Organism Summary (Bacteria) – Exam I

Gram-Positive Cocci

***#Staphylococcus aureus***

**Characteristics–** Gram positive cocci; catalase +; **coagulase +**; nonmotile; nonspore forming; clumps; β-hemolytic

**Virulence–** Capsule (polysaccharide) = antiphagocytic; Slime layer and Clumping factor (coagulase) = helps bacteria to adhere; Protein A = binds to IgG Fc receptors 🡪 makes difficult to phagocytose opsonized bacteria; Catalase = converts H2O2 🡪 H2O + O2; Coagulase 🡪 fibrin formation = protection against phagocytosis; Hyaluronidases = helps spread of bacteria (by hydrolyzing hyaluronic acids of our EC matrix helping bacteria move through); Staphylokinases = lyse fibrin clots. **Toxins:** Hemolysins (destroy blood cells); transpeptidases = resistant to penicillin; TSST-1 (toxic shock syndrome toxin) = superantigen = binds/holds together TCR-MHC II 🡪 elicits pro-inflammatory cytokine secretion (IL-1, IL-2, IL-6, TNF-α, IFN-γ) 🡪 patient goes into shock; Enterotoxins = heat stable food poisoning; Exfoliative toxins 🡪 skin sloughs off

**Reservoir/trasmission–** Interior nares = nasopharynx; skin;

mayonnaise containing foods (potato salad), custard-filled baked goods 🡪 food poisoning

**Disease/clinical manifestation–** Skin infections (impetigo, cellulitis, wound infections etc); acute endocarditis = heart valve infection; osteomyelitis = bone infection; septic arthritis = joint infection; meningitis; pneumonia (rare); gastroenteritis/food poisoning; scalded skin syndrome = skin peels off (exfoliative toxin); toxic shock syndrome = associated with high-absorbant tampons

**Lab diagnostics–** Gram positive cocci grouped in clusters; β-hemolytic (complete lysis); positive catalase (bubble in presence of H2O2) and coagulase test (clot in presence of serum); positive for protein A; mannitol-salt agar = will ferment mannitol = pH changes 🡪 pH color indicator (phenol red) changes from red to yellow = differentiates *S. aureus* from other *Staph* species

**Treatment–** Often very hard to treat since it may be penicillin resistant. Try methicillin or vancomycin (in MRSA = Methicillin Resistant *Staph. aureus*).

**Exam Keywords:** Coagulase positive; tampon use; potato salad/custard; superantigen; osteomyelitis; skin peeling; MRSA; TSST-1; endocarditis; septic arthritis; protein A; clumping factor; exfoliative toxin

***#Staphylococcus epidermidis***

**Characteristics–** Gram positive cocci; catalase +; coagulase –; nonmotile; nonspore forming; γ-hemolytic

**Virulence–** Capsule (polysaccharide) = antiphagocytic; Slime layer and biofilm; no exotoxins (like *S. aureus*)

**Reservoir/trasmission–** Normal human skin or mucous membrane flora. Transmitted via skin lesion or prosthetic devices.

**Disease/clinical manifestation–** Endocarditis on prosthetic heart valves; prosthetic hip infection; intravascular catheter infection; CSF shunt infection; neonatal sepsis

**Lab diagnostics–** Gram positive cocci; γ-hemolytic (non-hemolytic); positive catalase (bubble in presence of H2O2); negative coagulase test (does not clot in presence of serum); mannitol-salt agar = will NOT ferment mannitol = no color change = remains red (contrast to *S. aureus*); **Novobiocin sensitive** (vs *S. saprophyticus* = novobiocin resistant)

**Treatment–** May be antibiotic resistant. Usually treated with Vancomycin

**Exam Keywords:** Prosthetic devices; non-hemolytic; novobiocin sensitive; normal skin flora;

***#Staphylococcus saprophyticus***

**Characteristics–** Gram positive cocci; catalase +; coagulase –; nonmotile; nonspore forming; γ-hemolytic

**Virulence–** Not discussed

**Reservoir/trasmission–** Not really discussed. Just that it is found in young sexually active females.

**Disease/clinical manifestation–** Leading cause of UTI in newly sexually active females, AKA “honeymoon cystitis”

**Lab diagnostics–** Gram positive cocci; γ-hemolytic (non-hemolytic); positive catalase (bubble in presence of H2O2); negative coagulase test (does not clot in presence of serum); mannitol-salt agar = will NOT ferment mannitol = no color change = remains red (contrast to *S. aureus*); **Novobiocin resistant** (vs *S. epidermidis* = novobiocin sensitive)

**Treatment–** Penicillin

**Exam Keywords:** Newly sexually active female/honeymoon cystitis; novobiocin resistant; γ-hemolytic

***#Sreptococcus pyogenes* (Group A Streptococcus)**

**Characteristics–** Gram positive cocci; catalase – (all streptococci are); non-motile; nonspore forming; facultative anaerobes; grows in chain; **Group A/β-hemolytic *Strep***

**Biology/Virulence–** Hyaluronic capsule = antiphagocytic; F protein = adherence (binding to fibronectin); C5a peptidase = breaks down C5a (complement pathway = chemoattractant of phagocytic cells) = **M-protein** = major virulence factor = inhibits complement activation (no phagocytosis); Streptolysin O = oxygen labile (sensitive) and antigenic (induces immune response), Streptolysin S = oxygen stable and not antigenic = both are hemolysins (lyses blood cells); streptokinase = helps bacterial spread by lysing blood clots. **Toxins:** pyrogenic exotoxins (SPE A, B, C) = act as superantigen 🡪 TSST; erythrogenic toxin = causes the rash of scarlet fever (similar mechanism as TSST)

**Reservoir/trasmission–** Habitats in human pharynx and nares. Transmission is via respiratory droplets (sneezing/coughing)

**Disease/clinical manifestation– Local/exotoxin mediated:** Pharyngitis = inflamed & sore throat/enlarged tonsils with yellowish exudate; scarlet fever = rash (following strep throat) on chest which spreads to extremities; necrotizing fasciitis = flesh eating bacteria; streptococcal toxic shock syndrome. **Antibody mediated:** Rheumatic fever = type II hypersensitivity reaction due to M protein = antibodies to M protein cross-reacts with heart valves (also joints and brain tissue) usually after untreated group A pharyngitis 🡪 fever, rash, carditis, arthritis; acute post-streptococcal glomerulonephritis = antigen-antibody complexes deposit in glomerular basement membrane.

**Lab diagnostics–** Gram stain (positive); β-hemolytic on blood agar; bacitracin sensitive (vs. *S. agalactiae* = bacitracin resistance); PYR = enzyme-reaction (differentiates from other Group A)

**Treatment–** Penicillin or amoxicillin

**Exam Keywords:** Group A *Streptococcus*; scarlet fever; rheumatic fever; sore throat/pharyngitis; M protein; β-hemolytic; necrotizing fasciitis; glomerulonephritis; bacitracin sensitive

***#Sreptococcus agalactiae* (Group B Streptococcus)**

**Characteristics–** Gram positive cocci; catalase – (all streptococci are); non-motile; nonspore forming; facultative anaerobes; grows in chain; **Group B/β-hemolytic *Strep***

**Virulence–** Polysaccharide capsule

**Reservoir/trasmission–** Habitats in human vagina (25% of woman) and is transmitted during birth.

**Disease/clinical manifestation–** Neonatal meningitis, sepsis and pneumonia

**Lab diagnostics–** CAMP reaction = tests for CAMP factor which, in concert of β-lysin, result in enhanced hemolysis (differentiates from Group A, C, G *Streptococcus*); bacitracin resistant (vs *S. pygenes* = bacitracin sensitive)

**Prevention/Treatment–** During 3rd trimester, pregnant mothers are screened for Group B *Streptococcus* and treated

**Exam Keywords:** Group B *Streptococcus*; bacitracin resistant; child-birth; neonatal meningitis; CAMP reaction;

***#Sreptococcus pneumoniae***

**Characteristics–** Gram positive lancet-shaped cocci in pairs (diplococci); catalase – (all streptococci are); non-motile; nonspore forming; facultative anaerobes; **α-hemolytic *Strep***

**Virulence–** Polysaccharide capsule; IgA protease = breaks down IgA (= predominant mucosal antibody) 🡪 prevents bacteria clearance by IgA; pneumolysin = cytotoxin that creates pores in any cell that comes at it (e.g. phagocytes)

**Reservoir/trasmission–** Habitats in human upper respiratory tract (normal flora). Transmission via close contact/respiratory droplets. Also, when viral infection (flu) damages cilia, bacteria can slides down 🡪 pneumonia

**Disease/clinical manifestation–** Pneumonia in adults (usually secondary to viral infection) = typically lobar pneumonia = patient presents with blood-tinged “rusty” sputum (contrast to *Klebsiella* sputum = bright red (jelly) color); meningitis in adults/adolescence; otitis media (ear infection) and sinusitis in children; bacteremia.

**Lab diagnostics–** Gram stain; Quellung reaction = test for capsule (swelling); catalase test = negative; soluble in bile (lyses); optochin sensitive = growth is inhibited (contrast to viridians streptococci = optochin resistant)

**Prevention/Treatment–** not discussed but penicillin and some vaccination available for high risk group.

**Exam Keywords:** α-hemolytic; IgA protease; pneumolysin; otitis media (children); pneumonia/meningitis (adults); secondary to viral infection; rusty sputum; optochin sensitive; bile soluble; diplococcic; no lancefield antigen.

**Viridans Group Streptococci – (e.g. *S. Mutans*)**

**Characteristics–** Gram positive cocci; α-hemolytic; very sticky (sticks to our teeth)

**Virulence–** avirulent but can cause dental caries (decay) and subacute endocarditis

**Reservoir/trasmission–** Habitats in human oropharynx, GI tract, genitourinary tract (normal flora). But can enter bloodstream during dental procedures/gum bleed.

**Disease/clinical manifestation–** Subacute endocarditis (in patients with prosthetic heart valves = bacteria sticks to valves)

**Lab diagnostics–** Gram stain; catalase test = negative; optochin **resistant** (contrast to *S. pneumoniae* = optochin sensitive); does NOT dissolve in bile (vs *S. pneumonia*e = bile soluble); α-hemolytic on blood agar.

**Prevention/Treatment–** Penicillin to prevent endocarditis in patients with prosthetic heart valves before they undergo dental procedures.

**Exam Keywords:** α-hemolytic; teeth decay; subacute endocarditis; sticky; dental procedures in prosthetic heart valve patients; optochin resistant;

Gram-Positive Bacilii

***#Bacillus anthracis***

**Characteristics–** Gram positive rods; spore forming; Aerobic; capsulated; non-motile (other *Bacillus* are motile)

**Virulence–** Polypeptide capsule (**Unique** feature vs polysaccharide capsules in other bacteria) of poly-D-glutamic acid = antiphagocytic

Toxins: Edema factor = causes pulmonary/GI/cutaneous edema (by ↑ intracellular cAMP 🡪 fluid outpouring to extracellular); lethal factor = cause cell lysis; protective antigen = helps the other two to get into cell.

**Reservoir–** Animals (wool), skin, soil

**Transmission–** Skin inoculation; ingesting contaminated meat (most fatal form); inhalation of spores from animal wool/hair

**Disease/clinical manifestation–** Anthrax: Cutaneous = vesicles (blisters) and black eschar (central necrosis); GI = (rare, fatal) vomiting, bloody diarrhea; Pulmonary = (Wool-sorter’s disease) widened mediastinum (due to enlarged mediastinal LN), life-threatening

**Lab diagnostics–** Gram stain, PCR of nasal swab, serologic tests (e.g. ELISA), alert CDC

**Treatment–** Ciprofloxacin, doxycycline. Vaccine available for high risk individuals only.

**Exam Keywords:** Wool-sorter (farmer); postal workers; polypeptide capsule; spore forming; black eschar; widened mediastinum; non-motile

***#Bacillus cereus***

**Characteristics–** Gram positive spore forming rods; Aerobic; non-encapsulated; motile (B. *Anthracis* is encapsulated and non-motile)

**Virulence–** Toxin mediated. Two enterotoxins: **1)** Heat stable = toxin is ingested 🡪 vomiting, nausea, abdominal cramps. **2)** Heat labile (sensitive) = bacteria is ingested = true infection 🡪 ↑cAMP within enterocytes (acts like cholera toxin) 🡪 watery diarrhea

**Reservoir/ Transmission–** Heat stable toxin habitats in grains such as rice and transmitted via fried rice kept warm (e.g. at buffet). Heat labile toxin habitats and transmitted via contaminated meat and sauces

**Disease/clinical manifestation–** Food poisoning = nausea, vomiting, cramps (upper GI). Gastroenteritis (bacterial mediated) = non-bloody diarrhea (lower GI)

**Lab diagnostics–** None

**Treatment–** Not usually treated. In severe situations, vancomycin (not for toxin mediated)

**Exam Keywords:** Fried rice; toxin mediated

***#Corynebacterium diphtheriae***

**Characteristics–** Club-shaped Gram positive rods; non-spore forming; Aerobic.

**Virulence–** Diphtheria toxin = A-B toxin 🡪 inactivates eEF-2 by ADP ribosylation (elongation factor) = inhibits protein synthesis

**Reservoir–** Throat and nasopharynx of asymptomatic humans. **Diphtheroids** = normal flora = do NOT carry diphtheria toxin gene (but have same biological features).

**Transmission–** Respiratory droplets. U.S. immunization program (toxoid vaccine) controls this disease.

**Disease/clinical manifestation–** Sore throat; tough adherent white/gray pseudomembrane; myocarditis; laryngeal nerve palsy; Bull neck

**Lab diagnostics–** CTBA (tellurite blood agar) = gray/black colonies and PCR for tox gene. Or Loeffler’s coagulated serum agar and stain colonies with methylene blue. Elek test = used to test presence of diphtheria toxin (on

**Treatment–** Penicillin or erythromycin (for penicillin allergics); Antitoxin if clinical symptoms are present (myocarditis, laryngeal nerve palsy). DPT vaccine = part of regular tetanus vaccine (includes diphtheria toxoid)

**Exam Keywords:** Patient didn’t receive DPT vaccine (Non U.S. countries); myocarditis; laryngeal nerve paralysis (soft palate/pharynx); airborne droplets transmission; pseudomembrane; Elek test; CTBA plate; Loeffler’s agar

***#Mycobacterium tuberculosis***

**Characteristics–** Gram positive rods but thick outer waxy/lipid layer prevents from Gram staining; acid-fast stain is used instead. Ziehl-Neelson = one of the acid-fast methods; obligate aerobic; grows very slowly; facultative intracellular (usually in macrophages)

**Virulence–** No toxins. Sulfatides 🡪 inhibits phagosome-lysosome fusion (so it can live inside the macrophages). Cord factor (found in the cell wall) inhibits PMN (inflammatory cells) migration

**Reservoir/trasmission–** Habitats in human lungs. Transmitted via respiratory droplets (coughing/sneezing)

**Disease/clinical manifestation–** Causes Tuberculosis. Nonspecific malaise, weight loss, cough, night sweats. Could clear infection but after years it’s reactivated (especially in immunocompromised individuals) = will have granulomas visible on X-rays

**Lab diagnostics–** Auramine-rhodamine stain (green on fluorescence microscopy), and acid-fast stain (Ziehl-Neelson = one of the methods) on sputum to confirm; PPD skin test; grows slow on Lowenstein Jensen (LJ) medium.

**Treatment–** Not discussed for this year

**Exam Keywords:** Sulfatides; Cord factor; acid-fast stain (sputum); catalase (+); Ziehl Neelson; Lowenstein Jensen; airborne droplets; PPD test; granulomas on X-ray; waxy cell wall layer; inhibits phagosome-lysosome fusion

***#Mycobacterium leprae***

**Characteristics–** Gram positive rods but needs acid-fast stain to confirm (due to outer waxy layer); grow requires cooler than body temperatures for growth; obligate intracellular

**Virulence–** She didn’t really discuss.

**Reservoir/trasmission–** Habitats in human mucosa and skin. Also found in Armadillos (Texas, Louisiana) Transmitted via nasal secretion or skin contact

**Disease/clinical manifestation–** Causes leprosy = Hansen’s disease. Two types: Tuberculoid = better immunological response (Th1). Lepromatous = weak immunological response (Th2). Lesions occur in cooler body parts like skin and peripheral nerves.

**Lab diagnostics–** Acid-fast stain of nasal scraping or punch biopsy; Lepromin skin test: positive only in tuberculoid patients and negative in lepromatic patient;

**Treatment–** Not discussed for this year

**Exam Keywords:** Tuberculoid; lepromatous; obligate intracellular; nasal secretion; skin/nerve lesions; cooler than body tempartures; Lepromin skin test; acid-fast stain; Hansen’s disease; Armadillos

Gram Negative

***#Escherichia coli***

**Characteristics–** Gram negative rods; facultative anaerobic; lactose fermernter;

**Virulence–** Pili for attachment to mucosal surfaces; capsule (K1 = neonatal meningitis) = antiphagocytic;

**Reservoir/Transmission–** Found in human/animal colonic normal flora. Transmitted through consuming fecally contaminated food/water, vaginal/urinary tract colonization or undercooked meat (in O157 *E. coli*).

**Disease/clinical manifestation–** Urinary tract infection (UTI) = most common cause = feces contaminates urethra 🡪 pili in *E. coli* adheres and ascends to bladder causing infection also pyelonephritis = when bacteria moves up to kidney via ureters; Neonatal meningitis = due to K1 capsule = transmitted to baby from mom during delivery; septicemia (due to LPS); Gasteroenteritis = 5 different *E. coli* groups:

* **ETEC** (Enterotoxigenic *E. coli*): Two toxins **1)** Heat labile toxin (LT) activates adenylate cyclase 🡪 ↑ cAMP 🡪 watery/traveler’s diarrhea. **2)** Heat stable toxin (ST) activates guanylate cyclase 🡪 watery/traveler’s diarrhea. Acquired through consuming fecally contaminated food/water.
* **EPEC** (Enteropathogenic): Has EAF (E. coli adherence factor) = attaches/effaces mucosa 🡪 inflammatory response 🡪 infantile diarrhea (watery). Acquired through consuming fecally contaminated food/water
* **EAEC** (Enteroaggregative): Biofilm formation/ aggregation = bacteria pile up one on top of each other 🡪 very sticky 🡪 persistent watery diarrhea (several days), traveler’s diarrhea, sporadic diarrhea
* **EHEC** **= O157:H7** (Enterohemorrhagic): Produces shiga-like toxin (verotoxin) 🡪 inhibits 28S rRNA = inactivates 60S ribosome 🡪 protein synthesis inhibited 🡪 cells dying 🡪 bloody diarrhea; also hymolytic-uremic syndrome (HUS) (kidney). Associated with undercooked beef (hamburgers) and unpasteurized fruit juices
* **EIEC** (Enteroinvasive): invades intestinal cells. No toxins. Causes inflammatory dysentery (bloody diarrhea with high amounts of leukocytes in stool)

**Lab diagnostics–** Gram stain; MacConkey agar = pink = ferments lactose; Indole positive = degrades tryptophan (converts tryptophan to indole); EHEC ferments sorbitol;

**Treatment–** No antibiotic for EHEC = prevent spread of shiga toxin.

**Exam Keywords:** lactose fermenter; O157:H7; K1 capsule; infantile diarrhea; traveler’s diarrhea; shiga toxin/ verotoxin; undercooked meat; neonatal meningitis; pili; UTI; indole positive; bloody diarrhea

**Salmonella *– #S. typhi & #S. enteritidis***

**Characteristics–** Gram negative rods; motile & produces H2S (vs. Shigella = non-motile, does not produce H2S); non-lactose fermenters = clear on MacConkey (contrast to *E. coli*); sensitive to acid

**Virulence–** antiphagocytic capsule; intracellular growth/replication; LPS

**Reservoir/Transmission– *S. typhi*** *c*an survive in gallbladder of asymptomatic human carriers and transmit via fecal-oral route. ***S. enteritidis***: dairy products, raw eggs, undercooked contaminated poultry (chicken, turkey) are common reservoirs. Reptiles (turtles) are common carriers, specifically on the shell.

***S. typhi* Disease/clinical manifestation–** cause typhoid fever. Invading M-cells in Peyer’s patches and replicating in endocytic vacuoles 🡪 intestinal wall perforation/ulcer and bleeding. also interferes with lysosome-phagosome fusion (antiphagocytic) = 1-3 weeks after exposure = fever, headache, chills, abdominal pain in RLQ (illeocecal region), infects biliary system (gallbladder, liver)

***S. enteritidis* Disease/clinical manifestation–** nausea, vomiting diarrhea (watery & bloody) = 6-48 hours incubation; most common cause of osteomyelitis in sickle cell disease; septicemia

**Lab diagnostics–** Gram stain; MacConkey agar = clear; Hektoen-enteric agar tests for H2S (light green with black centers = H2S producers = *Salmonella*;

**Treatment–** not discussed

**Exam Keywords:** typhoid fever; dairy products; nonlactose fermenters; motile & produces H2S; invades M-cells; biliary system infection; turtle shell; osteomyelitis in sickle cell; intestinal ulcer/perforation/bleeding

**Shigella (*#S. dysenteriae, #S. sonnei, #S. flexneri, #S. boydii*)**

**Characteristics–** Gram negative rods; nonmotile & does NOT produce H2S (vs. Salmonella = motile, produces H2S); non-lactose fermenters = clear on MacConkey (contrast to *E. coli*)

**Virulence–*Shigella dysenteriae*:** invades M-cells in Peyer’s patches, cause cellular membrane ruffling, replicate in the cytoplasm where *Shigella* will make actin filaments to propel and jump from cell-to-cell. Toxin: Shiga toxin = like EHEC, this toxin will 🡪 inhibit 28S rRNA = inactivate 60S ribosome 🡪 protein synthesis inhibited 🡪 cells dye = necrosis 🡪 bloody diarrhea;

**Reservoir/Transmission–** Humans are the only reservoir. Transmission: fecal-oral via contaminated hands. In the U.S. transmission is seen in children’s daycare.

**Disease/clinical manifestation–** causes enterocolitis/shigellosis = fever, abdominal pain, bloody diarrhea; some cases of hymolytic-uremic syndrome (HUS) where toxin gets into blood 🡪 kidney 🡪 renal failure

**Lab diagnostics–** Gram stain; stool culture; MacConkey agar = clear (nonlactose fermenters); Hektoen-enteric agar tests for H2S (light green with black centers = H2S producers = Salmonella)

does NOT produce H2S; nonmotile

**Treatment–** Antibiotic is available but preferentially not used to prevent the Shiga toxin (and LPS) from being released. Give fluid and electrolyte replacement.

**Exam Keywords:** Shiga toxin; shigellosis; nonmotile & does NOT produce H2S; bloody diarrhea; HUS; daycare

**Yersinia (*#Yersinia pestis, #Yersinia enterocolitica*)**

**Characteristics–** Gram negative rods; *Yersinia enterocolitica* is motile at 25°C (refrigeration temperature) and non-motile at 37°C (body temperature); non-lactose fermenters = clear on MacConkey (contrast to *E. coli*)

**Virulence– *Yersinia pestis*:** facultative intracellular (in lungs) = avoid immune response; coagulase positive (its role is in flea’s gut); antiphagocytic capsule (F1 antigen)

**Reservoir/Transmission–**

* ***Yersinia pestis*:** carried by rats/rodents and transmitted to humans via rodents fleas (bite). Pneumonic plaque is transmitted person-to-person via respiratory droplets
* ***Yersinia enterocolitica:*** carried in livestock, pets and transmitted to humans via contaminated meat, milk, water, pet feces (puppies).

**Disease/clinical manifestation–**

* ***Yersinia pestis*:** bubonic plague = fever, bubos = lymph node swelling in groin/axilla area (bite is usually in lower legs area 🡪 bacteria gets into lymph). Short incubation ~7 days. If not treated 🡪 bacteremia and death. Bacteria can get into blood 🡪 lungs 🡪 alveolar spaces 🡪 pneumonic plaque = fever, malaise, pulmonary signs = highly contagious via respiratory droplets = shorter incubation ~2-3 days. Pneumonic plaque could be via flea bite or person-to-person. Gangrene of the feet is a common sign of *Y. pestis* = black death/plague
* ***Yersinia enterocolitica:*** enterocolitis = diarrhea, fever, abdominal pain for ~1-10 days. Pseudoappendicitis in children = signs and symptoms that mimic appendicitis but it’s not (pain in RLQ, enlarged mesenteric LNs)

**Lab diagnostics–** Gram stain; MacConkey agar = clear (nonlactose fermenters); *Yersinia enterocolitica* is motile at 25°C (refrigeration temperature) and non-motile at 37°C (body temperature)

**Treatment–** not discussed

**Exam Keywords:** grows in colder temperatures; fleas; bubonic plague; pneumonic plague; enlarged groin lymph nodes; pseudoappendicitis; gangrene foot; F1 antigen; coagulase positive

***#Vibrio cholerae***

**Characteristics–** Gram negative curved (comma-shaped) rods; Oxidase (+); polar flagella = motile; able to survive/replicate in contaminated salt water; associated with shellfish consumption (usually undercooked = bacteria survives in shell); susceptible to stomach acid (need to get infected by large dose or organism)

**Virulence–**Pilus = adherence = lysogenic bacteriophage binds 🡪 genes for colera toxin acquired. Pilus will also help adhere to mucosal epithelium (sticks); motility; Cholera toxin = A-B complex (similar mechanism as = keeps α-subunit of G-protein continuously on 🡪 over stimulation of adenylate cyclase 🡪 ↑↑ cAMP 🡪 water outflow into gut lumen. *V. Cholerae* non-O1 = only antiphagocytic capsule (no cholera toxin)

**Reservoir/Transmission–** Transmitted by contaminated water/food; fecal-oral spread

**Disease/clinical manifestation–** Causes cholera: explosive/profuse watery diarrhea; rice-water stool = watery stool containing mucus, no proteins, odorless, colorless feces 🡪 deadly if fluid and electrolyte balance is not restored. *V. Cholerae* non-O1 = no cholera toxin 🡪 mild watery diarrhea and can cause septicemia if gets into bloodstream

**Lab diagnostics–** Grow in salt agar (from stool) and TCBS agar

**Treatment–** Fluid and electrolyte replacement.

**Exam Keywords:** Gram(-) curved rod; Oxidase (+); shellfish; pilus; cholera toxin; A-B complex; cAMP; explosive watery diarrhea; rice-water stool; *V. Cholerae* non-O1; high salinity water; TCBS agar; fecal oral spread

***#Vibrio parahaemolyticus***

**Characteristics–** Same as *V. Cholerae*

**Virulence–**invasion/destruction of colonic epithelium

**Reservoir/Transmission–** Transmitted via consumption of undercooked/raw seafood

**Disease/clinical manifestation–** Most common cause of gastroenteritis in the U.S. of the Vibrio species = similar manifestations as cholera but not as severe= explosive watery diarrhea, vomiting, abdominal cramps, headache, low-grade fever.

**Lab diagnostics–** Grow in salt agar (from stool) and TCBS agar

**Treatment–** Usually self-limited

**Exam Keywords:** Similar charecteristics as *V. Cholerae;* gastroenteritis; undercooked/raw seafood; self-limiting

***#Vibrio vulnificus***

**Characteristics–** Same as *V. Cholerae*

**Virulence–**Antiphagocytic capsule

**Reservoir/Transmission–** Transmitted via consumption of undercooked/raw seafood and swimming/exposure to contaminated seawater (e.g. fisherman)

**Disease/clinical manifestation–** wound infections in skin lesions (e.g. fishing wounds) = cellulitis = severe infections = erythema (skin reddening), pain, bullae formation (fluid-filled sacs), tissue necrosis, septicemia. Spread rapidly. Difficult to treat. High incidence of fatal outcomes

**Lab diagnostics–** Grow in salt agar (from stool) and TCBS agar

**Treatment–** Tetracycline

**Exam Keywords:** Similar charecteristics as *V. Cholerae*; fisherman wound infection; cellulitis

***#Helicobacter pylori***

**Characteristics–** Gram negative spiral rods; polar flagella = highly motile (corkscrew motility); microaerophilic growth; oxidase(+); catalase(+); **urease(+)**

**Virulence–**urease = produces ammonia (from urea) 🡪 neutralizes gastric acid 🡪 bacterial survival in stomach acid; motility = migrates and adheres to gastric epithelial cells (by multiple adhesion proteins); VacA cytotoxin 🡪 inflammation 🡪 ulcer; mucinase = helps bacteria penetrate mucous layer

**Reservoir/Transmission–** Transmitted via oral-oral and fecal-oral contact; can colonize in stomach infected + untreated individual for their lifetime.

**Disease/clinical manifestation–** Gastritis = in the antrum = heartburns, fullness sensation, nausea, vomiting, ↓ acid production = asymptomatic/low level symptoms (often overlooked); peptic ulcer (gastric or duodenal) = found in ~90% of individuals with peptic ulcer disease. Associated with several stomach cancers: gastric adenocarcinoma, MALT cancers, B-cell lymphomas.

**Lab diagnostics–** Biopsy w/ culture; urease breath test = patient swallows carbon labeled urea, if *H. pylori* is present = urease present and patient will exhale the labeled carbon (urea broken down) in CO2, if *H. pylori* is not present, CO2 will not be labeled

**Treatment–** combination of proton pump inhibitor, macrolide, β-lactam drugs. Entire household is usually treated

**Exam Keywords:** urease(+); highly motile; VacA toxin; mucinase; antral gastritis; peptic ulcer; cancers; urease breath test; biopsy;

***#Klebsiella pneumoniae***

**Characteristics–** Gram negative rods; lactose fermenting = pink on MacConkey agar (*E. coli* is the only other one who also ferments lactose); mucoid-appearing colonies; oxidase(–)

**Virulence–**Major = capsule; endotoxin

**Reservoir/Transmission–** Transmitted via respiratory droplets

**Disease/clinical manifestation–** Pneumonia = necrotic destruction of alveolar spaces and forms cavities 🡪 produces blood-tinged “currant-jelly” sputum (especially in alcoholics, diabetics and those with poor lung function); UTI (especially in catheterized patients); speticemia

**Lab diagnostics–** Gram negative; pink on MacConkey agar

**Treatment–** Not discussed

**Exam Keywords:** lactose fermenter; capsule; pneumonia with bloody “currant-jelly” sputum; catheter related UTI

***#Pseudomonas aeruginosa***

**Characteristics–** Gram negative rods; blue/green (pyocyanin/fluorescin) pigment; fruity/grapelike odor; oxidase(+); β-hemolytic

**Virulence–**Alginate capsule = very sticky = protects organism from phagocytosis and antibiotics. Toxins: endotoxin; exotoxin A = inactivates eEF-2 by ADP ribosylation (elongation factor) = inhibits protein synthesis (similar to diphtheria toxin)

**Reservoir/Transmission–** Ubiquitous in environment and water; nosocomial spread via water reservoir

**Disease/clinical manifestation–** Hot tub folliculitis (infection) = bacteria gets in the follicles 🡪 red skin bumps usually from waist down; pneumonia/lung infections seen in cystic fibrosis patients (neutropenia or other chronic lung disease); endocarditis; UTI (mainly in hospital patients with long-term urinary catheters); eye infections; burn wound infection (mediated by exotoxin A) 🡪 septicemia; osteomyelitis; bacteremia

**Lab diagnostics–** Gram stain 🡪 pink; blue-green pigmentation; grapelike odor; β-hemolytic

**Treatment–** Different treatments based on antibiotic resistance

**Exam Keywords:** blue-green (pyocyanin-fluorescin) pigmentation; grapelike/fruity odor; exotoxin A; sticky capsule; hot tub folliculitis; pneumonia in CF; burn wound infection; inhibits eEF-2; β-hemolytic

***#Haemophilus influenza***

**Characteristics–** Gram negative rods; requires Factor X (heme) and Factor V (NAD) for growth (blood containing medium = chocolate agar); two types = type B/encapsulated and nontypeable/nonencapsulated

**Virulence–**Capsule (only type B) that contains polyribitol phosphate (PRP) which is used in vaccine; pili = mediates adherence/colonization of the oropharynx; LPS; IgA proteases

**Reservoir/Transmission–** Colonization of upper respiratory tract in all humans = nontypeable.

**Disease/clinical manifestation–** Type B causes: infantile/pediatric meningitis, pediatric epiglittitis = pharyngitis, fever, difficulty breathing as epiglottis swells (primarily in unimmunized children); Otitis media, septic arthritis (not most common cause. *S. aureus* and *N. gonorrhoeae* are most common cause), bronchitis and pneumonia = can be caused by both type B and nontypeable

**Lab diagnostics–** Blood and CSF 🡪 Gram negative rods. Grow on chocolate agar to provide factors X (hemin) and V (NAD).

- Satellite phenomenon boils down to the following: *H. influenza* needs blood products heme and NAD. *S. aureus* is a β-hemolytic = breaks down blood 🡪 releases heme (X factor) and NAD (V factor). So grow *S. aureus* in a blood agar together with *H. influenza* 🡪 *S. aureus* provides all the necessary factors for *H. influenza* growth. *H. influenza* is detected as small white dots growing around big white *S. aureus* colonies = hence the name satellite = another diagnostic tool to identify *H. influenza*.

**Treatment–** Hib vaccine at 2, 4, 6 and 15 months

**Exam Keywords:** Factor X and V; chocolate agar; type B/nontypeable; PRP capsule; IgA proteases; pediatric meningitis/epiglittitis; otitis media; satellite phenomenon; Hib vaccine

***#Haemophilus ducreyi***

**Characteristics–** Gram negative rods; requires Factor X (heme) but NOT Factor V (NAD for growth (vs *H. influenza*)

**Virulence–**Pili that adheres to genital and perianal mucosa

**Reservoir/Transmission–** Transmitted via sexual and direct contact

**Disease/clinical manifestation–** Sexually transmitted disease chancroid = painful genital ulcer (contrast to syphilis which is not painful) with or without painful inguinal buboes (lymphadenopathy).

**Lab diagnostics–** Gram stain and/or DNA probes (PCR)

**Treatment–** Not discussed

**Exam Keywords:** Factor X but not V required; chancroid = painful genital unlcer; painful buboes; STD

Spirochetes

***#Treponema pallidum***

**Characteristics–** Gram negative thin coiled spirochetes; anaerobic; motile

**Virulence–**No major virulence factors just induces host’s body immune response

**Reservoir/Transmission–** Sexually transmitted and also transmits through placenta

**Disease/clinical manifestation–** Causes syphilis = 3rd most common sexually transmitted disease in the U.S.

* Primary stage: **painless** chancres (skin ulcers) and non tender regional buboes. Highly infectious;
* Secondary stage: disseminated rash = generalized mucocutaneous rash; chondylomata lata = highly infectious painless wartlike lesion
* Latent stage: asymptomatic between secondary and tertiary stage = may be in this stage for years
* Tertiary stage: Gummas 🡪 granulomatous lesions; cardiovascular syphilis 🡪 aortic aneurysms; neurosyphilis 🡪 tabes dorsalis = dorsal column destruction. There are also different presentations of neurosyphilis; Argyll-Robertson pupil = does not react to light but constricts during accommodation (near vision)

Congenital syphilis (crosses placenta): born asymptomatic, may have runny nose (rhinitis), rash, chondylomata lata, Hutchinson’s teeth (central notch in upper incisor teeth and widely spaced)

**Lab diagnostics–** Dark field microscopy (since it’s too thin to be seen with Gram stain) = only used for primary stage syphilis; nonspecific treponemal test = tests for cardiolipin hence why it’s nonspecific (since mitochondria can also produce cardiolipin in nontreponemal individuals) = VDRL and RPR tests (also ART and ICE)= good for primary and secondary degree syphilis; specific treponemal tests = FTA-ABS for treponemal antibodies = good for all stages but mainly used for third stage syphilis

**Treatment–** Penicillin

**Exam Keywords:** **painless** chancres, rash, chondylomata lata, gummas, tabes dorsalis; crosses placenta; dark field microscopy; nontreponemal test for cardiolipin = VDRL and RPR; specific treponemal test = FTA-ABS

***#Borrelia burgdorferi***

**Characteristics–** Gram negative spirochetes (larger than *Treponema*); microaerophilic; twisting motility;

**Virulence–**cell mediated inflammatory responses

**Reservoir/Transmission–** Habitats in white-footed mice and white-tailed deers. Transmitted by *Ixodes* ticks = vector = Northwest (Connecticut) and Midwest (Wisconsin) usually during spring/summer months

**Disease/clinical manifestation–** Causes lyme disease:

* Early Localized Stage: (3-30 day incubation period) erythema migrans 🡪 “bulls eye” rash at tick bite site along with Flulike illness
* Early Disseminated Stage: (weeks-months after first stage) involves skin = lesions (smaller and more than first stage); neurologic = aseptic meningitis and 7th cranial nerve palsy (Bell’s palsy = facial nerve palsy); cardiac abnormalities; joint and muscle pain
* Late Stage (months-years later): chronic arthritis usually in larger joints like knee. Can also lead to chronic neurologic damage (e.g. memory impairment)

**Lab diagnostics–** Diagnostics are vague/difficult but serologic tests for antibodies IFA, EIA, ELISA, western blot

**Treatment–** Earlier stages treat with penicillin family antibiotic. Later on with doxycycline

**Exam Keywords:** lyme disease; *Ixodes* ticks; bulls eye rash (erythema migrans); Bells palsy; arthritis

***#Borrelia recurrentis***

**Characteristics–** Gram negative spirochetes (larger than *Treponema*); microaerophilic; twisting motility;

**Virulence–**Antigenic variation

**Reservoir/Transmission–** Transmitted via body louse (*Pediculus humanus*)

**Disease/clinical manifestation–** Causes relapsing fever: fever, chills, headaches, muscle aches. After a couple days the symptoms go away and then about a week later it reappears hence the name relapsing fever. The key to relapsing fever is *Borrelia*’s ability to change its surface antigens/proteins = antigenic variation 🡪 previously created antibodies are not effective for the new antigen 🡪 relapses

**Lab diagnostics–** Not discussed

**Treatment–** Erythromycin or doxycycline

**Exam Keywords:** relapsing fever; antigenic variation

***#Leptospira interrogans***

**Characteristics–** Long thin aerobic spirochetes

**Virulence–**Not discussed

**Reservoir/Transmission–** Found in animal urine (dogs, rats, livestock, wild animals). Transmitted via abraded skin and mucus membranes or swallowing contaminated water during swimming (Hawaii has highest incidence)

**Disease/clinical manifestation–** Two phases

* **First phase** (septicemic): Mild: fever, headache, abdominal pain, red conjunctiva (suffusion). Severe: jaundice, hemorrhage, myocarditis, renal failure
* **Second phase** (immune): meningitis, skin rash,

**Lab diagnostics–** Culture, serology, dark microscopy

**Treatment–** Erythromycin or doxycycline

**Exam Keywords:** animal urine; swallowing contaminated swimming water (Hawaii)

Anaerobes

***#Clostridium perfringens***

**Characteristics–** Gram positive large, boxcar-shaped rods; spore forming; anaerobic; ubiquitous; non-motile

**Virulence–**Alpha toxin has phospholipase C activity= lyses blood cells 🡪 increases vascular permeability; Degradative enzymes such as hyaluronidase and collagenase 🡪 tissue damage; heat-labile exnterotoxin 🡪 watery diarrhea

**Reservoir/Transmission–** Spores habitat in soil and food. Transmitted via wound (battlefield) or food.

**Disease/clinical manifestation– Gas gangrene/ myonecrosis** **(wound infection):** tissue necrosis involving muscle (deep since it’s anaerobic). Bacteria produces gas as a metabolic byproduct 🡪 crackling consistency upon palpitation due to pockets of gas = crepitus. Fatal if not treated.

**–Diarrheal illness (food poisoning):** ingesting food contaminated with spores (poultry, meat) 🡪 toxin production in the gut 🡪 watery diarrhea, or in a more severe case bloody diarrhea (due to hemorrhagic jejunal necrosis)

**Lab diagnostics–** Gram stain 🡪 Gram(+) boxcar shaped rods; grow in anaerobic conditions

**Treatment–** Penicillin, clindamycin; hyperbaric oxygen chamber; surgical debridement

**Exam Keywords:** Alpha toxin; blood cell lysis; watery (to bloody) diarrhea; boxcar-shaped; gas gangrene; crepitus

***#Clostridium tetani***

**Characteristics–** Gram positive small, tennis racket rods; anaerobic; ubiquitous; spore-forming; motile

**Virulence–**Tetanospasmin = carried to CNS intra-axonal via retrograde transport 🡪 blocks release of inhibitory neurotransmitters (e.g. GABA, glycine) 🡪 excitatory are unopposed = sustained muscle contraction.

**Reservoir/Transmission–** Spores through stab/puncture wound (e.g. rusty nail)

**Disease/clinical manifestation–** Causes tetanus = severe muscle spasms 🡪 lockjaw, risus sardonicus (grinning expression due facial muscles spasm), back arching in infants. Once stage of lockjaw is reached 🡪 high mortality

**Lab diagnostics–** Gram stain 🡪 Gram(+) tennis racket rods; grow in strict anaerobic conditions;

**Treatment–** DPT vaccination with toxoid. Supportive care include antitoxin = passive immunization with human tetanus immune globulin (anti-tetanus antibodies); primary wound debridement; penicillin.

**Exam Keywords:** Tetanospasmin; blockage of inhibitory neurotransmitter; rusty nail wound; tetanus; lockjaw; risus sardonicus; DPT vaccination; anti-toxin; muscle spasms

***#Clostridium botulinum***

**Characteristics–** Gram positive; anaerobic; spore-forming; motile

**Virulence–**Neurotoxin = blocks release of ACh 🡪 flaccid paralysis

**Reservoir/Transmission–** Found in soil and dust. Transmitted via wounds or food (canned-food, honey)

**Disease/clinical manifestation–** Causes botulism:

* Food poisoning (adult form) is associated with improperly sterilized canned food 🡪 flaccid paralysis, dizziness, double vision, weakness, respiratory paralysis.
* Wound botulism is more localized rather than systemic.
* Infant botulism is associated with contaminated honey 🡪 flaccid paralysis = floppy baby syndrome

**Lab diagnostics–** Gram stain 🡪 Gram(+); grow in anaerobic conditions; immunoassay of toxin in food, stool, serum

**Treatment–** Antitoxin; respiratory support; wound debridement; antibiotics

**Exam Keywords:** Canned food; botulism; flaccid paralysis; inhibits Ach release; honey; “floppy” baby;

***#Clostridium difficile***

**Characteristics–** Gram positive; anaerobic; spore-forming; motile;

**Virulence–**Antibiotic resistant (difficult to treat). Enterotoxin A 🡪 diarrhea by damaging mucosa = blowing cells up 🡪 fluid increase. Cytotoxin B = cytotoxic to colonic epithelial cells. Together Toxin A and B leads to cell death, inflammation 🡪 antibiotic-associated diarrhea

**Reservoir/Transmission–** Part of normal flora in some individuals but is kept in-check by normal intestinal flora but when put on broad spectrum antibiotic, normal flora dies and *C. difficile* is free to superinfect the colon

**Disease/clinical manifestation–** Pseudomembranous colitis = antibiotic-associated diarrhea = severe foul smelling diarrhea, cramps and fever.

**Lab diagnostics–** Colonoscopy will show pseudomembrane plaques; clinical symptoms (severe diarrhea after being treated with antibiotics); immunoassay for *C. difficile* toxin in stool.

**Treatment–** Terminate responsible antibiotics (to reestablish normal flora); metronidazole or vancomycin

**Exam Keywords:** Toxin A; antibiotic-associated diarrhea; pseudomembranous colitis; normal flora; pseudomembrane plaques; colonoscopy; metronidazole

***#Actinomyces israelii***

**Characteristics–** Gram positive branching rods; anaerobic; filamentous

**Virulence–**Not discussed

**Reservoir/Transmission–** Found in normal mouth, GI and vagina flora. Endogenous spread across mucosal barriers after trauma or disease

**Disease/clinical manifestation–** Forms sulfur granules, tissue swelling 🡪 sinus tracts with draining abscesses = invasive. The abscesses causes lumpy jaw (cervicofacial disease), pulmonary (thick gooey sputum), abdominal and pelvic actinomycosis = can be very painful, cellulitic and chronic infection that gets through tissue boundaries.

**Lab diagnostics–** Gram (+); anaerobic; yellow sulfur granules; culture forms “molar tooth” appearing colony on agar

**Treatment–** Surgical drainage of abscesses; penicillin G or ampicillin

**Exam Keywords:** sulfur granules; lumpy jaw; draining sinus tracts; molar tooth appearance; normal flora

***#Bacteroides fragilis***

**Characteristics–** Gram negative rod; anaerobic;

**Virulence–**Antiphagocytic capsule; modified LPS (no lipid A) = weakened endotoxin activity

**Reservoir/Transmission–** Part of normal flora (prominent in colon), but infects when trauma (e.g. GSW, stab wound) or surgery (perforated bowel) tears through the intestine 🡪 endogenous spread

**Disease/clinical manifestation–** Intraabdominal and pelvic infections, bacteremia and abscesses.

**Lab diagnostics–** Gram (-); anaerobic;

**Treatment–** Surgical drainage of abscesses; treat with clindamycin and metronidazole

**Exam Keywords:** Gun shot wound; perforated bowel; bacteremia; endogenous spread; weakened LPS; abscesses

Intracellular Bacteria

***#Rikettsia rikettsii***

**Characteristics (same for all *Rikettsia*)–** Small Gram negative (stain poorly) obligate intracellular requires arthropod vector; requires host’s ATP for energy (as well as *Chlamydia*)

**Virulence–**Not discussed

**Reservoir/Transmission–** Found in dogs and wild rodents. Transmitted via *Dermacentor* wood or dog ticks

**Disease/clinical manifestation–** Rocky Mountain Spotted Fever: infects endothelial cells in small blood vessels 🡪 small hemorrhages & throbi 🡪 rash that starts out on wrists, ankles, soles and palms and becomes more generalized (trunk) later on; high fever, conjunctival redness, severe headache, malaise, vomiting, confusion, muscle aching. Meningitis is usually suspected initially but the spotted rash then stands out

**Lab diagnostics (same for all *Rikettsia*)–** Fluorescein-labeled antibodies; PCR assays; Weil-Felix test = infected body produces *Rikettsia* antibodies that also cross react *Proteus vulgaris*’s antigens which can be easily grown in lab

**Treatment (same for all *Rikettsia*)–** First line of antibiotics: doxycycline. Chloramphenicol can be added

**Exam Keywords:** Rocky Mountain spotted fever; Weil-Felix positive; spotted rash; *Dermacentor* ticks; host’s ATP

***#Rikettsia prowazekii***

**Characteristics (same for all *Rikettsia*)–** Small Gram negative (stain poorly) obligate intracellular requires arthropod vector; requires host’s ATP for energy (as well as *Chlamydia*)

**Virulence–**Not discussed

**Reservoir/Transmission–** Reservoir is flying squirrels and untreated humans. Transmitted via vector = *Pediculus* louse

**Disease/clinical manifestation– Epi**demic Typhus = fever, headache, rash that spreads from trunk out (no palms, soles and face = contrast to *rikettsii*). Brill-Zinsser Disease: occurs in patients who recover from the louse-borne typhus without antibiotic 🡪 intracellular bacteria is latent and occasionally it breaks out with milder symptoms with no rash due to preformed antibodies from original infection. They now serve as reservoirs to this bacterium.

**Lab diagnostics (same for all *Rikettsia*)–** Fluorescein-labeled antibodies; PCR assays; Weil-Felix test = positive (infected body produces *Rikettsia* antibodies that also cross react *Proteus vulgaris*’s antigens which can be easily grown in lab)

**Treatment (same for all *Rikettsia*)–** First line of antibiotics: doxycycline. Chloramphenicol can be added

**Exam Keywords:** Flying squirrels/untreated humans; epidemic typhus; Brill-Zinsser disease; spreads from trunk out; human *Pediculus* body louse

Weil-Felix positive;

***#Rikettsia typhi***

**Characteristics (same for all *Rikettsia*)–** Small Gram negative (stain poorly) obligate intracellular requires arthropod vector; requires host’s ATP for energy (as well as *Chlamydia*)

**Virulence–**Not discussed

**Reservoir/Transmission–** Reservoir are rodents. Transmitted via vector = *Xenopsylla* flea (rat flea)

**Disease/clinical manifestation– En**demic Typhus = same symptoms as epidemic but not as severe = fever, headache and rash that spreads from trunk out (no palms, soles and face = contrast to *Rikettsia* *rikettsii*).

**Lab diagnostics (same for all *Rikettsia*)–** Fluorescein-labeled antibodies; PCR assays; Weil-Felix test = positive (infected body produces *Rikettsia* antibodies that also cross react *Proteus vulgaris*’s antigens which can be easily grown in lab)

**Treatment (same for all *Rikettsia*)–** First line of antibiotics: doxycycline. Chloramphenicol can be added

**Exam Keywords:** *Xenopsylla* flea (rat flea); **En**demic Typhus; Rodents; spreads from trunk out

***#Chlamydia trachomatis***

**Characteristics–** Tiny Gram negative; LACKS peptidoglycan layer; obligate intracellular (cannot produce its own ATP);

* Unique developmental cycle 🡪 can exist in 2 forms: Elementary bodies (**EB**) = metabolically inactive, resists harsh environments 🡪 infectious form (**E**nfectious). Reticulate bodies (**RB**) = metabolically active (**R**eplicates) 🡪 noninfectious. Note, they can switch from RB to EB (when nutrition are gone) infect another cell and go back to RB.

**Virulence–**Not discussed

**Reservoir/Transmission–** Found in human genital tract and eyes. Transmitted via sexual contact and mom-to-baby at birth. EBs also transmits from humans with infected eye secretions via flies.

**Disease/clinical manifestation–** Most common STD in the U.S.; Leading cause of preventable blindness

* STDs: **Nongonococcal urethritis (NGU)** = infection of the urethra that is not caused by *N. gonorrheae*, but patient can be infected by both organisms at the same time. Manifestation is mucopurulent discharge. How is the responsible organism determined? Well, after a successful gonorrhea treatment (penicillin) and urethritis persists (since *Chlamydia* lacks peptidoglycan layer, where penicillin works).
* **Cervicitis and Pelvic Inflammatory Disease (PID)**
* **Reiter’s syndrome**: urethritis, conjunctivitis, polyarthritis and mucocutaneous lesions that usually occurs in young caucasian men and is an HLA-B27 related illness.
* **Trachoma**: chronic conjunctivitis that is the leading cause of preventable blindness is the world. Inflammatory that leads to corneal abrasion/scarring and ulceration 🡪 blindness.
* **Fitz-Hugh-Curtis syndrome**: infection of the liver capsule (perihepatitis)
* **Lymphogranuloma venereum**: starts with a painless ulcer at infection site that heals spontaneously and follows inflammation and swelling of regional lymph nodes that drains that area. Genital elephantiasis can also develop. (Different than syphilis in that primary syphilis is followed by mucocutaneous phase whereas *Chlamydia* is followed by inflammation/swelling of lymph nodes).
* **Inclusion conjunctivitis** = swelling and discharges in babies. Aspiration of bacteria 🡪 **Infant Pneumonia**. They are both usually contracted at birth

**Lab diagnostics–** Cannot be Gram stained due to absence of peptidoglycan. Usually diagnosed based on clinical symptoms. But can also use PCR, immunofluorescence for EBs.

**Treatment–** Erythromycin, Azithromycin and in adults also with doxycycline. Given to all newborn babies in eyedrops at birth.

**Exam Keywords:** STD; no peptidoglycan; inclusion conjunctivitis; EB and RB; infant pneumonia; liver capsule infection; trachoma; lymphogranuloma venereum; PID; Reiter syndrome; NGU; Fitz-Hugh-Curtis syndrome;

***#Mycoplasma pneumoniae***

**Characteristics–** Smallest free-living bacteria; requires intracellular growth; NO cell wall = NO peptidoglycan; obligate aerobe; no definitive shape (rod or cocci) due to absence of cell wall; cell membrane is packed with cholesterol (sterols) to add strength (hence needs cholesterol for growth)

**Virulence–**P1 adhesin 🡪 adheres to respiratory epithelium 🡪 inhibits cilia in ciliated epithelial cells 🡪 ciliated cells die and slough off.

**Reservoir/Transmission–** Strict human pathogen. Transmitted via respiratory droplets

**Disease/clinical manifestation–** Mainly infects children ages 5-15 years old. Starts out as a tracheobronchitis = inflammation of bronchi marked by nonproductive cough (due to damaged cilia), fever and sore throat. Some patients may be able to fight off infection and heal while others may develop an atypical “walking” pneumonia = dry nonproductive hacking cough, low-grade fever, headache, myaglia (muscle pain)

**Lab diagnostics–** Has “fried egg” appearance on Eaton agar. Mulberry-shaped colonies on sterol-containing agars. Grow on agars that contains sterol. Can also PCR of sputum. Cold agglutinins (antibodies bind RBC’s surface antigens). X-ray will show patchy infiltrates that look worse than physical exam

**Treatment–** Cannot treat with antibiotics that target bacterial cell wall (penicillin, cephalosporins). Treat with macrolides = erythromycin, azithromycin (work on ribosome)

**Exam Keywords:** P1 adhesin; NO cell wall; cell membrane with↑ cholesterol content; atypical “walking” pneumonia; nonproductive cough; cilia; macrolides; cold agglutinins; “fried egg”; bronchitis

***#Ehrlichia* & *Anaplasma***

**Characteristics–** Gram negative rod; obligate intracellular; *Ehrlichia* grow in monocytes, *Anaplasma* grow in granulocytes (WBCs);

**Virulence–**Not discussed

**Reservoir/Transmission–** Both are transmitted through a vector: *Ehrlichia* via lone star tick (*Amblyomma*) and *Anaplasma* via *Ixodes* tick

**Disease/clinical manifestation–** Monocytic ehrlichiosis and Anaplasmosis. They both have similar manifestations with some minor differences: high fever, rash (40% in Monocytic ehrlichiosis and 10% in Anaplasmosis) leukopenia (↓ WBC count), thrombocytopenia, ↑ serum transaminases. With Anaplasmosis there are more severe complications such as peripheral neuropathies. Can be confused with Rocky Mountain spotted fever (*R. rikettsii*) with similar history (tick bite) and somewhat similar manifestation (except rash may not be as common as with *Rikettsia*) but treatment is identical for both (doxycycline) so misdiagnosis has no affect.

**Lab diagnostics–** Giemsa stain; serology

**Treatment–** Doxycycline

**Exam Keywords:** Ehrlichiosis/Anaplasmosis; lone star tick bite; *Ixodes* tick bite; Giemsa stain; doxycycline