SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Staphylococcus aureus	clusters of cocci -nonsporulating -nonmotile	positive	facultative anaerobe	coagulase positive catalase positive stains gold on sheep blood agar Tx: methicillin (oxacillin, nafcillin) **MRSA**: vancomycin, linezolid, streptogramins	Virulence: -Protein A binds Fc of IgG - protects from phagocytosis -fibrinogen/collagen binding proteins promote adhesion to host tissues -coagulase leads to fibrin deposition around bacteria, protects from phagocytosis -hemolysins (α,β,γ,δ) destroy RBCs, PMNs, macrophages and platelets -leukocidin destroys WBCs -alpha toxin may mediate sepsis -penicillinase (β-lactamase) inactivates penicillin's β-lactam structure -penicillin binding protein can be resistant to penicillin -hyaluronidase, staphylokinase, lipase, protease break down host tissues Diseases: <u>toxin-mediated</u> 1. Toxic shock by producing TSST-1; myalgias, fever, chills, hypotension, rash 2. Exfoliatin toxin produces "scalded skin syndrome" - skin sloughing 3. Enterotoxins (heat stable) cause gastroenteritis (food poisoning) <u>direct invasion</u> Pneumonia, meningitis, skin infections, endocarditis, sepsis, UTI, osteomyelitis, <u>nosocomial endocarditis, IV user endocarditis</u>
Staphylococcus epidermidis	clusters of cocci -nonsporulating -nonmotile	positive	facultative anaerobe	coagulase negative catalase positive Tx: vancomycin (high resistance)	commensal in nasopharynx in ~30% of population, also on skin elaborate extracellular polysaccharide capsule = biofilm (slime) highly resistant to antibiotics! Diseases: <u>nosocomial</u> 1. polysaccharide capsule great at adhering to<u>prosthetic</u> devices, endocarditis 2. UTI's (esp. from catheters) 3. bacteremia and sepsis 4. meningitis in premature babies (rare)
Staphylococcus saprophyticus	clusters of cocci -nonsporulating -nonmotile	positive	facultative anaerobe	coagulase negative catalase positive Tx: methicillin	common cause of UTI's in sexually active women can cause meningitis in premature babies (rare)
Streptococcus pneumoniae	diplococci (pairs) -encapsulated -nonsporulating -nonmotile	positive	facultative anaerobe	catalase negative alpha hemolytic sensitive to optochin sensitive to bile (+) Quellung Tx: erythromycin (covers other pneumo's)	can cause meningitis, common in older patients - significant inflammation Virulence: polysaccharide capsule interferes with phagocytosis cell wall contains teichoic acid with phosphorylcholine C (unique) - inflammation pneumolysin binds cholesterol in host cell membranes IgA proteases, PMN proteases positive Quellung reaction (used to detect encapsulated bacteria) Diseases: 1. Pneumonia - chills, fever, pain on breathing, SOB, bloody sputum alveoli filled with WBC's, can be seen on CXR as consolidation 2. Meningitis 3. Otitis media 4. Sepsis

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Streptococcus pyogenes (Group A)	strips of cocci -encapsulated	positive	micro-aerophilic	catalase negative beta-hemolytic sensitive to bacitracin Tx: penicillin	commensal in nasopharynx, rectum Virulence: M protein most important - interferes with phagocytosis hyaluronic capsule also interferes with phagocytosis antibodies can be made against M protein, but only for that strain! Protein F1, lipoteichoic acid - mediate binding to fibronectin on epithelial cells streptolysins O & S - responsible for beta hemolytic activity (only O is antigenic); SLO titer (aka ASO titers) measure recent infection pyrogenic exotoxins - cause scarlet fever Diseases:1. Pharyngitis common ("strep throat") - fever, lymphadenopathy, purulent exudate on tonsils, petechiae, headache 2. Scarlet fever - caused by exotoxins; rash from trunk→ extremities (not face), fever, pharyngitis, impetigo (facial pimple-like pustules) 3. Toxic shock syndrome - caused by exotoxins, occurs in the presence of infection (diff. from other TSS) 4. Rheumatic fever - follows untreated strep A infections Ab's against strep cross-react with myocardial antigens fever, myocarditis, arthritis, chorea, subQ nodules, rash appears 10-20 years after initial myocarditis as rheumative valvular dz, so need lifetime penicillin prophylaxis 5. glomerulonephritis - antibodies develop→ immune complex deposition occurs one week after primary infection fluid retention, blood in urine
Streptococci agalactiae (Group B)	strips of cocci -encapsulated	positive	facultative anaerobe	catalase negative beta-hemolytic Tx: penicillin 3rd gen. cephs	commensal in women of child-bearing age can cause meningitis, pneumonia, and sepsis in newborns
Enterococci	pairs of cocci	positive	facultative anaerobe	catalase negative mostly γ-hemolytic **R**: cephalosporins! Tx: ampicillin + aminoglycoside, linezolid, streptogramin	part of normal GI flora can cause meningitis, UTIs, bacteremia, and infectious endocarditis also can infect biliary tract and peritoneum highly resistant, tough to treat - MDR = multi-drug resistance VRE = vancomycin resistant enterococci
Viridans streptococci		positive	facultative anaerobe	catalase negative alpha-hemolytic Tx: penicillin	commensal in nasopharynx and sometimes GI Virulence: produces large amounts of dextran, an extracellular polysaccharide that helps in adhesion to platelet-fibrin vegetations common cause of dental cavities and therefore subacute bacterial endocarditis
Listeria monocytogenes	rods	positive	facultative anaerobe	catalase positive **R:** cephalosporins! Tx: ampicillin trimethoprim-sulfamethoxazole	found in contaminated food that was fertilized with manure commonly found in Mexican cheese, other dairy/poultry products Virulence: intracellular growth, lives in cytosol Diseases: Meningitis

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Clostridium tetani	rods -sporulating -motile	positive	anaerobic	 Tx: antitoxin penicillin	toxin-mediated diseases usually follows puncture wound by rusty nail or anything contaminated with spores toxin released is called tetanospasmin causes sustained muscular contraction (called tetany) and lockjaw by inhibiting inhibitory interneurons can cause respiratory failure
Clostridium botulinim	rods -sporulating	positive	anaerobic	 Tx: antitoxin penicillin	toxin-mediated diseases found in soil and water, ingested from home-canned vegetables and honey neurotoxin ingestion causes block of Ach release - flaccid muscle paralysis Diseases: Botulism is not food poisoning! Symptoms include weakness, dizziness, dilated pupils, dry mouth - no fever, cognitive abilities intact adults: food contains already synthesized toxin babies: honey contains spores - toxin synthesized in baby's GI tract Wound botulism (eg, in IV drug users) introduced into skin by trauma death by diaphragmatic paralysis
Clostridium perfringens	rods -sporulating	positive	anaerobic	 Tx: penicillin + clindamycin	toxin-mediated diseases produces alpha toxin (lecinthinase) which hydrolyzes membrane lecithin, resulting in cell death Diseases: 1. skin infections (gaseous gangrene) - bacteria infects deep wounds likes anaerobic environment and produces gas; intense pain in wounds can cause shock, renal failure, death from toxin production 2. food poisoning if ingested in food; fairly benign because toxin is heat labile
Clostridium difficile	rods -sporulating	positive	anaerobic	 Tx: metronidazole oral vancomycin	<5% population colonized, increases to 25-70% in hospitalized patients!! secretes toxins A and B (B needs A for toxicity); toxin A is directly cytotoxic, chemotactic for PMNs and causes inflammation and diarrhea; toxin B is cytotoxic to colonic cells spores are resistant to acidic pH of stomach (spores cause recurrences) antibiotic use causes colonization and overgrowth of C. diff - causes severe diarrhea, abdominal cramping, and fever
Actinomyces	irregular, filamentous -nonmotile	positive	anaerobic	 Tx: penicillin	commensal in oropharynx, GI tract, female genital tract frequent sites of infection: teeth, thorax, pelvis (women) causes eroding abscesses (draining sinus tracts) in the mouth and GI abscesses contain orange-yellow granules = "sulfur granules" that contain colonies of actinomyces and cellular debris (no actual sulfur)
Bacterioides (fragilis)	bacillus -nonsporulating	negative	anaerobic	 Tx: metronidazole clindamycin cefoxitime chloramphenicol	commensal in gut flora Virulence: encapsulated, but NO endotoxin! (No lipid A) Diseases: only occurs when bacteria are introduced into peritoneal cavity (during surgery, gun-shot wound, etc) forms abscesses in gut, fever may be present due to inflammation

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Escherichia coli	bacillus	negative	facultative anaerobe	oxidase negative beta-hemolytic ferments glucose and lactose Tx: cephalosporins aminoglycosides TMP-SMX fluoroquinolones aztreonam ampicillin	commensal in GI tract can cause UTIs - pili bind to gal-gal in uroepithelium also nosocomial pneumonia and sepsis (LPS), meningitis, diarrhea Enterotoxigenic E. Coli (ETEC): caused by enterotoxigenic E. Coli -profuse, secretory, noninflammatory diarrhea - toxins disrupt absorptive/ secretory functions of GI mucosal cells, but do not kill them -uses type III injection of Intimin receptor so that ETEC can bind with intimin -type 1 *cfa* pili bind mannose residues, helps in adhesion -toxin = ADP-ribosylating enzyme -heat-labile toxin (LT): like cholera toxin, increases cAMP→ water out of cell -heat-stable toxin (ST): increases cGMP→ water out of cell -disease manifests as "rice water" diarrhea, no inflammation, no fever Shiga Toxin E. Coli (STEC = EHEC = 0157:H7): shiga-like toxin producing EC -toxin interferes with host cell protein synthesis -intimin protein binds tightly to host cell; induces actin cytoskeleton rearrangement for uptake of organism into cell -needs very few organisms to cause disease; found in bad water, meat, juice -disease manifests as bloody diarrhea, inflammation, damaged GI mucosa -potential to develop Hemolytic Uremic Syndrome - hemolytic anemia, thrombocytopenia, acute renal failure Enteroinvasive E. Coli (EIEC): bacteria invade and destroy intestinal epithelial cells -plasmid encoded virulence is same as *Shigella,* some shiga-toxin produced -disease manifests as inflammatory, bloody diarrhea with white cells fever K1 subtype (capsular antigen) can cause meningitis in neonates
Campylobacter jejuni	curved rods -motile	negative	micro- aerophilic	oxidase positive Tx: fluoroquinolones macrolide	commensal in gut flora commonly found in GI tracts of animals, contaminated water Virulence: invasive, LT enterotoxin increases cAMP→ more secretions, cytotoxin destroys mucosal cells Disease: Diarrhea - common cause! Bloody, secretory
Klebsiella pneumoniae	rods	negative	facultative anaerobe	ferments lactose Tx: 3rd generation cephalosporins fluoroquinolone	commensal in gut flora Virulence: encapsulated (O antigen) , but nonmotile Diseases: 1. Sepsis (nosocomial) 2. UTI's from catheters 3. Pneumonia - bloody sputum (usually), destruction of lung tissue - common in alcoholics

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Psuedomonas aeruginosa	rods	negative	obligate aerobe	oxidase postive doesn't ferment anything! Tx: ceftazidime piperacillin ticarcillin/clavulanate imipenem aztreonam fluoroquinolone	commensal in GI tract opportunistic pathogen, affects hospitalized patients Virulence: weak invasive ability, but once inside many exotoxins are produced quorum sensing exoproducts = alkaline protease, elastase, exotoxin A (which stops protein synthesis in target cell), exoenzyme S, phospholipase (hemolysins), neuraminidase adhesins = pili, flagella, biofilm exopolysaccharide Diseases: 1. Pneumonia, commonly in CF patients or otherwise immunocompr. 2. Osteomyelitis - common in diabetics, children, IV drug users 3. Sepsis - high mortality 4. UTIs, pyelonephritis 5. Endocarditis - right-sided heart valves in IV drug users Misc: burn-wound infections, otitis, corneal infections Very difficult to treat because resistant to many drugs!
Salmonella (non-typhi)	rods	negative	facultative anaerobe	nonlactose fermenter produces H2S Tx: quinolones ceftriaxone TMP-SMX ampicillin	never commensal facultative intracellular parasite, lives within macrophages in lymph nodes commonly found in raw eggs and uncooked chicken - from GI tracts of animals taken up by M cells in gut or direct invasion, multiply, hematogenous spread, replication in liver and spleen, excretion into bile, repeat entry into gut lumen Virulence: Vi antigen is polysaccharide capsule protecting from phagocytosis -prevents phagosome-lysosome fusion once inside Disease: 1. gastroenteritis:watery diarrhea with abdominal pain, nausea; if ileal inflammation, diarrhea will be mucoid 2. Sepsis 3. Osteomyelitis - especially in sickle cell patients 4. Meningitis 5. Typhoid-like fever in immunocompromised patients (eg, HIV)
Salmonella typhi	rods	negative	facultative anaerobe	Tx: quinolones ceftriaxone TMP-SMX ampicillin	resevoir is human, but not commensal transmission is fecal-oral route see above for life cycle and virulence Disease: Typhoid fever - fever, abdominal, liver/spleen enlargement
Shigella	rods	negative	facultative anaerobe	Tx: fluoroquinolones TMP-SMX ampicillin tetracycline	never commensal cell-mediated disease, needs very few organisms for infection transmission via fecal-oral route from infected individual - common in daycare Virulence: Ipa (invasion plasmid antigens) bind to host cells, mediate invasion into M cells; lives in the cytosol -IpaB induces apoptosis -induces rearrangment of actin cytoskeleton for uptake into host cells -IcsA (intracellular spread protein A) propels through to adjacent cells -IcsB for actual lysis of protrusion into next cell -replicates intracellularly→ produces pore-forming cytotoxin to escape phagosome -death of mucosal cells - interference with cellular mechanisms, apoptosis Disease: dysentery = bloody, mucoid, *small volume* diarrhea mucosal destruction results in PMN recruitment and inflammation

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Vibrio cholera	curved rods with long flagella	negative	facultative anaerobe	oxidase positive nonlactose fermenter Tx: doxycycline fluoroquinolones	transmitted via fecal-oral route genome consists of 2 chromosomes; larger encodes most virulence factors, smaller contains essential genes uses type II secretion Virulence: pili, cholera toxin (also called choleragen) -these bacteria do not invade mucosal cells, they secrete toxin onto them -cholera toxin = B subunit binds to GM1 ganglioside on mucosal cells, allowing A subunit entry into cells (mechanism unknown) -A subunit ADP ribosylates G protein→ activates adenylate cyclase→more NaCl and water secretion -other toxins include zot toxin (zonula occludens toxin) Disease: <u>toxin-mediated</u> 1. watery diarrhea (no WBCs) with severe dehydration and hypotension
Neisseria meningiditis	kidney-shaped diplococci -encapsulated	negative	strict aerobes	oxidase positive ferments glucose and maltose Tx: penicillin ceftriaxone	Commensal in respiratory & GI tracts, spread via cough droplets LOS (endotoxin) for adhesion and is immunogenic; survives intracellularly! Needs IgG and/or complement for efficient phagocytosis Complement (C5-C9) deficienct people are more susceptible to Neisseria Diseases: 1. Meningitis: has IgA protease, and mimics neuronal adhesion molecules enters via nasopharynx - strains A,B,C,D,X,Y,W135,129E vaccines available for A,C,Y,W135 - not for B!<u>B is not immunogenic</u> No vaccine for group B, also won't be detected on latex agglutination test Groups B, C, Y, W135 cause endemic dz; Group A strains cause epidemics 2. Bacteremia which can lead to Waterhouse-Friedrichsen syndrome (DIC which leads to adrenal failure and sever hypotension)
Neisseria gonorrhoeae	kidney-shaped diplococci	negative	strict aerobes, needs CO2!	oxidase positive catalase positive ferments only glucose culture on Thayer-Martin media: chocolate agar vancomycin colistin nystatin Tx: ceftriaxone (+ doxycycline for chlamydia) fluoroquinolone erythromycin eye drops	sexually transmitted bacteria Virulence: LOS (lipo-oligosaccharide) helps in adhesion and is immunogenic Protein I: *por* proteins form pores in outer membrane Protein II: *<u>opa</u>* proteins facilitate adhesion and binding Protein III: *imp* proteins stimulate antibacteriocidal ab's T1 and T2 <u>pili</u> for virulence - spread by sexual contact almost exclusively OMP (outer membrane proteins) I and II also immunogenic Men: 95% of infected men are symptomatic purulent urethral discharge and dysuria gram stain very sensitive in detection Women: 50% of infected women are symptomatic purulent vaginal discharge from infected cervix, dysuria gram stain not useful due to normal flora can progress to PID (pelvic inflammatory disease) - risk of sterility Can cause -gonococcal bacteremia -septic arthritis (bacteria*inside* WBC's)

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Treponema pallidum	spirochete	negative	aerobic	VDRL test for non-treponemal antibodies FTA test for treponemes (may be + in people who have Lyme Disease) Tx: erythromycin	transmitted by sexual contact, through tiny breaks in epithelium treponemes multiply in the dermis →enter bloodstream painless chancre (papule) appears at site of inoculation Disease: Syphilis - if untreated 1. Primary - inguinal lymphadenopathy, painless genital ulceration, skin lesions appears at ~3 weeks, although serologic tests may be negative 2. Secondary - flu-like illness: fever, malaise, arthralgias, rash, lymphadenopathy rash on palms and soles, condylomata lata (genital lesions), other: nephrosis, cranial nerve palsy, meningitis, hepatic dysfunction 3. Tertiary a) neurosyphilis: meningitis, people become schizophrenic-like b) cardiovascular: damages aorta→ root dilation, aortic regurgitation c) gummatous: granulomatous-like lesion in soft tissues
Chlamydia trachomatis	spirochete	not seen on gram	intracel-lular	tough to culture test for chlamydial antibodies or complement fixation test Tx: doxycycline erythromycin for eye drops ceftriaxone for gonnorhea	common cause of PID→ can cause pneumonia in infant born to mother with untreated chlamydial infection occurs frequently with gonnorhea, must clinically treat both infections contains LPS but not really gram-negative; has trilaminar outer membrane look for intracellular inclusions in cells Life cycle unique: elementary body is infectious particle, gets inside non-ciliated cells and alveolar macrophages via endocytosis, converts to reticulate body, uses host cell energy to replicate by binary fission, converts back to elementary body for further spread (same for both chlamydial species) Diseases: 1. Urethritis - asymptomatic or symptomatic with dysuria, discharge 2. neonatal pneumonia - coughing, no fever, diffuse infiltrates on CXR, rapid RR 3. conjunctivitis - neonatal or not, can cause blindness, spread by hand 4. trachoma - chronic conjunctivitis, causes blindness 5. Cervicitis, PID - can scar fallopian tubes, leading to infertility 6. Lymphogranuloma venereum - painful lesions caused by types L1, L2, L3 may spread to lymph nodes
Chlamydia pneumoniae	spirochete	not seen on gram	intracel-lular	 Tx: erythromycin doxycycline	specific strain (TWAR) is responsible for causing pneumonia see above for life cycle transmission is via aerosolized droplets, prolonged incubation up to 21 days Disease: pneumonia - nonspecific upper respiratory symptoms, eg runny nose, sore throat, no fever; can persist to chronic cough; lobar consolidation on CXR linked to atherosclerosis - elementary bodies found in some plaques
Hemophilus influenzae	coccobacillary pleomorphic	negative	micro-aerophilic	gram stain, + Quellung test Tx: 2nd or 3rd generation cephalosporins TMP-SMX macrolides!	there are both encapsulated and unencapsulated forms (carbohydrate capsule) Virulence: CHO capsule Disease: unencapsulated: otitis media, sinusitis encapsulated: certain types (B, E) can cause meningitis in unvaccinated population - severity correlated with levels of TNF in CSF also sepsis, pneumonia

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Hemophilus ducreyi	pleomorphic rods	negative	micro-aerophilic	Tx: macrolide, ceftriaxone ciprofloxacin	sexually transmitted, occurs more in males Disease: Chancroid - after 2-5 days, a macule appears which ulcerates regional lymphadenopathy, possibly satellite lesions increases HIV risk due to open sores
Legionella (usually pneumophila or micdadei)	rods	negative	aerobic	stain with Dieterle's silver stain grow in supplemented media (Fe, cysteine) detected by DFA (direct fluorescent antibody test) if 3+ weeks into illness Tx: macrolide	transmissible via aerosolized droplets intracellular pathogen lives in alveolar macrophages and monocytes binds to complement receptor on MΦ's, endocytosed, prevents phag/lys fusion facultative intracellular ubiquitous aquatic saprophyte - lives inside amoebas! Virulence: produces proteolytic enzymes, phosphatase, lipase, nuclease which kill cells when vacuole is lysed - causes microabscess formation need T cells to kill organism Diseases: 1. Legionnaires' disease - presents as pneumonia or flu-like illness high fevers, cough, rigors, headache progression to elxtrapulmonary sites = mental confusion, diarrhea, abd. pain death from respiratory failure, renal failure, shock 2. Pontiac fever - self-limited flu-like illness, resolves spontaneously
Mycoplasma pneumoniae	pleomorphic	not seen on gram	strict aerobe	Tx: erythromycin	no cell wall - resistant to antibiotics that target cell wall cell membrane contains sterols not found in other bacteria does not culture well, usually detected via serology cold agglutinin test: these are autoantibodies formed after infection; they are IgM cross-reactive to the I antigen on RBCs frozen blood will have flaky white appearance (antibody precipitate) extracellular bacteria - destroys cilia and epithelial cells Virulence: P1 protein binds to sialic receptors on RBCs and respiratory epithelium can act as super antigen, resulting in PMNs, MΦ's, TNF, IL-1, IL-6 Disease: 1. Upper respiratory infection (esp. young children) - low grade fevers, malaise, headache, nonproductive cough, sore throat, râles 2. Walking Pneumonia (atypical) - lower resp. tract involvement; same symptoms as above; CXR looks worse (diffuse infiltrate) than patient appears

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Bordetella pertussis	coccobacillary pleomorphic	negative	aerobic	Tx: supportive - antibiotics don't really change course of illness macrolide *if before* paroxysmal phase	transmissible via aerosolized droplets disease caused by toxins that cause tissue damage; no bacteremia Virulence: pertussis toxin and filamentous hemagglutinin help organism bind to ciliated resp. epithelial cells -pili and pertactin aso mediate adhesion -pertussis toxin = A unit: toxic S1 subunit↑ cAMP - increased mucus and resp. secretions B subunits (binding S2-5 subunits) -adenylate cyclase toxin inhibits PMN chemotaxis, phagocytosis, killing -hemolysin toxin causes more cAMP -heat-labile toxin causes local tissue destruction -tracheal cytotoxin destroys ciliated cellls, causes release of IL-1→ fever -lipopoysaccharide (lipids A and X) activate complement and cytokine release Disease: "Whooping Cough" 1st (catarrhal) stage - looks like common cold, low-grade fever 2nd (paroxysmal) stage - "whoop" sounding coughs, vomiting after common 3rd (convalescent) stage - cough subsides, may get pneumonia (other bact.), seizures, encephalitis
Helicobacter pylori	spirochete	negative	micro-aerophilic	Urea breath test, PCR, serology, dental plaque test Tx: Pepto B. + metronidazole + amoxicillin	fastidious organism Virulence: ureases, can live in low pH, has specific adhesins for gut mucosa Cytotoxin-associated gene (Cag) protein causes IL-8 response Disease: 1. Peptic ulcers 2. erosive gastritis 3. gastric carcinomas/lymphomas
Borrelia burgdorferi (treponeme)	spirochete	negative	micro-aerophilic	serology - look for antibodies Tx: tetracycline or IV ceftriaxone if disseminated	transmissible via tick bites - mice, deer, cattle horses, dogs Disease: Lyme Disease 1. local erythema migrans 2. early disseminated - cardiac block, myocarditis, myopericarditis musculoskeletal - joint pain, esp. in knee, joint effusion neurological - meningitis, Bell's palsy, neuropathy 3. chronic disseminated (years after bite) destructive arthritis, cardiomyopathy, stroke, dementia
Borrelia recurrentis	spirochete	negative	micro-aerophilic	blood smear Tx: tetracycline	transmission via human louse bite Virulence: antigenic variation Disease: Relapsing fever with lymphadenopathy, rash, headache
Rickettsia rickettsii	pleomorphic	negative	intracel-lular	hard to culture and stain diagnose by skin biopsy, PCR, serology Tx: doxycycline chloramphenicol	obligate intracellular pathogens transmissible via tick bite - bacteria regurgitated into skin bacteria carried via lymphatics then blood vessels→ invades endothelial cells Disease: Rocky mountain spotted fever - blanching petechial rash starts on wrists, ankles, soles, palms, then spreadsproximally 1st erythematous macule, 2nd macular papule, 3rd hemmorhage 7-14d incubation, then sudden onset of fever, headache, malaise, myalgia GI problems, hepatomegaly, jaundice in later stages

SPECIES	MORPHOLOGY	GRAM	O2 REQ.	DIAGNOSTIC TEST	STUFF TO KNOW
Rickettsia akari	pleomorphic	negative	intracel- lular	Tx: doxycycline chloramphenicol	obligate intracellular pathogen transmissible via mite bite - bacteria regurgitated into skin bacteria carried via lymphatics then blood vessels invades endothelial cells Disease: Rickettsialpox - papular-vesicular rash with fever, headache, lymphadenopathy, chills, myalgias bite and rash looks like scabby chicken pox disease self-limited
Ehrlichia		negative	intracel- lular	Tx: doxycycline (**R**: chloramphenicol)	obligate <u>intracellular</u> pathogen, prevents phagosome-lysosome fusion erlichiosis has same symptoms as rickettsial disease transmissible via tick bite, spread via lymphatics into blood infects granulocytes and monocytes Disease: "Spotless" fever - thrombocytopenia, leukopenia, elevated LFTs, (but sometimes rash), elevated fever, severe headache