## Bacteria Chart: Toxicogenic bacteria

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Corynebacterium Diphtheriae | Diphtheria | Gm+, pleomorphic<br>Telluride medium<br>No (γ) hemolysis<br>Small colonies<br>"Chinese characters" | Respiratory diphtheria: by respiratory droplets and skin carriers<br>Pathogenesis: Non-invasive, produce toxin; toxin can result in myocarditis, paralysis due to myelin degeneration, and lethargy<br>Epidemiology: ↓↓ since 1970s, but has ↑ recently due to ↓ immunizations. | Toxin: phage-encoded, normally repressed by toxin repressor; low iron results in ↓ repressor (toxin production).<br>Receptor: EGF precursor prot.<br>Target: Elongation factor-2, by **ADP-ribosylation** (i.e. shuts down protein synthesis) | Dx: Telluride media<br>Rx: Inactivate toxin by antibody (grown In horses).<br>DPT Vaccine used for prevention |
| Pseudomonas aeruginosa | **True opportunist**; Bone & joint Infections; **meningitis**, otitis externa (swimmer's ear), otitis media, keratitis, enterocolitis, endocarditis, **pneumonia**, UTIs, septic shock | Gm- rod, oxidase+, β-hemolytic, lactose non-fermenting; **Blue-green hue** when grown on blood agar | Transmission and successful infection often occur in hospitalized patients and the immunocompromised; examples are burn patients, cystic fibrosis patients, and infection by catheters | Pilin: Adhesins assist in colonization<br>Alginate slime layer: Especially in patients with cystic fibrosis<br>Exotoxin A: Targets EF-2<br>** **Iron-regulated**<br>Elastase | Dx: API strips; easy to culture<br>Rx: fluoroquinolones & later aminoglycosides, due to resistance against penicillin, ampicillin, cephalosporins, chloramphenicol |
| Vibrio cholerae | Cholera | Small Gm- curved rod, polar flagellum, **lives in $H_2O$**; oxidase +; grows in **TCBS agar** & turns color from blue to yellow | Transmission by contaminated food/water, flies, water contaminated with human feces<br>Pathogenesis: Colonization in **mucosal intestinal tract**; production of cholera toxin results in massive loss of fluid and electrolytes (i.e. the worst diarrhea known worldwide)<br>- Day 1: 14 L<br>- Day 2: 8 L<br>- Day 3: 5 L<br>- Day 4: 2 L<br>- Day 5: 1 L | Cholera toxin: Complex A-B type; ctxA portion of cholera operon's ribosome-binding site is 5x less efficient than the ribosome-binding site of ctxB. **Toxin works by ADP-ribosylation of $G_S$ subunit so that it can't be hydrolyzed, keeping adenylate cyclase active and ↑↑↑ cAMP.** This increase alters pump activities in intestinal cells; NaCl, which is normally absorbed by cells, is not; water follows NaCl, which results in diarrhea<br>Receptor: $GM_1$ ganglioside<br>gal---gal---gal---glc------HOST<br>NANA<br>Sialic acid<br>**Note**: Neuraminidase cleaves off sialic acid side chains, save the one that is in the receptor; this ↑↑ cells with binding sites. | Dx: Clinical sympt., stool sample culture<br>Rx: Rehydration plus one of the following: Sulfamethoxazole/ trimethoprim; fluoroquinolone; tetracycline<br>Prevention: Water examination for contaminated water or ice, vaccine also may be indicated for certain situations. Avoidance of eating raw/undercooked shellfish is also advisable. |
| Bordatella pertussis | Whooping cough | Gm- coccobacillus (rod); **obligate human parasite, grows in the area between the airway and the lung; not invasive** | Transmission: by droplet nuclei; usually infects middle respiratory tract<br>Pathogenesis: Catarrhal stage (profuse, mucoid rhinorrhea) for 1-2 weeks; **paroxysmal stage** (severe, repeated coughing) for 2-4 weeks; convalescent stage (fading of symptoms) for 3-4 weeks. | Adhesins: filamentous hemagglutinin; pertussis toxin<br>Pertussis Toxin: Complex A-B; A: S1, B: S2, S3, 2xS4, S5 Works by ADP-ribosylation of $G_I$ subunit, ↓ inhibition of adenylate cyclase, ↑↑↑ cAMP.<br>**Note**: Targets ciliated lung cells<br>Tracheal cytotoxin: injures respiratory epithelium<br>Invasive bacterial adenylate Cyclase: requires calmodulin to work; possible anti-phagocytic activity by interfering with chemotaxis and superoxide formation by PMNs. | Dx: Cllinical sympt., Culture on special charcoal-blood agar<br>Rx: Supportive treatment, also can use erythromycin or chloramphenicol; $O_2$ available may prevent death<br>Prevention: DPT vaccination |
| Clostridium tetani | Tetanus (lockjaw) | Gm+ slender rods<br>Strict anaerobe;<br>Spore-formers | Spores enter host by splinter, rusty nail, or injection site<br>**Does not invade**, spores germinate and multiply locally; some bacteria autolyze, releasing tetanus toxin.<br>Toxin enters bloodstream, affects CNS. | Toxin: blocks release of inhibitors of neural transmission, resulting in **spastic paralysis** ("lockjaw") due to prolonged neuronal firing | Dx: Clinical sympt.<br>Rx: Support, by keeping patient away from stimuli (i.e. dark room); also use antitoxin (made from volunteers); then, use antibiotics such as penicillin G or metronidazole to remove the bacteria |

## Bacteria Chart: Toxicogenic bacteria (continued)

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Clostridium botulinum | Botulism | Gm+ slender rods; Strict anaerobe; Spore-formers | Food-borne botulism: by cans; toxin is produced in food, food is ingested and makes its way to the bloodstream, causing **flaccid paralysis**<br>Wound botulism: by introduction to wound Bacteria multiply, toxin made, toxin enters bloodstream, again…flaccid paralysis<br>Infant botulism: by ingesting honey; b/c C. botulinum spores will germinate and produce toxin…then toxin enters bloodstream, resulting in flaccid paralysis | Toxin: A-B type, prevents acetylcholine (ACh) release<br>Receptor: Gangliosides at neuromuscular junction | Dx: Clinical sympt., stool assays<br>Rx: Emptying patient's stomach to remove source of toxin; then giving antitoxin; if flaccid paralysis has already set in, use of respirators may be necessary |
| Clostridium perferingens | Gas gangrene | Gm+ thicker rods; Strict anaerobe; Spore-formers | Enters by wounds; gases ($CO_2$, $H_2$) formed by fermentation of muscle carbohydrates; wounds are usually dirty wounds with a region of reduced redox potential | Toxins: Enterotoxin, lecithinase, DNase, collagenase, hyaluronidase, proteases<br>**Key toxin**: α-toxin, which is membrane-disrupting – it's a phospholipase specific for lecithin. Toxin moves along muscle, destroying cells and causing necrosis, shock, and bacteremia. | Dx: **Clinical** sympt.; gas can also be palpated and seen on X-ray and cultures taken, but this takes too long.<br>Rx: Excision of all infected regions; penicillin used right away<br>Prevention: Debridement of severe wounds |
| Bacillus cereus | Food poisoning, resulting in diarrhea and/or vomiting | Gm+ large rods; spore formers; can grow in a wide variety of temps. | Usually found in **fried rice** left at room temperature; bacteria multiphy in GI tract and produce enterotoxin, causing symptoms | 2 toxins:<br>- Causes diarrhea by ↑ cAMP<br>- Induces vomiting (heat-stable enterotoxin is an emetic) | Dx: Clinical sympt.<br>Rx: Monitoring of fluid loss, and replenishment of fluid loss as needed<br>**NO Antibiotics!** |
| Helicobacter pylori | Chronic gastritis, **Gastric ulcers**<br>Possibly stomach cancer-causing | Gm- curved rods; Mult. polar flagella Catalase+, Oxidase+, Urease+; weakly hemolytic | **Does not invade the mucosa**, but produces substances eroding the mucosa. This can eventually result in ulcers. | Toxins:<br>- Adhesins for colonization<br>- Urease to lower pH and promote inflammation of the gastric mucosa by producing ammonia ($NH_3$)<br>- Flagella for motility<br>- **Vacuolating cytotoxin**, whose function isn't well described | Dx: Biopsy, culture, antibody tests, urea breath test<br>Rx: Combination of clarithromycin and, omeprazole (pump inhibitor)<br>**Note**: The goal is to eliminate all bacteria, so follow-up tests must be done to ensure this is the case! |
| Listeria monocytogenes | GI infections, UTIs<br>Causes premature delivery of an infected infant, meningitis in neonates | Gm+; cultured on chocolate agar; multiple flagella when grown at 20-25°C; β-hemolytic & catalase+; ferments glucose, trehalose; **tumbling motility at 4°C** | Found in prepared foods of all kinds; can be found in improperly processed milk products; grows at 4°C.<br>Adults: Influenza-like symptoms<br>Pregnant women: stillbirth, bacetemia, neonatal infections<br>Pathogenesis: **Cell-to-cell spread using host actin to propel itself**<br>- Listeria phagocytosed, use listeriolysin O to escape phagocytic vacuole (Listeria can live in/infect macrophages)<br>- Listeria gathers host actin and uses it to propel itself and make a pseudopod to the next cell it infects. | Virulence factor: ability to mobilize iron from human transferrin<br>Listeriolysin O is definitely a virulence factor also. | Dx: History; patients that are elderly, immunocompromised or pregnant are more susceptible to listeriosis; it can also be cultured in patients who develop meningitis as a result, on sheep agar or CNA agar.<br>Rx: Susceptible patients w/ampicillin or sulfa/trimethoprim. In immunocompetent adults, the disease is usually self-limiting. |
| **Escherichia coli: See enteric bacteria section** | | | | | |

## Bacteria Chart: Staphylococcus species

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Staphylococcus aureus | Nosocomial inf., food-borne disease, folliculitis, furuncles (boils), carbuncles, impetigo, pneumonia, osteomyelitis, scalded skin syndrome, toxic shock synd. (TSS), endocarditis | Gm+ coccus, grows in grape-like clusters; **catalase+**, **coagulase+**, β-hemolytic; has Protein A, ferments mannitol | Transmission: Hands of hospital clinical staff; catheters, bedsheets, clothes, med. eq.<br>Pathogenesis:<br>- Folliculitis, furuncles, carbuncles: enters through hair follicles and causes local pustule (folliculitis); may spread into subcutaneous tissue (furuncle); can then spread under the skin (carbuncle)<br>- Impetigo: Runny noses and nose picking spreads bacteria<br>- Pneumonia: Esp. after secondary influenza infection, or in the immuno-compromised or impaired pulmonary fxn.<br>- Osteomyelitis, endocarditis: Result from bacteremia of some origin (osteomyelitis from orthopaedic surgery, endocarditis from burns or acute staph. Enteritis)<br>- Scalded skin syndrome: Colonization by S. aureus on skin<br>- Toxic shock syndrome: From boil or superabsorbent tampon; TSST-1 toxin causes shock.<br>- Food poisoning: from individuals with S. aureus on hands or nose; improper storage of food results in multiplication of bacteria and enterotoxin production; **note** that this toxin is **HEAT-STABLE** and cannot be destroyed by warming | Lipases: digest fats, possibly assist in boil formation<br>α-hemolysin (α-toxin): lyses cells by forming transmembrane channels (dermal necrotic factor)<br>β-hemolysin: sphingomyelinase that lyses RBCs.<br>γ-hemolysin: function unknown<br>δ-hemolysin: damages cells, elicits platelet-activating factor<br>Staphylococcal enterotoxin (SEs): phage-carried gene; stimulate local neural centers in the gut that travel to the vomiting center during food poisoning; seven types (SE-A,B, C1,C2,D,E,F); SE-B plays a role in toxic shock syndrome (TSS)<br>Exfoliative toxin: destroys intracellular connection between skin layers (i.e. destroys desmosomes). Seen in scalded skin syndrome.<br>Toxic Shock Syndrome Toxin-1 (TSST-1): Superantigen which ↑ IL-2 and sensitivity to LPS, leading to shock and multiple organ failure | **Dx**:<br>- Folliculitis, furuncles, carbuncles: culture of material from staph. pyoderma on blood agar<br>- Impetigo: Culture of organisms from lesions<br>- Pneumonia: Culture from sputum, blood, transtracheal aspirates<br>- Osteomyelitis, endocarditis: Culture from lesion exudate and blood<br>- Scalded skin synd: clinical Dx b/c skin is scalded but NOT infected<br>- TSS: Culture from boil or tampon<br>**Rx**:<br>-Folliculitis, impetigo, furuncles, carbuncles: muciprocin, ERY.<br>- Pneumonia:systemic antibiot. (methicillin, vancomycin)<br>- Scalded skin synd.: penicillinase-resistant penicillin is used; vancomycin used with methicillin-resistant S. aureus (MRSA)<br>- TSS: vancomycin, supportive measures for shock<br>- Food poisoning: monitor hydration and electrolytes |
| Staphylococcus epidermidis | Endocarditis, UTIs | Gm+ coccus, grows in grape-like clusters; variable hemolysis; **requires biotin for growth**; coagulase **–**; catalase + | Infection in implanted device; endocarditis seen in IV drug users; UTIs in elderly men | Multi-drug resistance, growth in implanted device makes bacteria hard to reach | Rx: vancomycin due to multi-drug resist. |
| Staph. saprophyticus | UTIs | Gm+ coccus, grows in grape-like clusters; variable hemolysis; coagulase **-;** catalase+ | UTIs common in sexually active young women because it normally (but transiently) inhabits in skin near/around urethra | | Rx: Trimethoprim/ sulfamethoxazole; can use ampicillin, amoxicillin, or fluoroquinolone as an alternative |

## Bacteria Chart: Enterococcus species

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Enterococcus faecalis | Nosocomial inf., endocarditis, bacteremia, UTIs, abdominal infections | Gm+ coccus; var. hemolysis; grow in wide temp. range (10-45°C); **grows in 6.5% NaCl medium** | Transmission: by hands, sheets, clothes, catheters<br>Pathogenesis: resistance to antibiotics allows spread | ** Some strains are known to be resistant to every known antibiotic, including vancomycin | Dx: blood culture<br>Rx: Combination therapy with penicillin, a glycoside (vancomycin, teicoplanin) plus an aminoglycoside |
| Enterococcus faecium | Neonatal meningitis | Gm+ coccus; var. hemolysis; grow in wide temp. range (10-45°C); **grows in 6.5% NaCl medium** | Transmission: hands of clinical workers to medical equipment | ** High antibiotic resistance | Dx: blood culture<br>Rx: Combination therapy with penicillin, a glycoside (vancomycin, teicoplanin) plus an aminoglycoside |

## Bacteria Chart: Streptococcus species

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Streptococcus pyogenes (Group A Streptococcus) | Streptococcal pharyngitis ("strep throat"); impetigo, scarlet fever, toxic streptococcal synd., acute rheumatic fever, acute glomerulo-nephritis, necrotizing fascitiis, erysipelas, puerperal sepsis, otitis media | Gm+ cocci, facultative anaerobes, catalase -, grow in chains; **bacitracin-sensitive**; **M-proteins separate different strains** | **Transmission**:<br>Pharyngitis: Infected mucus on hands, clothes, sheets<br>Impetigo: Contact with infected material<br>Scarlet fever: Same as pharyngitis, but infection is by strain producing an SPE<br>Toxic Streptococcal Syndrome**:** Caused by S. pyogenes in wounds, which produce superantigen (and toxin) SPE, which ↑ IL-2 and sensitivity to LPS.<br>Erysipelas: by puncture, abrasion, laceration; hyaluronidase produced by S. pyogenes allows spread.<br>Acute rheumatic fever: sequela of pharyngitis; rheumatic heart disease can follow<br>Acute glomerulonephritis: caused by nephritogenic strains after pyoderma<br>Necrotizing fascitiis: Minor trauma area Becomes infected; progressively destroys fascia and fat, sometimes sparing the overlying skin.<br>**Pathogenesis**:<br>- Adhesion; spread via hyaluronidase; SPEs; virulence factors involved in bacterial survival | Streptolysin O: hemolyzes blood oxygen-labile, antigenic<br>Streptolysin S: hemolyzes blood oxygen-stable, nonantigenic<br>Exotoxins: erythrogenic toxins, scarlet fever toxins, SPEs (SPEs responsible for scarlet fever and toxic streptococcal syndrome)<br>Streptodornase: DNAse<br>Streptokinase: hydrolyzes fibrin in blood clots (used clinically)<br>C5a peptidase: degrades C5a<br>IgAase: cleaves secretory IgA<br>Hyaluronidase: "spreading factor" responsible for spread in tissue in pyoderma<br>M-protein: blocks opsonization and phagocytosis | Pharyngitis, Scarlet fever: throat culture; Rx with penicillin V<br>Impetigo: Clinical appearance of lesions; culture of lesions; Rx is cephalosporin or erythromycin<br>Toxic Strep. Synd.: Blood and tissue cultures of GABHS, Rx by penicillin and debridement<br>Necrotizing fascitiis: clinical presentation, blood & tissue cult., Rx by debridement and combination of ampicillin, gentamicin and clindamycin<br>Erysipelas: clinical Immobilization and moist heat applied to affected area; dicloxacillin or erythromycin is given<br>Acute rheum. fever: ↑ titers of SLO antibody; Rx by penicillin V or G<br>Glomerulonephritis: Edema of face and legs, proteinuria, hematuria, azotemia, hypertension; Rx for uremia & hypertens. |
| Streptococcus agalactiae (Non-Grp. A Streptococcus) (Group B Streptococcus) | Meningitis, arthritis, bacteremia, pneumonia, osteomyelitis; **neonatal and peripartum infections** | Gm+ cocci, grow in chains; facultative anaerobe; catalase -; **bacitracin-resistant**; hydrolyzes hippurate; CAMP +; | Neonatal fluids; immunocomprised patients are susceptible; endometritis. | | Dx by lab tests and also by anti-grp. B antibody tests<br>Rx by penicillin G & ampicllin<br>Prevention: screening pregnant women for rectovaginal infection and treating with ampicillin, penicil. G, cephalothin, or ERY. |
| Streptococcus viridans **group** (S. mutans, S. sanguis) | Dental caries, endocarditis | Gm+ cocci, grow in chains, facultative anaerobe; catalase -; **α-hemolytic**; | Transmission: is normal flora, causes disease by opportunistic infection;<br>Dental caries: S. mutans attaches to dextran, erodes teeth by converting sucrose to lactic acid and lactate<br>Subacute endocarditis: dental procedures, vigorous toothbrushing allows bacteria to invade | | Endocarditis: Rx w/ penicillin,gentamycin |

## Bacteria Chart: Streptococcus species, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Streptococcus pneumoniae (Pneumococcus) | Pneumonia; otitis media, conjunctivitis; sinusitis, meningitis | Gm+ cocci, grow in chains, facultative anaerobe; **optochin-**sensitive, bile-soluble; α-hemolytic BUT β-hemolytic when grown anaerobically; type-specific **capsule** is key antigen (ID'd by Neufeld reaction) | Pneumonia: Aspiration into lung; infection in lower/lower middle lobes in patients who are immunocompromised; infection results in PMN attraction, resulting in congestion<br>Meningitis: Caused by bacteremia, which in turn is secondary to other infections (i.e. pulmonary pneumonia)<br>Otitis media: From upper respiratory tract infections | Neuraminidase: Promotes pneumococcal access to the lings by thinning mucous secretions<br>IGA protease: Cleaves sIgA, IgA, IgG, IgM.<br>Capsule: Protects from phagocytosis and also assist in inhibiting deposition of opsonic C3b on the bacterial surface. | Pneumonia: Dx by X-ray, blood studies and blood culture; Rx by Penicillin G, possibly vancomycn if strain is resistant.<br>Meningiitis: Dx by neurologic tests and laboratory testing; Rx depends on age infants (1-3 months) get ampicillin plus cefotaxime; children (3 months to 7 years) get ceftriaxne; adults are given ampicillin or penicillin G, plus ceftriazone or cefotaxime<br>Otitis media: Dx by culture from needle biopsy; Rx: 4 yrs or younger is treated w/ ERY and sulfa; older than 4 yrs w/ tri-sulfa, cefuroxime<br>Conjunctivitis ("pink eye"): Dx by clinical grounds; Rx with drops containing sulfa or neomycin and polymyxin<br>Sinusitis: Dx on clinical grounds; Rx by decongestants and with antibiotics (ampicillin, 2nd gen. cephalosporins)<br>Prevention: Pneumovax, to elderly/immunocomp. |

## Bacteria Chart: Respiratory infection-causing

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Mycoplasma pneumoniae | **Primary atypical pneumonia** ("walking pneumonia") | **NO CELL WALL** Sterols in membrane stabilize it; slow-growing; acidifies glucose-containing agar; alkalinizes arginine or urea-containing medium | **NOT** secondary to other condition; M. pneumoniae attaches to host cells via specialized tip with P1, an adhesin to neuraminic acid residues; afterwards, the bacteria damages host cells by producing LOTS of $H_2O_2$. | Adhesin: Promotes attachment to epithelium | Dx: History and physical examination; mucous sputum that is mucoid or mucupurulent; most Dx on clinical grounds.<br>Rx: Macrolides, doxycycline; BUT note that walking pneumonias are **self-limiting** and that antibiotics only reduce the duration of the disease. |
| Chlamydia trachomatis | Trachoma; Lymphogranuloma venereum; Non-gonococcal urethritis (NGU) (**50% of NGU cases**); Pelvic inflammatory disease (PID) | Gm- bacteria; **obligate intracellular parasite**; two forms are inert extracellular elementary bodies; and metabolically active intracellular reticulate bodies, which take ATP from the host and multiply within host vacuoles. | Transmission by elementary bodies released into environment, causing infection<br>Pathogenesis by reticulate bodies | **Ability to live intracellularly** | Trachoma: Clinical dx; Rx: doxycycline or erythromycin<br>Urethritis: Clinical dx, but no bacteria are seen in the exudate. Rx by doxycycline<br>Lymph. venereum: Dx is clinical; Rx by doxycyclin or ERY. |

## Bacteria Chart: Respiratory infection-causing, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Chlamydia pneumoniae | Acute respiratory pneumonia | Same as Chlamydia trachomatis, but elementary bodies are oblong and pear-shaped; also contain miniature bodies; reservoirs are **only human!** | Not usually transmitted person-to-person; however, pathogenesis still by elementary bodies | Able to live intracellularly | Dx: clinical sympt., lab tests<br>Rx: prolonged, high-dose tetracycline or doxycycline treatment |
| Chlamydia psittaci | Psittacosis | Same as Chlamydia trachomatis | Spread by breathing air contaminated with feces of diseased birds; spread person-to-person by respiratory droplets.<br>Pathogenesis: Makes its way into lung, causing symptoms such as pneumonitis, CNS symptoms. | Able to live intracellularly | Dx: Lab tests of macrophages will reveal inclusion bodies; sputum will ID antibody to Chlamydia<br>Rx: tetracycline and isolation to prevent disease spread |
| **Bordatella pertussis: See toxicogenic bacteria section** | | | | | |
| Coxiella burnettii | Q fever | Gm- rods; obligate intracellular parasite; complex life cycle; multiples within host vacuoles rather than cytoplasm; do **NOT** require arthropod vector (as rickettsiae do); have endospores but aren't dormant or resistant to envir. extremes | Spread by aerosols containing coxiellae; contact with infected placentae; ingestion of infected meat or milk<br>Pathogenesis by LPS | LPS of coxiallae undergoes phase variation; Phase I are more virulent and more resistant to phagocytosis than Phase II | Dx: Positive Hx of working in slaughter-houses or with meat; chest films<br>Rx: respiratory isolation; doxycycline for acute cases; fluoroquinolone or rifampin for chronic cases |
| Legionella pneumopnila | Legionnaire's disease | Gm- rods; free-living; grow on **charcoal-yeast extract** (CYE) agar; require L-cysteine to grow; reside in **fresh-water potable systems** and **cooling systems** | Transmission by water droplets; enters lung via respiratory tract (**note**: it does **NOT** infect the pharyngeal mucosa).<br>Pathogenesis: Can live inside PMNs, monocytes, macrophages; **coiling phagocytosis** results when bacteria is left in a whirling vacuole lined with ribosomes, making it easier for the bacteria to multiply.<br>**NOTE**: Only enters cells after C3 is activated by Legionella antibody and binds to the MOMP and cellular C3 receptor. | Major outer membrane protein (MOMP):: anchors LPS to Outer membrane of host cells<br>Macrophage infectivity potentiator (Mip): necessary for survival within macrophages; mechanism unknown<br>Phosphatase: Inhibits prod. of superoxide by ↓ DAG and $IP_3$<br>Peptide toxin: Cytotoxic, blocks action of phospholipase C<br>**Zinc metalloproteinase**: Major virulence factor; cytotoxic, inhibits superoxide formation, inhibits NK cell activity; degrades IL-2, TNF-$\alpha$ and CD4 antigen, $\alpha$1-antitrypsin | Dx: Clinical sympt. of pneumonia; PMNs are present, but there are few/no organisms showing up on Gm stain. Definitively diagnosed by culture on CYE agar. Also, serologic tests for antibody titer (a four-fold or greater rise is needed to confirm)<br>Rx: ERY in combo. with rifampin |

## Bacteria Chart: Respiratory infection-causing, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Mycobacterium tuberculosis | Tuberculosis | Gm+ **acid-fast** rods; facultative aerobe; hydrophobic, complex cell wall which contains waxes | Transmission: Droplet nuclei from coughing; droplets can remain airborne and infectious for 30 minutes after release; droplets make their way down the bronchi to the alveolus<br>Pathogenesis: grows and survives in alveolar macrophages; if M. tb enters the bloodstream, systemic TB results | Adenylate cyclase: Inhbits macrophage degranulation<br>Arabinogalactan: Elicits futile antibody response<br>Lipoarabinomannan: Elicits futile antibody activity; suppresses T-cell activity; inhibits antigen presentation; induces TNF-α production; inhibits IFN-γ-mediated macrophage activation<br>Mycolic acids: Confers acid-fast property; protects against acids and alkalines<br>Mycosides (ex. cord factor): Inhibits leukocyte migration, stimulates granuloma formation, destroys mitochondrial membranes; inhibits cellular response, inhibits IL-6 release, inhbits fusion of macrophage lysosomes with phagosomes<br>Sulfatides: Potentiates cord factor effects, immobilization of macrophagic hydrolytic enzymes, blocks macrophage degranulation<br>Tuberculoproteins: interfere w/ immune response, promote cellular invasion | Dx: Acid-fast staining of sputum, Chest X-ray, + PPD; Bactec lab culture, PCR; however, **Dx is often CLINICAL** with symptoms of active TB (fever, coughing with bloody sputum, weight loss, fatigue & night sweats)<br>Rx: Rifampin, streptomycin, new fluoroquinolones; **isonizaid (INH), ethionamide**, **ethambutanol, pyrizinamide**<br>** **Resistance due largely to non-compliance** (b/c of asymptomatic infection); also, Rx is usually a combination of antibiotics<br>Prevention: Vaccine (controversial), or natural protection by exposire to mycobacteria other than M. tb |
| Mycobacterium bovis | Cow TB | Similar to M. tb | | | |
| Mycobacterium africanum | TB | Similar to M. tb | | | |
| Mycobacterium microti | TB | Similar to M. tb | | | |
| Mycobacterium avium-Intracellulare complex (MAC) | Pulmonary inf., infections of lymph nodes, disseminated disease | Similar to M. tb | Contamination of tap water; reservoir of bacteria in birds. Enters immuno-compromised patients through GI tract, by adhering to and invading intestinal epithelium and fibroblasts. | Inhibition of lysosomal fusion with phagosome; heat shock proteins allow the phagocytosed MAC to avoid being killed | Dx: Culture from sputum, blood, stool, biopsy<br>Rx: Clarithromycin + one of (ethambutol, clofazimine, ciprofloxacin); removal of infected lymph nodes |
| Mycobacterium kansasii | Similar to MAC | | | | |
| Mycobacterium chelonae | Fast-growing; otherwise similar to MAC | | | | |
| Mycobacterium fortuitum | Fast-growing; otherwise similar to MAC | | | | |
| Mycobacterium marinum | Slow-growing; Variable and non-specific infection seen in fishermen/fish-handlers and swimmers | | | | |
| Mycobacterium leprae | Hansen's disease (Leprosy) | Gm+ **acid-fast** rod; Extracted w/pyridine; **intracellular**, grows within macrophages, Schwann cells, endothelial cells, epithelial cells; **longest doubling time of ALL pathogens (11-13 days)**; only cultured in mice and nine-banded armadillos | Transmission by respiratory secretions of patients w/lepromatous leprosy; or by zoonotic transmission<br>Pathogenesis by vigorous antibody response elicited when macrophage degranulation is blocked, and its activation by IFN-γ is also blocked; results in an Arthus-type reaction<br>**Lepromatous**: large lesions, many bacteria, weak CMI, large amount of neural involvement, i.e. no feeling in extremities<br>**Tuberculous**: Few lesions, few/no bacteria, very strong CMI, rare neural involvement | Arabinogalactan, mycolic acids, adenylate cyclase, sulfatides (see M. tb) | Dx: of Tuberculous, borderline, and lepromatous leprosy, by history, physical exam, & microscopic examination of samples taken from lesions<br>Rx: Combination of dapsone and rifampin; physical and psycho-social therapy b/c of social unacceptability |

## Bacteria Chart: Actinomyces

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Actinomyces israelii<br>Actinomyces naeslundii<br>(A. israelii most common) | Actinomycosis (chronic suppurative and granulomatous infection) | Gm+ club-shaped, branched rods; rough growth on agar, slow-growing, hydrolyzes esculin, inhbited by bile, ferments glucose, mannitol, rhamnose; facultative anaerobes | Transmission from trauma, surgery, or dental work, b/c A. israelii resides in oropharynx; also from soil entering a deep wound<br>Pathogenesis: Abscesses of connective tissue are destructive; lesions are purulent and often walled-off | | Dx:**sulphur granules** (yellowish, 1 mm granules composed of macrophages, fibrin, and bacteria); specific species is then ID'd.<br>Rx: Clindamycin, ERY, penicillin G; surgical excision of |

## Bacteria Chart: Nocardia

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence Factors | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Nocardia asteroides<br>Nocardia brasiliensis<br>Nocardia otitidiscaviarum | Nocardiosis | Gm+ **weakly acid-fast**, branching filamentous; strict aerobes; contain mycolic acids | Transmission by opportunistic infection in immunocompromised patients; airborne spores are inhaled, make their way to the lung, where bacteria multiply and survive within macrophages.<br>(Immunocompromised patients: those w./ corticosteroid Rx, immunosuppression, organ transplants, HIV+, TB, alcoholics)<br>**Note**: **NOT** transmissable between people<br>Pathogenicity by spread to blood to organs and brain | Inhibition of lysosome fusion; Use of external catalase and superoxide dismutase to detoxify oxygen-derived products | Dx: Visualizing organism in exudate and culture<br>Rx: Trimethoprim/ Sulfamethoxazole; surgical drainage or resection may be required |

## Bacteria Chart: Zoonotic Bacteria

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Yersinia pestis<br>(Pasteurella pestis) | Plague<br>sylvatic: flea bite<br>urban: rats ➔ fleas ➔ humans | Gm- coccobacillus; **bipolar-staining**; encapsulated, can live both intra- and extra-cellularly | Flea bites, then enters lymph nodes; creates necrotizing toxin in nodes; results in suppurative lymph nodes, fever, malaise; Plasminogen activator portentiates the dissemination of Y. pestis in the body; this results in necrosis of fingers, toes, nose, ears via disseminated intravascular coagulation (DIC) — the "black death," quite literally. | Coagulase – causes fleas to vomit up blood from their stomachs, usually directly into the bite in its victim; Y. pestis enters the body in this manner<br>Murine exotoxin: depletes NAD by splitting it into ADP-ribose and nicotinamide; interferes with sympathetic NS's ability to regulate body temp.<br>V and W antigens: immuno-suppressive, antiphagocytic, protect bacteria, promotes $Ca^{2+}$ dependence<br>Yersinia outer membrane proteins (Yops): involved in rapid growth of Yersiniae in tissues<br>LPS: Endotoxin<br>Pigmentation Peptide F: allows Yersinia to obtain iron<br>Capsule: is antiphagocytic | **Dx**:<br>- Bubonic plague: classic triad of high fever, buboes, and conjunctivitis (buboes usually in inguinal area)<br>- Septicemic plague: no buboes, but early DIC and vascular collapse<br>- Pneumonic plague: respiratory distress probably caused by inhaling droplets from an infected patient<br>**Rx**: tetracyclines, sterptomycin<br>**Prevention**: Vaccine, improved sanitation (i.e. get rid of the rats!) |
| Francisella tularensis | Tularemia | Gm- pleomorph. rod; Facultatively intra-cellular; nonmotile, ferment glucose, oxidase -, urease -, produce no $H_2S$ on triple sugar iron (TSI) agar; don't reduce nitrate; grows in charcoal agar | Large reservoir in mammals, birds and insects; **transmission usually by insect bite**. Pathogenesis by ulcerative lesion; if subsequent spread of a septicemic ulcer to lymph nodes, endotoxic shock may follow. Can also cause bronchitis and pneumonia. In terms of exam questions, look for a Hx of **hunting** or **pelt curing** | Ability to survive in unstimulated macrophages | Dx: ulcerative lesion, fever, malaise, lab test of antibody titer<br>Rx: Streptomycin (penicillin and sulfa-resistant)<br>Prevention: avoid sick/dead animals |
| Brucella melitensis (goats)<br>Brucella abortus (cattle)<br>Brucella suis (swine)<br>Brucella ovis (sheep)<br>Brucella canis (dogs)<br>Brucella neotomae (wood rats) | Brucellosis (malta fever, undulent fever) | Gm- coccobacillus; strict aerobes, lack spores and capsules; fermentation of sugars, urease & $H_2S$ production, and $CO_2$ requirements for growth differentiate between species | Carried by diseased animals; transmitted through ingestion of **contaminated** food & **milk**<br>Brucellosis has 1-5 week incubation, 3 month course of fever, headache, myalgia, and malaise; relapses can occur over 25 years!! | Ability to survive in unstimulated macrophages; S form is better suited for living in macrophages; transition from S (smooth) to R (rough) forms in the presence of D-alanine | Dx: Clinical sympt.; Agglutination test for Brucella antibody; also, cultures<br>Rx: Controversial b/c treatment is long-term and patients often suffer from relapses; also, antibiotics work at therapeutically effective conc.'s, so Rx consists of combo of tetracycline for 3 wks. & streptomycin for 2 wks.<br>Prevention: No human vaccine, but cows are vaccinated against brucellosis |
| Pasteurella multocida | Bite infection from dogs and other domesticated animals | Gm- rod; bipolar-staining; oxidase+, catalase+, ornithine decarboxylase+, indole+, urease -; nonhemolytic; encapsulated | Scratch of bite of dog or cat; lymphatic spread to lymph nodes and adjacent tissues; possible suppurative resp. tract infection in those with chronic lung disease; possible sepsis in those with liver disease or cancer | Toxin: growth factor which makes wound worse | Dx: Culture of infection (probably)<br>Rx: Cleansing wound, penicillin G, doxycycline, amoxicillin plus clavulanate |

## Bacteria Chart: Zoonotic Bacteria, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Leptospira interrogans<br>Leptospira canicola (swine)<br>Leptospira pomona (dogs) | Leptospirosis | Gm- helical spirochete; aerobic; 1 axial filament; **most tightly coiled spirochete**. | Spread by contact with urine of infected animals, or indirect contact w/contaminated water or mud in flood streets, rice paddies, jungle swamps<br>** Rats are key reservoirs **<br>Pathogenesis: Entrance through skin or mucous membrane, then entrance to bloodstream and organs; after a period of latency, immune-mediated damage results in symptoms such as hemorrhage, diarrhea, jaundice, severe renal impairment, hypovolemia, aseptic meningitis | Endotoxin-like activity may play a role in effects on vascular function | Dx: Clinical history, slide agglutination test or immunosorbent assay for antibodies<br>Rx: doxycycline or penicillin G, although Rx is controversial |
| Borrelia burgdorferi | Lyme disease<br>- 3 areas:<br>- New England, NY, PA, Atlantic Seaboard<br>- WI, MI, MN<br>- Western NV, CA, OR | Gm- helical spirochete; aerobic; 7-11 axial filaments; **least tightly coiled spirochete class; longest & narrowest of the Borrelia**; cultured in modified Kelly's medium; **linear plasmids**! | Vector spread – I. ricinus complex of ticks<br>Pathogenesis: 3 stages<br>- Stage 1: Results in skin lesions, flu-like illness<br>- Stage 2: Neurologic and cardiac abnormalities<br>- Stage 3: Persistent arthritis<br>Methods of pathogenesis:<br>1. LPS-elicited production of IL-1<br>2. Macrophage/TH-1 responses via IL-12 | Tropism of B. burgdoferi for skin, joints, and CNS<br>Ability to persist for years in skin, joints, and CNS for years!<br>Immune response to persistent organisms are associated with symptoms and damage to host<br>Antigenic variation!! | Dx: Symptoms of erythema chronicum migrans (ECM) -- papules with rings around them; chronic fatigue-like synd., arthritis of large joints **plus** patient's being in an endemic area, **plus** detection of antibodies to B. burgdoferi in serum or CSF<br>Rx:<br>- ECM: oral doxycycline or amoxicillin<br>- Neuro, cardiac, arthritic symptoms: ceftriaxone<br>Prevention:<br>- **No (real) vaccine**<br>- Avoid tick bites by wearing long-sleeved shirts, using repellants with permethrin, etc. |
| Borrelia recurrentis | Relapsing fever (epidemic) | Gm- helical spirochete; aerobic; least tightly coiled spirochete class | Spread by louse bite (man-louse-man)<br>Variable major proteins, immunodominant proteins on their outer membranes, cause the disease to wax and wane.<br>- VMPs carried on linear plasmids<br>- 1st VMP recognized by host, most bacteria die; howver, some bacteria expressing a different VMP survive, multiply, and cause relapse<br>**Note**: Epidemic relapsing fever is NOT Considered zoonotic b/c there is no animal reservoir. Also, there is no transovarian transmission in the louse. | VMPs (see transmission & pathogenesis column) | Dx: Wright stain of blood smear, animal inoculation, serology tests (VDRL +, OXK agglutinins +)<br>Rx: Penicillin and Tetracyclines |
| Borrelia hermsii<br>Borrelia duttonii | Relapsing fever (endemic) | Same as above | **Note**: Endemic relapsing fever IS considered zoonotic, because it is spread by ticks from animal to man. Also, there is a large rodent reservoir. | Same as above | |
| Bacillus anthracis | Anthrax | Gm+ large rod; capsule only seen in high $CO_2$ levels; **non-motile**; Medusa's head colonies seen when grown on blood agar; **spore-former** | Transmission by individuals **handling** animal **wool**, fur, hides or feces containing anthrax spores; or by ingesting meat containing vegetative B. anthracis<br>Pathogenesis by capsule and toxin; direct contact, then spore enters skin; spores germinate, producing a malingnant pustule; dissemination results in fatal septicemia. Pulmonary anthrax is invariably fatal; this is a possible biological weapon against armies, so some military personnel are being vaccinated against anthrax. | Requires **BOTH**:<br>- Capsule: antiphagocytic b/c it protects bacteria from antibody and complement.<br>- Toxin: Three proteins: edema factor (EF), an adenylate cyclase activated by calmodulin; EF ↑ cAMP, resulting in hyper-secretion and edema; lethal factor (LF)'s function is not well defined; and protective antigen (PA) is a carrier protein that binds to cell surface receptors. PA is cleaved by cell proteases, binds to EF or LF, and enters the cell; EF and LF then exert their effects. | Dx: cultivation of pustule, blood, sputum, gastric washing, depending on type of infection cutaneous, pulmonary, GI, or meningeal)<br>Rx: **Rapid** administration of penicillin<br>Prevention: Kill infected cattle, incinerate carcasses, prevent contaminated hides from entering the US. |

## Bacteria Chart: Zoonotic Bacteria, continued (the remainder of these are minor but are still MB exam-worthy)

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Spirillum minus | Rat bite fever (sodoku) | Helical Gm- rod; cannot be cultured on artificial media | Spread by bites of cats, ferrets, rats, weasels<br>Pathogenesis: fever that appears for 1-2 days, remits for 3-9 days, then **recurs** with **regional lymphadenopathy**. | (Not described in text/syllabus) | Dx by lab culture;<br>Rx by ampicillin, penicillin G or streptomycin |
| Streptobacillus moniliformis | Rat bite fever, Haverhill fever | Gm- rod; pleomorphic, cannot be cultured on standard media; requires special biphasic serum agar | Spread: Rodent bites for rat bite fever; contaminated milk for Haverhill fever<br>Pathogenesis: systemic disease with fever, rash, arthiritis. **Chancre sore** can form and result in an abscess, bacteremia, causing possible penumonia and other infections. | (Not described in text/syllabus) | Dx by lab culture;<br>Rx by ampicillin, penicillin G, streptomycin or tetracycline |
| Erysipelothrix insidiosa (Erysipelothrix rhusiopathiae) | Erysipeloid | Gm+ rod; α- or non-hemolytic; ferments glucose; catalase -, $H_2S$ +, esculin -; **only species in its genus** | Spread: Contact of skin abrasion with pig feces; affects fishermen, meat handlers, and veterinarians/animal handlers b/c sea animals, horses and turkeys carry it too.<br>Pathogenesis: Hard, swollen, burning, itching, purplish swelling on hand **w/out** pus in the lesion. Lesion is indolent – it simply exists, and sits there. | (Not described in text/syllabus) | Dx: occupational history, lab culture from hand specimen<br>Rx: penicillin G, ampicillin, or cephalothin<br>**Note**: most strains are **resistant to vancomycin**!!<br>Prevention: wearing gloves |
| Pseudomonas mallei | Glanders | (Not described in text/syllabus) | Disease of horses than can wipe out an entire herd; leads to acute pulmonary infections with local production of pus | (Not described in text/syllabus) | Dx: not described<br>Rx: CaRTS<br>Chloramphenicol<br>Rifampin<br>Tetracycline<br>Sulfa drugs |
| Pseudomonas pseudomallei | Melioidosis (acute Pneumonia) | Gm- rod; inhabits soil, ponds, rice Paddies, primarily in SE Asia, Philippines, Indonesia; grows well on many standard media, with wrinkled colonies; bipolar-staining; oxidase +, reduces nitrate to nitrite, neutral TSI butt at 24h, acid at 72h | Spread by contact of abrasions, cuts, or ulcers with bacteria in warm water or wet soil.<br>Pathogenesis: Enter through abrasions, cuts, ulcers; possible inhalation<br>Other pathogenesis is not well-known. | Endotoxic LPS<br>Extraceullular protease<br>** Can survive quietly in liver and spleen for yeears, then suddenly activate, causing a rapid, life-threatening infection. | Dx: sputum or blood culture<br>Rx: CaRTS (above)<br>Trimethoprim/ sulfamethoxazole |

## Bacteria Chart: Rickettsiae, Ehrlichiae, Bartonella

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Rickettsia prowazekii<br>Rickettsia typhi | Typhus<br>R. prow: epidemic<br>R. typhi: endemic<br><br>R. prow: worldwide<br>R. typhi: worldwide | Gm- rods; obligate intracellular parasites; polymerize actin; require NAD & CoA; acquire cell wall precursors from host; needs host to replicate; killed by intracellular NO, induced by IFN-γ and TNF | Transmission by **human louse**: louse feces infect bite, louse dies from typhus<br>• **NO** transovarial transmission!<br>Pathogenesis: Infection and reproduction in endothelium, causing thrombosis, hemorrhage, plasma leakage, hemoconc. and shock. Headache, malaise, fever in 2-3 days, **chest rash** in 4-7 days, spreading to extremities, persistence for 2 weeks, even w/treatment; 10-70% mortality; increasing mortality with increased age.<br>• Mild version (**Brill-Zinsser disease**, recrudescent typhus) may occur years after initial infection | Intracellular; lives in & lyses endothelial cells & unstimulated macrophages, leading to ↑ permeability of the circulation, a ↓ in blood flow, leading to shock | Dx: Antibodies to typhus rickettsiae (Weil-Felix test, or indirect fluorescent antibody test)<br>Rx: Doxycycline and chloramphenicol; supportive measures for those in shock<br>Prevention: Vaccine exists, but does **NOT** prevent the disease; it only lessens the disease's effects. |
| Rickettsia tsutsugamushi | Scrub typhus, seen in Asia, Japan, and South Pacific | Gm- rods; obligate intracellular parasites; polymerize actin; require NAD & CoA; acquire cell wall precursors from host; needs host to replicate; killed by intracellular NO, induced by IFN-γ | Tranmission by **trombiculid mite vector**, found in scrub vegetation<br>Pathogenesis: Organism enters via bite and replicates intracellularly; **exits host cell individually**. Results in a **black eschar**; also results in headache, trunk rash spreading to extremities, CNS involvement. **10-60% mortality**. | **Transovarial transmission** from mite to its offspring | Dx: Mainly clinical (if suspected, Rx is started). ELISA has has been used to Dx the organism.<br>Rx: Chloramphenicol and vector control |

## Bacteria Chart: Rickettsiae, Ehrlichiae, Bartonella, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Rickettsia rickettsii | Rocky Mountain Spotted Fever, seen primarily in New England and Atlantic seaboard; & Western US (Montana esp.) | Gm- rods; obligate intracellular parasites; polymerize actin; require NAD & CoA; acquire cell wall precursors from host; needs host to replicate; killed by intracellular NO, induced by IFN-γ and TNF | Tranmission by **dog and wood ticks**; rabbit tick maintains reservoir in rodents, rabbits, hares. Other reservoirs are dogs, deer and other animals.<br>Pathogenesis: Headache, fever, chills, malaise, myalgia 2-12 days after bite; **rash on wrists, ankles, proceeding to trunk, palms, soles**. Pathogenesis by severe endothelial damage, resulting in thrombosis, DIC. Mortality rates of 5-90% depending on infecting strain. | **Transovarial transmission** from tick to its offspring | Dx: Mainly clinical (Rx started if disease suspected); confirmed by lab tests<br>Rx: Tetracyclines or chloramphenicol<br>Prevention: Insect repellants, frequent tick removal |
| Rickettsia akari | Rickettsial pox (mild illness) | Gm- rods; obligate intracellular parasites; polymerize actin; require NAD & CoA; acquire cell wall precursors from host; needs host to replicate; killed by intracellular NO, induced by IFN-γ and TNF | Tranmission by **house mouse mite**<br>Pathogenesis: Mild disease; eschar at bite site with trunk, palm, sole, oropharyngeal mucosal rash; fever precedes rash.<br>(cp. chicken pox, where fever follows rash, and also, no eschar). | | Dx: Clinical<br>Rx: None; recovery in 10 days<br>Prevention: Control of house mice and mites |
| Ehrlichia chaffeenis | Ehrlichiosis (human monocytic) | Gm- coccus, obligate intracellular bacteria; infect macrophages, monocytes, platelets &endothelial cells | Transmission via **arthropod vector** (Lone Star tick, American dog tick)<br>Pathogenesis: leukopenia, thrombocyto-penia develop 3-4 weeks after bite; fever may also be seen. Symptoms are thus to RMSF, but without the rash.<br>Death due to opportunisitc infections (i.e. pneumonia) | Not thoroughly understood | Dx: Clinical Dx, confirmed by **exclusion of RMSF** and demonstrating inclusion bodies in macrophages of the liver, spleen, and bone marrow<br>Rx: Doxycycline |
| Ehrlichia equi | Ehrlichiosis (human granulocytic) | Gm- coccus, obligate intracellular bacteria; infects neutrophils | Transmission via **arthropod vector** (blacklegged or deer tick – these also transmit Lyme disease)<br>Pathogenesis: fever, chills, headache, malaise, myalgia 8 days after exposure<br>Death due to opportunistic infections (i.e. pneumonia) | Not thoroughly understood | Dx: Clinical Dx, confirmed by showing PMNs w/vacuoles containing Ehrlichia; also indirect fluorescent antibody test fro E. equi<br>Rx: Doxycycline |
| Bartonella quintana | Trench fever | Gm- coccobacillus; extracellular, grows on enriched media w/5% sheep blood | Transmission via **human lice**; bacteria grows in louse gut; **NO** transovarial transmission in louse. Organism is passed from louse feces into bite, organism enters skin when patient scratches the area.<br>Pathogenesis: fever, anemia, and malaise 4-35 days after infection; shins are painful; bacteremia & endocarditis in immunocompromised hosts | Grows inside lymphocytes | Dx: Clinical Dx.<br>Rx: Tetracyclines |
| Bartonella hanselae | Cat-scratch disease | Gm- coccobacillus; extracellular, grows on enriched blood agar; slow-growing. | Transmission by cat srcatch, licks, or bites<br>**Note**: Actual transmission might be from cat fleas!<br>Pathogenesis: Mechanisms unknown; macule develops at site, resulting in swollen lymph nodes in the head, neck and upper extremities. Granulomas cause lymphadenopathy.<br>**Note**: In immunocompromised patients, bacillary angiomatosis and bacillary peliosis hepatitis may result. | | Dx: Clinical Dx, also skin test using extract from lymph nodes of patients<br>Rx: Rifampin, ciprofloxacin, gentamicin, tri/sulfa; aspiration of swollen lymph nodes |

# Bacteria Chart: Enteric Bacteria

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Salmonella typhi | Typhoid fever<br>**Week 1**: insidious rising temp, fever, diarrhea.<br>**Week 2**: fever, rash<br>**Week 3**: continued fever, 2-10% death<br>**Week 4**: fever ↓, slow recuperation | Gm- rods; motile; H, O, **Vi** antigens; lactose -; indole -; ONPG -; $H_2S$ +; citrate + | Transmission by contaminated food/water or direct fecal-oral contact; common in 3rd world countries<br>Pathogenesis: Penetrate epithelial lining of small intestines, are phagocytosed by macrophages (those taken up by PMNs are killed). Grows well in gallbladder due to presence of bile (excellent growth medium for S. typhi). Eventually lyses the cell, then enters blood and reinfects the small intestine, inflame Peyer's patches, resulting in diarrhea, hemorrhages, and perforation | Vi antigen: capsule antigen protecting bacteria inside the phagosome | Dx: Culture of blood, stool, then urine, possibly bone marrow<br>Also, culture on MacConkey agar shows white colonies b/c they're lactose -; Widal test tests for ↑ H&O antibodies, or Vi, which occurs later in the disease.<br>Rx: Chloramphenicol or 3rd generation cephalosporin<br>Prevention: removal of gallbladder in carriers of S. typhi; Short-term vaccine available |
| Salmonella cholerae-suis | Septicemia | Same as S. typhi | Similar to S. typhi | Same as S. typhi | Dx: Same as S. typhi, **but** culture is only done on blood and bone marrow, never on urine or feces.<br>Rx: ciprofloxacin, ceftriaxone, tri/sulfa, cefoperazone |
| Salmonella enteritidis | Gastroenteritis | Gm- rods; motile; H, O, **Vi** antigens; lactose -; indole -; ONPG -; $H_2S$ +; citrate + | Transmission: Reservoirs in humans, chickens, other birds. Frequent method of contamination from chicken to eggs, and then ingestion of raw/uncooked eggs.<br>Pathogenesis: Invasion of intestinal wall, resulting in nausea, vomiting, and diarrhea, bacteria usually do not spread beyond GI tract | Vi antigen: capsule helps bacterial survival | Dx: Clinical picture: acute gastroenteritis without ulceration, sudden onset of diarrhea without other complications; also, cultures of feces will demonstrate PMNs, and stool cultures will show S. enteritidis.<br>Rx: Usually none; self-limiting, lasts 5 days. However, electrolytes and fluids should be watched, especially in children and the elderly. |
| Shigella sonnei | Bacterial dysentery | Gm- rod, O antigen for serotyping; **non-motile**, so **no H-antigen**; lactose -; oxidase -; $H_2S$ -; lys. decarboxylase -; orn. decarboxylase +; β-galactosidase + | Transmission by fecal/oral route: seen in infants. Also spread by contaminated food and water, as seen on passengers on cruise ships and prison populations.<br>Pathogenesis: Bacteria contacts cell and **induces phagocytosis**; it then dissolves the phagosome made in the cell, multiplies, and then kills the cell and moves on. This results in blood, WBCs, pus, and mucous stools; the cell death is primarily of the **colonic mucosa**. | Invasion plasmid antigen D: adhesin facilitating phagocytosis<br>Hemolysin: dissolves phagosome wall<br>Outer membrane protein: polymerizes actin and allows the bacterium to move, in the same manner as Listeria | Dx: Culture of stool sample or swab of rectal ulcer during sigmoidoscopy<br>Rx: Adequate hydration, especially for young children; ampicillin, tri/sulfa if necessary |

## Bacteria Chart: Enteric Bacteria, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Escherichia coli (in general) | Opportunistic infections of lung, kidney, bladder, meninges, & wounds; UTI, esp. acute pyelonephritis; pneumonia, sepsis | Gm- rod; facultative anaerobe; ferments lactose; lysine decarboxylase activity; green sheen on eosin-methylene blue agar; grows well on simple media; motile, peritrichous flagella; **O, H, K** antigens | Opportunistic inf.: Fecal contamination of wounds; E. coli in catheters handled by contaminated hands; traumatic delivery<br>UTI: Self-infection in women; intercourse in women (ahem); prostatic hypertrophy in men over 45 is a risk factor<br>Diarrhea: Only caused by certain strains, but is a killer of infants and toddlers in developing countries (see special strains below) | Endotoxin: From Lipid A (LPS)<br>Adhesins: Pili and P-fimbriae involved in adhesion to mucosa<br>Hemolysin used by E. coli to lyse RBCs and damage a variety of host cells by damaging cell membranes; particular of strains causing acute pyelonephritis | ID: O, H, K (L,A,B) serotypes<br>Dx: Culture of blood or infected wounds for opportunistic infections; midstream clean catch urine culture for UTIs & cystitis (bladder infetions); stool sample culture for diarrhea to identify strain responsible;<br>Rx: antibiotic depends on strain involved; for diarrhea, rehydration is essential to Rx<br>Prevention: strict aseptic techniques, avoiding unnecessary use of catheters |
| Escherichia coli (Enteropathogenic: EPEC) | Diarrhea (**P**ediatric) | Same as above | Transmission: fecal-oral route; seen often in day-care centers and hospitals<br>Pathogenesis: Close adherence to enterocyte membranes via type IV pili, bacteria then cause calcium levels to rise and the microvilli to efface. Then, intimin promotes tight adherence to the enterocyte. Not all that invasive, but results in copious watery disrrhea. **Stool contains no blood**! | Adhesins: Pili and P-fimbriae involved in adhesion to mucosa<br>Type IV pili greatly enhance adhesive capabilities | Dx: Serotyping bacteria from stool culture<br>Rx: Tri/sulfa plus rehydration |
| Escherichia coli (Enteroinvasive: EIEC) | Diarrhea | Same as above, **but** are **lactose -**; also, **non-motile!** | Transmission: Contaminated food/water<br>Pathogenesis: **Resembles Shigella** dystentery infection b/ c blood, WBCs are found in stool; EIEC also attaches to **colonic mucosa** and then invade the mucosa and the lamina propria | Same as E. coli in general, plus…**Shiga-like toxin** | Dx: Watery diarrhea that **progresses** to dysentery; Sereny test tests eye of guinea pig with organisms; if keratoconjunctivitis is elicited, results +.<br>Rx: Tri/sulfa plus rehydration |
| Escherichia coli (Enterotoxic: ETEC) | Diarrhea ("**Travellers**'") | Same as above | Transmission: Ingestion of feces-**contaminated food or water**<br>Pathogenesis: Colonization of proximal small intestine, followed by adherence to mucosa and production of ST or LT. ST/LT causes hypersecretion of fluids and electrolytes, causing watery diarrhea | Heat-stable enterotoxin (ST): non-antigenic, plasmid-encoded toxin; results in watery diarrhea; 2 types<br>- STa: hypersecretion by ↑ activity of guanylate cyclase<br>- STb: mechanism unknown<br>Heat-labile enterotoxin (LT): antigenic, plasmic-encoded toxin; results in watery diarrhea; binds to GM1 gangliosides on the surface of intestinal epithelial cells;↑ adenylate cyclase activity by ADP-ribosylating G-protein on cell membrane in same way as cholera toxin (but structurally unrelated); causes hypersecretion of water and electrolytes | Dx: Tests for ST/LT (usually by gene probes and bioassays)<br>Rx: Tri/sulfa plus rehydration |
| Escherichia coli (Enterohemorrhagic: EHEC) | Diarrhea | Same as above, **but sorbitol negative**! | Transmission: Ingestion of undercooked beef (reservoir in cattle) containing unacceptably high levels of bovine feces<br>Pathogenesis: Adherence to **colonic mucosa**, then producing verotoxins; can result in HUS and bloody diarrhea | Verotoxin: Phage-encoded toxin only in certain strains; 2 types<br>- Verotoxin-1: blocks protein synthesis, killing cells<br>- Verotoxin-2: damages endothelium, resulting in HUS and bloody diarrhea | Dx: ID of sorbitol - E.coli on MacConkey agar, then serotyping to find strain<br>- **O157:H7** in US<br>- O26:H11 in Canada<br>Rx: Tri/sulfa plus rehydration |
| Escherichia coli (Enteroadherent: EAEC) | Diarrhea (mostly seen in SE Asia | Same as above | Transmission: Not described<br>Pathogenesis: Adhere to surface of specific cell types and clumps; non-invasive; produces a low-MW heat-stable enterotoxin that causes watery diarrhea | Low-MW heat-stable enterotoxin different from ST & LT; causes watery diarrhea<br>Bundle-forming pili aid greatly in bacterial attachment | Dx: DNA probe<br>Rx: Rehydratiion, but otherwise not well detailed in text or syllabus |

## Bacteria Chart: Enteric Bacteria, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| **Vibrio cholerae: See toxicogenic bacteria** | | | | | |
| Vibrio parahemolyticus | Gastroenteritis | Gm- curved rods; lives in 2% salt water (**halophile**); multiple antigenic types | Transmission: Ingestion of contaminated shellfish (crabs, oysters, shrimp)<br>Pathogenesis: Toxin production results in abdominal cramping for 2-3 days, diarrhea | Murine toxin | Dx: Growth in 2% salt water<br>Rx: Rehydration, usually no antibiotic Rx, but tetracycline or fluoroquinolones can be administered |
| Vibrio vulnificus | Diarrhea, septicemia (especially during warm weather months: Apr →Nov) | Gm- curved rods; **halophile**, thrives in warm sea water, oxidase + | Transmission: Contaminated raw/uncooked seafood, **especially raw oysters**<br>Pathogenesis: Spread to blood causes septicemia; ingestion results in diarrhea<br>**Susceptble groups**:<br>- Diarrhea: seashore bathers, fishermen, those ingesting raw/undercooked seafood<br>- Septicemia: **Immocompromised** patients have ↑ risk, those with **liver disease** have ↑↑↑ risk | Endotoxin (LPS) | Dx: History of ingestion, with liver disease history or immunocompromise<br>Rx: Tetracycline or 3rd gen cephalosporin; ICU for sepsis |
| Yersinia enterocolitica | Yersiniosis (diarrhea) | Gm- rod, several serotypes | Transmission: Contaminated milk<br>Pathogenesis: Multiplication in mesenteric lymph nodes, then prodution of ST-like toxin of E. coli, resulting in diarrhea which may (but not usually) lead to complications such as arthritis, carditis, septicemia. | Inv proteins promote ability to invade host cells<br>ST-like toxin resulting in watery diarrhea<br>Endotoxic LPS | Dx: Differentiation from appendicitis, cat-scratch disease, salmonellosis, tularemia; stool or blood cultures confirm Dx<br>Rx: 3rd generation cephalosporin plus aminoglycoside, doxycycline, or tri/sulfa |
| Campylobacter jejuni<br>Campylobacter fetus | Diarrhea, septicemia<br>**Note**: C. jejuni is associated with Guillain-Barre syndrome! | Gm- helically curved rods; **gull-shaped** a**ppearance**; motile with single polar flagellum; also microaerophilic, and can be cultured on Campy-BAP at 43°C, 6% $O_2$, 10% $CO_2$ ; nitrate-reducing; urease - | Transmission: Human to human, or from dogs, cats, poultry carcasses; also found in 3-11% of diarrheal stool specimens<br>Pathogenesis: unknown, but results in symptoms ranging from **none** to dysentery; usually no dehydration | Heat-labile enterotoxin: made by C. jejuni | Dx: Growth in Campy-BAP medium<br>Rx: Erythromycin |
| **Helicobacter pylori: See toxicogenic bacteria section** | | | | | |
| Clostridium difficile (toxicogenic strain) | Pseudomembranous enterocolitis, antibiotic-induced diarrhea | Gm+ rod; spore former; obligate anaerobe | Transmission: Invasion after antibiotic Rx (esp. clarithromycin)<br>Pathogenesis: Pathogen colonization of areas once occupied by normal flora | Enterotoxin A: Causes hyper-secretion by ↑ intracellular $Ca^{2+}$ levels, resulting in fluid accumulation in the bowel<br>Enterotoxin B: Cytopathic toxin | Dx: Detection of toxin in stool<br>Rx: Vancomycin |

## Bacteria Chart: Sexually Transmitted Disease-Causing Bacteria

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Neisseria gonorrhoeae | Gonorrhea, ophthalmia neonatorum, pelvic inflammatory disease (PID), disseminated gonorrhoeic infection (skin lesions, arthritis, bacteremia) | Gm- diplococcus; grows in VCN medium (chocolate agar w/vancomycin, cholesthin, nistatin) oxidase +; glucose +; lactose -, sucrose -, maltose -; extremely fragile; can undergo **phase variation** to resist phagocytosis (pilE gene encodes pilin protein, pilS gene contains fragments of internal portions of pilE gene; deletion/interference w/the pilE gene may cause phase variation) | Transmission: Sexual intercourse (humans are only known host of N. gonorrhoeae)<br>Pathogenesis: Pili assist in adherence to squamous (but not ciliated) epithelium; outer membrane proteins assist in adhrence, resulting in inflammation. Gonococci are phagocytosed by epithelial cells and are exocytosed into the submucosa, where they multiply and further erode the epithelium. Antibody and PMN response will eliminate the bacteria, but repeated bouts can result in scarring and possible sterility.<br>Male-specific symptoms: Urethritis, purulent discharge<br>Female-specific symptoms: Urethritis, but infection is often cryptic (purulent discharge in the vagina is only seen by speculum, but is otherwise asymptomatic in women) | Phase variation: avoids phagocytosis, helps in reinfection<br>Pili, outer membrane proteins assist in adherence to epithelium<br>**Note**: hypervariability in pilin antigens make reinfection a grim possibility (original pili are shed, then new ones made to stay ahead of the immune system<br>Capsules are anti-phagocytic<br>Lipooligosaccharide (LOS): like LPS, but **NO O-antigen**<br>Gonobactin: Scavanges iron<br>IgA protease: Cleaves IgA<br>Can hitch a ride on sperm to the uterus, since it can't survive on the vaginal surface. | Dx: Isolation and ID intracellularly in urethral smears<br>Rx: N. gonorrhoeae are penicillin-resistant due to β-lactamase transmitted by plasmid (plasmid-producing N.g., PPNG), or chromosome-mediated penicillin resistance (CMPR); use 3rd generation cephalosporin (ceftriaxone) plus doxycycline |

## Bacteria Chart: Sexually Transmitted Disease-Causing Bacteria, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Treponema pallidum | Syphilis | Gm- spirochete; seen best under darkfield microscopy; axial filament keeps struct. of bacteria helical; entire genome completed in 1998 | Transmission: Intimate sexual contact<br>Pathogenesis: Enters body through Hunterian chancre (**primary syphilis**), which **heals spontaneously**; then, secondary syphilis, shown by rash on hands, feet, chest, mouth; also by fever, headaches, systemic syumptoms. 2° syphilis is still infectious. Then, a period of latency follows. Tertiary syphilis follows, w/gummatous lesions of organs, skin, CNS problems (tabes dorsalis, damage to sensory or motor neurons), cardiovascular disease, and Charcot's joint (swelling and damage to joint and bone). The damage caused by 3° syphilis is **irreversible**.<br>**Note**: Congenital syphilis – infants infected by infected mother; proceed through 1° syphilis rapidly, born with 2° syphilis | Hyaluronidase produced by virulent strains helps bacteria penetrate into host cells<br>Prostaglandin $E_2$ ($PGE_2$) made by bacteria is immuno-suppressive | Dx: ID of spirochetes in chancre or rash; non-treponemal antibody tests such as VDRL, ART, RPR react to the high lvls of cardiolipin in the bloodstream; if non-specific tests are +, then specific tests for Treponemes are used:<br>FTA-ABS: + if antibodies fluoresce<br>MHATP: micro-hemoagglutinination assay: + if you see clumping of cells<br>TPI-T: Looks for loss of motility of organism.<br>**Note**: The reason why non-specific tests are used FIRST is b/c non-specific Abs ↓ after a cure, whereas specific ones do not.<br>Rx: Penicillin, or doxycycline, tetracycline if person allergic to penicillin; 1° syphilis most amenable to Rx; Jarisch-Herxheimer reaction of fever and headache may follow treatment |
| Hemophilus ducreyi | Chancroid (soft chancre) | Gm- rod, **lines up linearly** ("school of fish" appearance); non-spore forming; non-motile; oxidase +, catalase -; requires chocolate agar, with small yellow-gray translucent colonies; likes $CO_2$, humidity; optimal temperature: 33-35°C | Transmission: sexual contact<br>Pathogenesis: Papule results in ulcer; bacteria then spread to lymph nodes, producing buboes. | | Dx: ID from ulcer, aspirate of buboes<br>Rx: erythromycin, ceftriaxone; **no penicillin due to antibiotic resistance** |
| Calymmatobacterium granulomatis | Granuloma inguinale (not common in US or temperate regions) | Short Gm- rod; grows in chains; anaerobic; requires special media; associated with small intracellular **Donovan bodies**; visible under Silver/Wright's stain | Transmission: Sexual contact<br>Pathogenesis: Progressive ulceration of skin & mucous membranes of genital regions | | Dx: Look at **tissue**, not exudates; look under silver/Wright's stains; also look for **Donovan's bodies**<br>Rx: Streptomycin and tetracyclines |
| **Chlamydia trachomatis: See respiratory infection-causing bacteria** | | | | | |
| Ureaplasma urealyticum | Nongonococcal urethritis (NGU) (**10-15% of cases**) | NO CELL WALL; Sterols in membrane stabilize it; slow-growing | Transmission: Sexual contact<br>Pathogenesis: Cause urethritis, also can result in epididymitis in males and cervicitis, endometritis, ectopic pregnancies in females | | Dx: Exclusion of gonococcus and other causes of NGUs; Gm stain with NO Gm - diplococci; culture is really expensive; antigen tests exist; PCR being developed<br>Rx: Tetracycline and doxycycline (recall that this species has NO cell wall, so β-lactams are ineffective |

## Bacteria Chart: Sexually Transmitted Disease-Causing Bacteria, continued

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| (Trichomonas vaginalis) (**NOTE: This is a protozoan!**) | Trichomoniasis | Largest trichomonad; motile, w/ flagella; single nucleus | Transmission: Sexual contact<br>**Note**: Infection usually asymptomatic in men – important in spread.<br>Pathogenesis: Adherence to epithelial cells with subsequent contact-dependent killing process; this results in vaginitis and a vaginal discharge. | Adherence proteins | Dx: Wet smear<br>Rx: Metronidazole |
| Gardnerella vaginalis, (several Bacteroides also) | Bacterial vaginosis: Non-spec. vaginosis caused by at least **3** of the following:<br>- Excessive malodorous discharge<br>- Vaginal pH > 4.5 (normally < 4.5)<br>- Presence of **clue cells** w/scraping (vaginal epithelial cells coated with bacteria)<br>- **Fishy**, amine-like **odor** when KOH is applied to vaginal secretion | Anaerobic, otherwise not much in text or syllabus | Transmission: Sexual contact<br>Pathogenesis: Many causes, so not well described here. | | Dx: Gm variable; culture is expensive<br>Rx: **Metronidazole** b/c it eliminates anaerobes contributing to a mixed bacterial infection; also, clindamycin is used |

## Bacteria Chart: Bacteria causing CNS infections

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins | Dx, Rx, Prevention |
|---|---|---|---|---|---|
| Neisseria meningitidis | Meningitis in closed Areas (dormitories, barracks; septicemia; Waterhouse-Frederich syndrome (catastrophic destruction of the adrenal glands) | $Gm^-$ diplococcus; piliated, encapsulated; ferments **m**altose and **g**lucose | **Humans are the only natural host** | **Capsular polysaccharides** comprise antigenic structure; capsule and IgA protease are virulence factors. | Dx: Ferments **m**altose and **g**lucose; agglutination tests; culture on Thayer-Martin medium |
| H. influenzae | Meningitis in kids and young adults (incidence has ↓↓ b/c of Hib vaccine); epiglottitis, otiitis media, septicemia, conjunctivitis, pneumonia; hearing loss and mental retardation are possible sequelae | $Gm^-$ coccobacillus; encapsulated | **Humans are the only natural host**<br>Transmission by airborne droplets | | Dx: Gm stain of CSF; CSF & blood culture on chocolate agar and 10% $CO_2$, **with factor x** (hemin) **and factor v** (NAD, NADP); shows satellism with S. aureus on blood agar; β-lactamase test; latex bead agglutination<br>Rx: Antibiotics, but treatment may make the condition worse! |
| Streptococcus agalactiae | Meningitis (neonates) | See section on Streptococcus species. | | | |
| E.coli (K1 strain) | Meningitis (neonates) | See section on E.coli. | | | |
| Listeria monocytogenes | Meningitis (neonates) | See section on L. monocytogenes | | | |
| Streptococcus pneumoniae | Meningitis in adults and elderly | See section on Streptococcus species. | | | |

# Bacteria Chart: Anaerobic bacteria of note

Factors suggesting possible anaerobic infection:

- **FOUL-SMELLING DISCHARGE**
- Location of infection near a mucosal surface
- Necrotic tissue, gangrene, pseudomembrane formation
- Gas in tissue or discharges
- Endocarditis with **negative** routine blood cultures (anaerobes can't survive in oxygen environments such as blood)
- Infection associated with malignancy or other process producing tissue destruction (i.e. surgery)
- Infection related to aminoglycoside use (oral, parenteral, or topical)
- Septic thrombophlebitis
- Bacteremia picture with jaundice
- **Black discoloration** of blood-containing exudates, possible red fluourescence under UV light (Prevotella spp., Porphyromonas spp.)
- Presence of sulfur granules (actinomycosis)
- Classical clinical features of gas gangrene (C. perfringens)
- Clinical setting suggestive of anaerobic infection (septic abortion, infection following GI surgery, etc.)

Lab findings suggesting anaerobic infection:

- Unique morphology on Gram stain
- **Failure to grow aerobically** (fluid thioglycollate medium doesn't count)
- Growth in anaerobic zone of fluid media of agar deeps
- Growth anaerobically on media containing kanamycin, neomycin, or panomomycin (7.5 μg/mL of vancomycin for $Gm^-$ anaerobic bacilli)
- **Gas, foul odor, in specimen or culture**
- Characteristic colonies on agar plates when cultured anaerobically
- Young cultures of Porphyromonas spp. and some Prevotella spp. may **fluoresce red** under UV light

| Bacteria | Disease(s) Caused | Characteristics | Transmission and Pathogenesis | Virulence/Toxins |
|---|---|---|---|---|
| Bacteroides fragilis | Abdominal infections | $Gm^-$ rod; nonmotile; encapsulated; has lots of plasmids and phages; grows in 20% bile | | Ability to secrete succinic acid inhibits phagocytosis for itself and other bacteria in the vicinity |
| Prevotella melaninogenica | Dental infections, infections of head, neck, respiratory tract | $Gm^-$ rod; produces dark heme-like pigment; growth **inhibited** by **bile** acids | | Phospholipase A activity releases arachidonic acid; derivatives (PGs, LTs) injure host tissues |
| Porphyromonas gingivalis | Oral infections | | | Extracellular protease cleaves C5, contributing to inflammation |
| Fusobacterium nucleatum | CNS, respiratory, GI infections | ; **pointy ends** | | |
| Peptostreptococcus spp. | Mixed infections in female genital tract | | | |

**Dx**:

- Quick, careful handling, transportation, special media with reducing agents to ↓ redox potential and antibiotics to prevent contamination from facultative anaerobes
- Growth requires special chambers with reduced $O_2$
- Mixed infections: Require multiple IDs

**Rx**:

- Surgical resection o fabscesses and drainage important
- Aminoglycosides **ineffective** against anaerobes
- Bacteroides fragilis: Use clindamycin or metronidazole
- Prevotella, Porphyromonas: Clindamycin, metronidazole, some others too