNA
            5. Low water content
      3. *2 classes*

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| *Bacilli* | *Clostridia* |
| Low numbers in the gut | Most abundant group of bacteria in the gut |
| Non-spore forming | Spore forming |
| Facultative anaerobes that get energy from fermentation | Obligate anaerobes that get energy through fermentation |
| Commensal/mutualistic – used as a probiotic | Opportunistic pathogen |

* 1. *Clostridium difficile*
     1. Chronic diarrhea
     2. *Process:* Normal microbiota > antibiotics > shift in the microbial community > susceptible microbiota > changes in such a way to allow opportunities for colonization by diff organisms > c. diff exposure > c diff infection > treatment > transitional microbiota > recovery > normal microbiota
  2. 3 reasons why composition of gut bacteria varies
     1. Diet
        1. Ex: *Clostridia* from a lot of meat (grows well on AAs)
        2. Ex: *Bacteroidetes* from eating a lot of whole grains/veggies
     2. Ppl you interact with
     3. Environmental conditions

1. Skin and muscle tissue
   1. Overview of the skin
      1. *3 skin conditions that restrict microbial growth*
         1. dry
         2. high salt concentrations
         3. sweat gland secrete moisture and fatty acids
      2. *3 normal gram + residents*
         1. *Proprionibacterium*
         2. *Staphylococcus*
         3. *Corynebacterium*
   2. Genus *Staphylococcus* – Phylum *Firmicutes,* Class *Bacilli*
      1. *Location:* skin, nose, mouth
      2. *6 virulence factors*
         1. slime layer – avoid immune system
         2. catalase – increase ability to grow in atmospheric oxygen and break down ROS in phagolysosomes
         3. protease – degradation of immune system components like complement and Abs
         4. coagulase – lyses cells
         5. hemolysin – lyses cells
         6. enterotoxin
   3. *Staph. Aureus* – Genus *Staphylococcus*
      1. MRSA
      2. Nosocomial
   4. *Streptococcus* – Phylum *Firmicutes*, order *Lactobacillales*
      1. *3 virulence factors*
         1. extracellular enzymes – break down host molecules/dissolve clots
         2. streptolysins – kill leukocytes
         3. capsules (M cells) – resist phagocytosis (2 components)
            1. M proteins – form fimbriae
            2. Capsule – made of hyaluronic acid to avoid immune system by mimicking CT
   5. *Streptococcus pyogenes* (group a)
      1. Cellulitis = infection of the dermal tissue
      2. Necrotizing fasciitis = infection of the muscle and bone tissue
   6. *Streptococcus agalactiae* (group b)
      1. Pregnant women are at increased risk
2. Genitourinary tract and STDs
   1. Normal microbiota

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| Urinary tract | Female genital tract |
| Skin and intestinal organisms in distal portion | *Lactobacillus acidophilus* (maintain pH ~4-5) |
| Upper portion and bladder microbe-free | Community changes w menstrual cycle |
|  | Urethritis (UTI) caused by opportunistic pathogens (e.g. ExPEC) |

* 1. Phylum *Spirochaetes*
     1. outer sheath made of lipids, proteins, and carbohydrates that houses outer flagella (axial fibril)
     2. gram –
  2. *Treponema pallidum*
     1. Syphilis
     2. Treatment with penicillin
     3. *3 stages of syphilis*
        1. Primary – chancre 10-90 days after exposure
        2. Secondary – 3-8 weeks after primary
        3. Tertiary – 10 years after consisting of lesions on infected tissue
     4. Spreads through lymph and blood to skin, bone, and NT
  3. *Neisseria gonorrhoeae* – Class *Betaproteobacteria*
     1. Gonorrhea
     2. Binds to epithelial cells in the urethra and divide in them and spread
     3. Variation in surface proteins lead to avoidance of immune response and reinfection
     4. Infection of fallopian tubes in women > sterility
  4. *Chlamydia trachomatis*
     1. Chlamydia, most common STD in US
     2. Lack peptidoglycan, small genomes
     3. *2 different cell types*
        1. *Elementary body* = ones that spread throughout the environment and bind to host cells to be phagocytosed or endocytosed
        2. *Reticulate body* = vegetative cell that has differentiated from the EB that will do all the division and growth
     4. *Reproduction process*:attach EB to host cell surface > become endocytosed > held in endosome > differentiates in the RB > differentiates until host cell dies
  5. Eye microbes
     1. *How it protects itself:* secrete lysozyme and antimicrobials to breakdown peptidoglycan
     2. *Normal microbiota:* *staphylococcus* species and some skin bacteria

1. Nose and respiratory system
   1. 2 types of respiratory systems

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| Lower respiratory system | Upper respiratory system |
| Lungs | Nose |
| Normally no microbe residents | 4 normal residents: *Staphylococcus aureus, staphylococcus epidermidis, streptococcus pneumonia, Corynebacterium* |
| Infections can spread 2 ways (1) airborne droplets (2) direct contact w infected person |  |

* 1. *Chlamydia pneumonia*
     1. Generally mild
     2. EB and RB
  2. *Streptococcus pneumonia*
     1. Stronger affinity for the lungs
     2. Causes 3 diseases: meningitis, pharyngitis, pneumonia
     3. Primary virulence factor = capsule made of hperolonic acid which resembles our CT
     4. *Process:* as bacteria adhere to and grow on the epithelium of the alveoli in the lungs > fill with fluid > pus (plasma from blood carrying neutrophils and complement and immune cells) > damages lungs > interferes with pneumonia
  3. *Streptococcus pyogenes*
     1. *2 causes*
        1. Pharyngitis (strep throat)
        2. Otitis (ear infection)
  4. *Neisseria meningitides*
     1. Meningitis
  5. *Haemophilus influenza type b*
     1. Pneumonia and co-infects with the flu
  6. *Bordetella pertussis*
     1. Pertussis
     2. *Process:* after inhalation of the cells, attach to tracheal epithelial tissue and grow > secrete toxins > cause necrosis of tissue > cause body to secrete thick mucous > blocks airways > hard to breath
  7. *Mycoplasma pneumonia*
     1. Common cold, mild sore throats, atypical pneumonia
     2. *Atypical in 5 ways*
        1. Long-lived
        2. Slow growing
        3. Cause chronic respiratory infections > asthma
        4. Unusual shape: stalk for adhesion to epithelial tissue, flexible shape. No cell wall, no peptidoglycan. Not Gram (+) or gram (-). Lost fimbriae. No flagella. No LPS. (Aka lost PAMPs from which they can be detected by the immune system)
        5. Complex nutritional requirements bc they’ve lost biosynthetic and catabolic pathways
           1. Can ferment glucose
           2. No TCA cycle or electron transport chain. Can’t make nucleotides. Must get from home
        6. Only 1000 genes in genome which is advantageous so they don’t need to a copy a larger genome when they’re dividing or make as many proteins when growing
  8. *Legionella pneumophila*
     1. Legionnaire’s Disease
     2. Transferred from aerosolized droplets from contaminated water pools
     3. *Process:* once aerosolized > carried through air > inhaled > taken into lungs > taken in by a macrophage or neutrophil > multiplies within phagocytic via prevention of fusion btwn lysosome and phagosome > necrosis of lung tissue as infection spreads
  9. Tuberculosis
     1. *Statistics:* 9 million clinical cases/yr, 1.5-2 million deaths/yr
     2. *History:* Edward Trudo and Edward Kock found condition got better in cool climates > told ppl to watch coughing and sneezing > womens hemlines went up, men shaved, Kleenex made
     3. *Mycobacteria*
        1. High GC, gram +, Phylum *actinobacteria*, grow slowly
        2. *Have outer membrane made of 2 things*
           1. Polysaccharide backbone
           2. Mycolic acids on the backbone that don’t allow diffusion of molecules into the cell to help evade the immune system and avoid antibiotics
     4. *Mycobacterium tuberculosis*
        1. Causative agent
     5. *3 things can happen once Mycobacterium TB is inhaled*
        1. Abortive infection = immune system is able to clear it and kill all bacteria (does this even occur)
        2. Uncontrolled spread = immunocompromised individuals (disseminated TB)
           1. Bacteria will then spread to bone tissue and kidneys (miliary tuberculosis = spread to many diff places in the lungs and grow lesions in the lungs)
        3. Containment of infection = healthy person (latent-dormant TUB)
           1. Granuloma formation where pus and proteins and immune cells come together around the area of infection to block of infected site > bacteria slowly grows inside granuloma

Tubercle/granuloma = infected immune cells, protein, bacteria surrounded by fibrous tissue

May contain infection for many years

Can calcify and harden or can liquefy and drain and leave cavities where the tubercle once was indicating spreading of bacteria

* + - * 1. If body’s immune system is weakened, infection spreads and symptoms are shown

1. Arthropod-borne microorganisms
   1. *Rickettsia rickettsiafu*
      1. Rocky mountain spotted fever
      2. Via ticks
      3. Invades endothelial cells of BVs
   2. *Borrelia burgdorferi* - Phylum *Spirochetes*
      1. Lyme disease
      2. Via ticks
      3. Pathogens migrate to joints and nervous tissue
      4. *2 term-side effects*
         1. short term – ring-like shape on the skin
         2. long term – 2nd stage includes arthritic feelins, heart inflammation, neurological abnormalities, 3rd stage involves nerve damage