

Medfools Bacteriology a la chart for the USMLE I

Adapted from notes from UCLA., with additional corny mnemonics

<i>Staphylococcus aureus</i> (virulent)		(nonmotile, nonsporeforming, facultative anaerobe)			Gm+ cocci	
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
<p>*Skin infections: impetigo, cellulitis, erysipelas, abscess, furuncle, carbuncle</p> <p>*Bacteremia/sepsis: hematogenous spread</p> <p>*Acute endocarditis: DESTRUCTIVE (compare to <i>S.viridans</i> and <i>S.faecalis</i>)</p> <p>*Pneumonia –damaging process, cavitations, empyema, effusions</p> <p>*Osteomyelitis/septic arthritis- hematogenous and traumatic spread</p> <p>*Food poisoning – 1-8 hr onset, vomiting, <u>preformed</u> toxin</p> <p>*Tox shock syndrome- fever, vomiting, diarrhea, diffuse <u>erythematous rash</u></p>	<p>Gm + cocci in grapes/clusters</p> <p>Catalase + coagulase +</p>	<p>Ubiquitous in environment; normal flora of skin/nose</p> <p>Spread through lesions, fomites</p>	<p>Enterotoxin- vomiting, diarrhea, heat resistant, (actually released in gut)</p> <p>TSST-1 – tampon use, wounds, <i>superantigen</i></p> <p>Exfoliatin- scalded skin</p> <p><u>TISSUE SPREAD:</u></p> <p>Alpha toxin(leththrinase)- skin necrosis;hemolysis</p> <p>Hyaluronidase- degrades proteoglycans</p> <p>Fibrinolysin- lysis fibrin clots</p> <p><u>IMMUNE EVASION:</u></p> <p>Protein A- binds IgG-Fc, blocks opsonization and complement fixation</p> <p>Coagulase- activates prothrombin</p> <p>Hemolysin- destroys RBCs, PMNs, M0s, platelets</p> <p>Leukocidin- destroys WBCs</p>	<p>Gm + cocci in grapes, Catalase differentiates from Strep.</p> <p><i>S.aureus:</i> Beta hemolysis, coagulase, Yellow (Au) pigment</p> <p>(coagulase <i>causes coagulation!</i>)</p> <p>Coagulase neg:</p> <p><i>S. epidermidis:</i> novobiocin sensitive “sensitive skin”</p> <p><i>S. saprophyticus:</i> Novobiocin resistant</p>	<p>Beta lactamase production is common! Use methicillin, nafcillin, dicloxacillin</p> <p>MRSA- vancomycin</p>	<p>none</p>

S. epidermidis: associated w/ IV catheters, damaged/prosthetic heart valves: **INSIDIOUS** onset, **Nosocomial**, **LESS virulent**.
Blood culture Contaminant

S. saprophyticus: **Community acquired UTI** in young women

Streptococcus viridans (GABHS) (nonmotile, nonsporeforming)

Gm+ cocci

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
<p>*Pharyngitis- “strep throat”, erythema, tonsillar exudate, fever</p> <p>*Skin/soft tissue infections- impetigo, cellulitis, necrotizing fasciitis</p> <p>*Scarlet fever- centrifugal, red rash, erythrogenic toxin, slap cheek, strawberry tongue</p> <p>*Tox shock syndrome- clinically like <i>Staph</i> TSS</p> <p>*Rheumatic fever- fever, myocarditis, polyarthritis, chorea, subcutaneous nodules, erythema marginatum rash. Mitral valve disease follows pharyngitis, NOT skin infections. Abs vs. bacteria cross react w/ joint and heart antigens</p> <p>*Acute GN- hypertension, hematuria, edema of face/ankles. Follows both pharyngitis AND skin infections. Cross reactive antigens deposited in GBM.</p>	<p>Gm + cocci in chains or pairs</p> <p>Beta-hemolytic are classified by Lancefield groups (A,B,D) according to C-carbohydrates</p>	<p>Human throat/skin, Transmission by respiratory droplets</p>	<p>Hyaluronidase- degrades proteoglycans (TISSUE SPREAD)</p> <p>Erythrogenic toxin- scarlet fever, lysogenized <i>S.pyogenes</i></p> <p>Streptolysin 0- results in beta hemolysis, target of ASO antibodies</p> <p>M protein- antibody target, but inhibits complement/phagocytosis</p> <p>Streptokinase- converts plasminogen to plasmin, dissolves fibrin clots</p> <p>IgA protease</p> <p>“HE’S an MSI”</p>	<p>All Strep are Catalase –</p> <p>Beta hemolysis and Bacitracin sensitivity point to GABHS, esp with inc. ASO titer.</p>	<p>Penicillin to prevent rheumatic fever.</p> <p>Penicillin DOES NOT treat post strep disease or enterococcus.</p>	
<i>S. agalactiae (Group B strep)</i>						
Neonatal meningitis, sepsis pneumonia	Beta-hemolytic	Female urinary tract				
<i>S. faecalis (enterococcus)</i>						
Subacute endocarditis, UTI “Oh crap! I’ve got Heart problems!”	Not hemolytic	GI tract		Grows in 6.5% NaCl		
<i>S. bovis (group D)</i>						
UTI	Not hemolytic	GI tract		Hydrolyze esculin in presence of bile. NOT grow in 6.5% bile		
<i>S. pneumoniae (pneumococcus)</i>						
Lobar pneumonia, ADULT meningitis, URI (kids)	Alpha-hemolytic	Nasopharynx	85 different capsular polysaccharides	Quellung rxn		23 valent vaccine, for AIDS, elderly, asplenic
<i>S. Mutans , mitis (Viridans group)</i>						
Subacute endocarditis, caries	Alpha-hemolytic	Oropharynx				

<i>Neisseria</i>		<i>(Chocolate agar, Oxidase +, kidney bean shape)</i>			<i>Gm- cocci</i>	
N. meningitidis (meningococcus)						
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
<p>*Meningococcemia- fever, arthralgias, myalgias, petechial rash, inc. in people w/ complement deficiencies</p> <p>*meningitis- fever, headache, stiff neck, photophobia, inc.PMNs in CSF</p> <p>* Waterhouse-Friedrichsen- fever, purpura, DIC, adrenal insufficiency due to <i>bilateral adrenal hemorrhage</i>, shock, death (like a bad meningococcemia)</p>	<p>Gm – cocci kidney beans.</p> <p>Thayer-Martin, chocolate agar</p>	<p>Airborne droplets, colonized nasopharynx, establishes carrier states in some</p>	<p>Polysaccharide capsule, endotoxin (LPS), IgA protease</p> <p>Capsular polysaccharides are antigenic serve as markers for classification.</p>	<p>Ferments maltose</p> <p>Presumptive diagnosis by Gm stain of petechiae or CSF</p> <p>LATEX agglutination test b/c capsular polysaccharides</p>	<p>Penicillin or Ceftriaxone (G3)</p>	<p>Chemoprophylaxis with Rifampin (excreted into saliva)</p> <p>Polysaccharide vaccine in military recruits.</p>
N. gonorrhoeae (gonnococcus)						
<i>(most common notifiable disease in US)</i>						
<p>Males- symptomatic dysuria, penile discharge b/c of urethritis. Leads to epididymitis, prostatitis, urethral strictures</p> <p>Female- asymptomatic, vaginal discharge, dyspareunia, due to cervicitis, Infertility, PID, ectopic, tubo-ovarian abscess, perihepatitis (Fitz-Hugh-Curtis syndrome), ophthalmia neonatorum</p> <p>Both: Septic arthritis</p>	<p>NO CAPSULE</p> <p>Gm – cocci kidney beans.</p> <p>Thayer-Martin, chocolate agar</p>	<p>Sexual transmission</p> <p>OFTEN coexistent WITH Chlamydia AND Syphilis (tx w/ tetracycline or chloramphenicol)</p>	<p>Pili/fimbriae (ANTIGENIC variation)</p> <p>LPS</p> <p>OMPs</p> <p>IgA protease</p> <p>NO CAPSULE!</p>	<p>Men: Gm – diplococci in PMNs</p> <p>Does NOT ferment maltose</p> <p>No serologic testing, no capsule!</p>	<p>Ceftriaxone (G3) b/c penicillinase producing <i>N.gonorrhoeae</i> PPNG common</p>	<p>Erythromycin eye drops in newborns (also protects vs. Chlamydia)</p> <p>No Vaccine.</p>

NOTE: bacterial meningitis: 0-6 months (Group B *Strep*, *E.coli*, *Listeria*); 6 months – 3 years (*H.influenzae B*), 3-15 years (*N. meningitidis*), >15 years (*S. pneumoniae*)

Clostridium (Anaerobic, spore-forming, with Exotoxin)

Gm+ Rods

C. tetani

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Tetanus – tetany, risus sardonicus “joker smile”, exaggerated reflexes, respiratory failure		Spores, ubiquitous in soil, enter wounds and germinate in anaerobic environment of necrotic tissue	Tetanus toxin travels intra axonally to CNS, blocks release of inhibitory glycine neurotransmitter		Penicillin , ventilatory support, muscle relaxants Tetanus immune globulin , preformed Ig	Tetanus toxoid (formaldehyde treated tox)

C. botulinum

Botulism “flaccid paralysis”, descending weakness, diplopia, flaccid paralysis, resp failure. Wound botulism- spores to wounds, germinate, release toxin Infant botulism- ingestion of spores in honey- floppy baby		Spores, in soil, inadequate sterilization of canned foods. Alkaline veggies, smoked fish.	Botulinum toxin ingested preformed. Tox spreads in blood, to nerves blocks Ach RELEASE Toxin can be used to Tx torticollis, blepharospasm		Antitoxin , ventilatory support NO PENICILLIN!! Will burst cells and release toxin	Watch swollen cans!
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C. perfringens

Gas gangrene (myonecrosis): war wounds, septic abortions Food poisoning- ingestion of cooking resistant spores in foods. Watery diarrhea, cramps, little vomiting	Results in crepitus- gas production and Hemolysis	Normal flora of colon and vagina	Alpha tox- lecithinase degrades cell membranes- hemolytic	Morphology, exudate smears, culture, sugar fermentation, organic acid production	Debridement, O2 gas, Penicillin	
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C. difficile

Antibiotic associated pseudomembranous colitis- esp in hospitalized pts.		Normal flora in 3% of people	Suppression of normal flora allows overgrowth, usually by clindamycin, ampicillin, cephalosporins Exotox A (severe diarrhea) Exotox B (damage to colonic mucosa)	ID C-diff tox in stool	Metronidazole- poorly absorbed orally, inc. colonic dose Vancomycin	
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Bacillus (Aerobic, spore-forming, with Exotoxin)						Gm+ Rods
B. anthracis						
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Woolsorter's disease- pulmonary anthrax, pneumonia	Large w/ square ends, nonmotile	Common in animals. Humans infected by spores on animal products (skins/hides) Transmission through skin, GI tract, respiratory tract	Antiphagocytic capsule made of d-glutamate [only one w/ Amino acids!] (not a polysaccharide) Tripartite anthrax toxin: protective antigen, lethal factor, edema factor. Protective factor inhibits phagocytosis.	Morphology and blood agar growth.	Penicillin	Sterilization of animal products, and vaccination of animals. Vaccine (protective antigen) for humans at risk
B. cereus						
Vomiting with 4 hr incubation period (like <i>S.aureus</i>)- heat stable toxin--	Distinguished from <i>B. anthracis</i> by motility and lack of capsule.	Spores on grains survive cooking and germinate when food is warmed.	Preformed heat-labile enterotoxin (like <i>E.coli</i> , <i>Cholera tox</i>) - diarrhea		Treat symptoms	Avoid reheated rice

Corynebacterium diphtheriae (nonmotile, nonsporeforming, Chinese)						Gm+ Rods
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Diphtheria – throat inflammation, gray fibrinous exudate (pseudomembrane), airway obstruction, myocarditis , recurrent laryngeal nerve palsy	Club shaped, in palisades, Chinese characters Polyphosphate granules stain metachromatically	Airborne droplets , colonization of throat and production of Diphtheria tox.	Diphtheria tox: inhibits protein syn by ADP ribosylation of eukaryotic ef-2 . Toxin produced by lysogenized bacteria (like erythrogenic toxin of GABHS)	Tellurite plate, Loeffler's Toxin assessed by animal inoculation or gel diffusion precipitin test.	Antitoxin, Penicillin to reduce transmission	Diphtheria toxoid vaccine. (disease in US is <i>iatrogenic</i> due to inoculation by inadequately killed toxin.

Listeria monocytogenes

(Facultative intracellular anaerobes, Non-sporeforming, tumbling motility)

Gm+ Rods

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Neonatal meningitis and sepsis , abortion, premature delivery	Gm + rods, in clumps, Chinese characters, NON-sporeforming , Tumbling distinguishes it from corynebacterium	Newborns, immunocompromised are high risk groups. Transmitted to humans from animal feces, veggies, unpasteurized milk/cheese .	Only Gm+ with LPS Infects monocytes and induces granulomas. Listeriolysin O punches holes in cells	Gm+ rods, beta hemolysis , motility	Ampicillin	No vaccine

ENTERIC GRAM NEGATIVE RODS

Note: Not all gram negative enterics belong to *Enterobacteriaceae* family: 1) *colonic location* 2) *facultative anaerobes* 3) *ferment glucose*, 4) *oxidase negative*, and 5) *reduce nitrates to nitrites*. ALL are members here EXCEPT: Vibrio, Campylobacter, Helicobacter, Pseudomonas, Bacteriodes. (“Vile People Can’t Be Happy”) As a group, *Enterobacteriaceae* are often **normal flora**. Pathogenesis is by endotoxin/LPS, exotoxins. **O** (Outer polysaccharides), **H** (flagella), **K** (Kapsular polysaccharides) are **important antigens**. Inoculation on **MacConkey’s** or **Eosin-Methylene Blue (EMB)** agar differentiates family members by **lactose fermenting** ability. Fermenters are pink-purple, non-fermenters are colorless. Also keep an eye on **motility**.

ENTERIC		<i>(Intestinal AND non-Intestinal disease)</i>			Gm- Rods	
<i>E. coli</i> (<i>Enterobacteriaceae</i>)						
Diseases	Character	Hab/Trans	Pathogenesis	Diagnosis	Treatment	Prevent
Most common UTI, Gm- sepsis, traveller’s diarrhea . 2 nd most common cause of Neonatal meningitis . Enterotoxigenic strains: Do NOT invade! heat labile enterotoxin binds GM1 ganglioside receptor, activates adenylate cyclase via ADPribosylation of G protein. (like <i>Cholera tox</i>) Watery diarrhea . Enterohemorrhagic: verotoxin inhibits 60s ribosome (like <i>Shigella</i>) Bloody diarrhea . 0157:H7 type causes hemolytic-uremic syndrome (anemia, thrombocytopenia, renal failure) associated w/ fast food outbreaks Enteroinvasive: factor mediated invasion of epithelial cells, sepsis. Bloody diarrhea with WBCs .	As other <i>enterobacteriaceae</i> family	Normal flora, but need virulence factors to cause disease.	Pathogenesis by pilus and enterotoxin, capsule, and endotoxin. Serotype ID by O,H,K antigens	Ferments lactose , unlike <i>Salmonella, Shigella</i>	G3 Cephalosporin	No vaccine
<i>Salmonella</i> (<i>Enterobacteriaceae</i>)						
<i>S. enteritidis</i> causes gastroenteritis via Cholera like tox. Large inoculum needed. (Peptic acid kills) Tx symptoms. <i>S.typhi</i> – Typhoid fever , init by asymptomatic infection of gut phagocytes and dissemination to liver, Gall bladder (carrier state), Fever, RLQ abdominal pain, rose spots . Tx Cipro or ceftriaxone . <i>S. cholerae-suis</i> - Gm- sepsis. Esp patients with Sickle cell (risk for osteomyelitis b/c func. Asplenia)	As other <i>enterobacteriaceae</i> family	Normal flora of <i>animals</i> . Contamination food, poultry / eggs	K antigen/Vi antigen Flagella antigenic variation	Does NOT ferment lactose . Production of H ₂ S gas distinguish from <i>Shigella</i> .	<i>S. typhi</i> - by Cipro or ceftriaxone	Hand washing, cooking, water chlorination

ENTERIC (INTESTIAL disease)					Gm- Rods	
Shigella (Enterobacteriaceae)						
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Enterocolitis (dysentery) by <i>S.dysenteriae</i> , <i>S.sonnei</i> , <i>S.flexneri</i> , <i>S.boydii</i>	Nonmotile Small inoculum <100 bugs	Not normal flora. Humans only host. 4Fs: fingers flied food feces (fecal-oral)	Distal ileal and colonic mucosal invasion and cell death. Does NOT enter bloodstream (unlike <i>Salmonella</i>)	NO H₂S gas, nonmotile. Non lactose fermenting on EMB, MacConkey's agar PMNs in smear w/ fever suggest invasive bug.	Fluid replacement, avoid antiperistaltic drugs which prolong excretion of organism.	
Vibrio (Not Enterobacteriaceae)						
Cholera - Massive watery diarrhea (Rice water stool) like enterotoxigenic <i>E.coli</i> <i>V.parahemolyticus</i> is a marine bug in contaminated raw seafood. Japan	Comma shaped, single flagella. Large inoculum needed.	Infects humans only, transmission by fecal-oral.	Mucinase aided colonization of small intestine, bipartite enterotoxin: binds GM1 gangliosides on enterocyte, ADP-ribosylation of G protein. (like ETEC)	Diagnosis clinically in endemic areas: Asia, Africa, Latin America.	Oral rehydration	No effective vaccine.
Campylobacter (Not Enterobacteriaceae)						
More frequently causes enterocolitis than Salmonella or Shigella. Can cause bloody diarrhea	Comma or S-shaped, Microaerophilic, urease negative	Domestic animals via fecal oral, unpasteurized milk	Probably enterotoxin	Blood agar w/antibiotics, C.jejuni grows at 42C, produces oxidase, nalidixic acid sensitive <i>C.intestinalis</i> grows at 25C, oxidase neg, resistant to nalidixic acid	Antibiotics	No Vaccine
Helicobacter pylori (Not Enterobacteriaceae)						
Gastritis, peptic ulcer, risk factor Gastric carcinoma	Urease + (protects from stomach acid)	Fecal-oral.	Attaches to gastric mucosa, mediated by NH ₃ production, host inflammatory response		Bismuth sulfate, tetracycline, metronidazole	No vaccine.

ENTERIC

(EXTRAIESTIAL disease)

Gm- Rods

Klebsiella-Enterobacter-Serratia (Enterobacteriaceae)

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
<p>Opportunistic pathogens cause UTI, pneumonia, usually nosocomial <i>Klebsiella</i> is nonmotile, with capsule, mucoïd colony appearance. <i>Klebsiella</i> pneumo renowned for severity, bloody “currant jelly” sputum, lung cavitations. <i>Serratia</i>- bright red pigment. Nosocomial- Ab resistant</p>	<p>All ferment Lactose</p> <p><i>K.pneumoniae</i>, <i>E.cloacae</i>, <i>S.marcescens</i> difficult to distinguish clinically</p>	Large intestine, soil water		Ferment lactose on EMB, MacConkey’s agar		No vaccine

Proteus-Providencia-Morganella (Enterobacteriaceae)

<p>Community and nosocomial UTI, b/c high motility</p> <p>(important species: <i>Proteus mirabilis</i>, <i>Proteus vularis</i> <i>Providencia rettgetii</i> <i>M. morganii</i>)</p>	<p>Non lactose fermenting, urease + (alkalinizes urine)</p> <p>Only enterobac that makes phenylalanine deaminase</p>	Large intestine, soil water		<p>Swarming appearance on blood agar. Use antigens from <i>Rickettsiae</i> cross react with <i>Proteus</i>.</p> <p><i>P.mirabilis</i> is indole neg unlike others of this group.</p>		No vaccine
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Pseudomonas (Not Enterobacteriaceae)

<p>P aeruginosa: opportunistic, nosocomial: Pneumonia, osteomyelitis, burn infections, sepsis, UTI, endocarditis, malignant otitis externa, corneal infections. P. cepacia colonizes CF patients</p>	<p>Strict aerobic, Not glucose fermenting, Not reduce nitrates, oxidase +</p>	Normal flora of colon .	Exotox like <i>C.diphtheriae</i> (ADP-reibosylation)	Produces pyocyanin, pyoverdin	Highly resistant. Combo piperacillin, ticarcillin and aminoglycoside. Ceftazidime	
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Bacteroides fragilis (Not Enterobacteriaceae)

<p>Peritoneal abscesses. Growth favored by growth w/ facultative anaerobes to exhause local oxygen</p>	<p>Anaerobic, non sporeforming, non LPS, polysaccharide capsule. No exotox, No LPS</p>	<p>Predominant flora of colon. NOT communicable. Exits colon via break in mucosa (Chronic disease, PID, trauma)</p>	Polysaccharide capsule provides virulence factor		Treat as mixed infection. Clindamycin, or metronidazole	No Vaccine
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RESPIRATORY

Gm- Rods

H. Influenzae (chocolate agar w/ heme and NAD)

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
<p>Leading cause of meningitis in kids. Peak at 6m to 1 yr. (decline in maternal IgG, inability of infants to mount attack vs. polysaccharide capsule)</p> <p>Fatal epiglottitis by type B influenzae.</p>	<p>Coccobacillus w/ polysaccharide Capsule</p>	<p>Upper respiratory tract, respiratory droplets</p>	<p>ONLY encapsulated forms like type B cause invasive disease. Nonencapsulated cause URI, pneumonia in pts with preexisting lung disease (COPD). IgA protease,</p>	<p>Chocolate agar, w/ heme and NAD.</p> <p>Quellung rxn</p> <p>“Hmmm Chocolaate!”</p>  <p>H.Simpson</p>	<p>Rifampin prevents meningitis and transmission from close contacts b/c secreted into saliva better than Ampicillin</p>	<p>HIB vaccine of capsular polysaccharide conjugated to carrier protein.</p>

Legionella pneumophila (Cysteine and Iron agar)

<p>Atypical pneumonia with high fever, nonproductive cough(differentiate from <i>Mycoplasma</i>, influenza, psittacosis, Q fever)</p>	<p>Poor gm stain</p>	<p>Airborne from water sources. Smoking EtOH, Immunosuppressed are at risk.</p>		<p>High concentration</p> <p>Error! No table of figures entries found.of cysteine and iron. Urine antigen test. Suspect when inc. PMNs with no organisms!</p>	<p>Erythromycin (also good for <i>Mycoplasma</i>)</p>	<p>Disinfect water sources</p>
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Bordetella pertussis (Bordet-genou agar)

<p>Whooping cough- acute tracheobronchitis with URI symptoms, paroxysmal hacking cough 1-4 wks, copious mucus</p>	<p>Small gm- rods</p>	<p>Airborn droplets (highly contagious)</p>	<p>Polysaccharide capsule and pili are essential for virulence. Does NOT invade. Pertussis tox (ADP-ribosylation), and tracheal cytotoxin.</p>	<p>Culture on Bordet-genou agar. Ab agglutination, stain</p>	<p>Erythromycin reduces complications, doesn't change clinical course. Resp tract already damaged.</p>	<p>Killed B.pertussis vaccine 2,4,6 months, Boosters at age 1, school. Acellular vax for booster only.</p>
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ZOONTIC

Gm- Rods

<i>Brucella (virulent, facultative intracellular, tx: aminoglycoside)</i>						
Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Brucellosis - influenza like syndrome w/ undulating fever (higher during day, lower at night). Lymphadenopathy, H/Smegaly, no boboes/ulcers.	Small gm- rods	Animal reservoirs: B <i>melitensis</i> (goats/sheep) B. <i>abortus</i> (cattle) B. <i>suis</i> (pigs) Non pasteurized milk products (travelers), through skin (meat packers, vets, farmers)	Organisms localize in RES . Persist in macrophages, induce granulomas	Serology, biochemistry	Antibiotics	Animal vaccination, pasteurization. No Human vaccine.
<i>Francisella tularensis (virulent, facultative intracellular, tx: aminoglycoside)</i>						
Tularemia - influenza like syndrome w/ ulceroglandular lesions (hole in skin, black base, swollen LN, draining pus)	Small gm- rods	Ubiquitous in US in wide variety of animals. Tick/mite vectors. Humans as accidental dead end hosts by bites or animal skin handling.	Enters through skin, localizes in RES . Persist in macrophages, induce granulomas	Serology	Streptomycin	Live attenuated vaccine (like BCG)
<i>Yersinia pestis (virulent, facultative intracellular, tx: aminoglycoside)</i>						
Plague - Hematogenous spread results in fever myalgias, hemorrhage. Also septic shock, pneumonia.	Small gm- rods with bipolar stain	Endemic in prairie dogs in US, 99% cases in SE Asia. Rats/fleas in urban centers. Also wound-person respiratory droplets.	Bacteria spread to regional LN, enlarged tender buboes.	Immunofluorescence	Antibiotics	Quarantine. No Vaccine.
<i>Pasteurella multocida</i>						
Cellulitis rapid onset at bite site. Osteomyelitis as complication. Sutures predispose to infection	Small gm- rods	Normal flora of dogs and cats. Transmitted to humans by animal bite.		Presumptive Dx by rapid onset cellulitis at animal bite.	Penicillin	Ampicillin prophylax.

MYCOBACTERIA

(Obligate aerobe, facultative intracellular organisms)

Acid Fast Rods

M. tuberculosis

Diseases	Character	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Tuberculosis: chronic low grade fever, night sweats, productive cough, hemoptysis, weight loss. Elderly, immunocomp, malnourished at risk.	Obligate aerobe, intracellular, infect M0, persist for years. Mycolic acid walls	Only infects humans. Respiratory aerosol. Infects M0s in mid/lower lobes , init granuloma formation, widely disseminate the infection. T cells help M0 kill some intracellular mycobacteria at expense of bystander cell damage. Result: necrotic host cells, viable mycobacteria. Walled off w/ giant cells, fibroblasts, collagen, calcification to form granuloma or tubercle . This 1^o infection = Ghon focus on CXR (when including Ca tubercles in perihilar lymph nodes = Ghon complex .) Reactivation prefers upper lobe (obligate aerobe) of lung. Reactivation can infect any organ. Cervical LN (scrofula), spine (Pott's disease .)	Mycolic acids confer acid fastness. WaxD is active ingredient in Freund's adjuvant . Cord factor is virulence factor (mycoside= 2 mycolic acids + disaccharide)	PPD tests for prior exposure or to BCG vax . Positive test if both redness, induration 48-72 hr after injection (DTH rxn.) Note: <i>candida</i> and mumps as controls in immunocomp. Acid fast stain. NaOH concentrate on Lowenstein-Jensen medium . Slow culture, 6-8 wks. Niacin production	Prolonged, multiple Tx. (INH, rifampin, pyrazinamide, ethambutol) Protracted tx b/c: intracellular life cycle, granuloma blocks penetration of drug, metabolically inactive mycobac persist in lesion	Chemoprophylax w/ INH (watch hepatotox in people >35 y.o.) Live attenuated M.bovis (BCG) induces some protective immunity.

M. avium-intracellulare

Clinical TB indistinguishable from <i>M tuberculosis</i> in AIDS.	Atypical mycobacterium	Found in water, soil, not pathogenic in guinea pigs (infects birds)			Azithromycin Clarithromycin	Macrolide prophylax when CD4 count < 50
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M. leprae

Leprosy- preferential growth in < 37C, skin, superficial nerves. Tuberculoid- good cellular immune response, few AFB, granulomas, positive lepromin skin test. Anesthetized skin lesions and thickened superficial nerves. Lepromatous- poor cellular immune response, lots of organisms, foamy histiocytes, negative lepromin skin test (poor response.) Skin lesions, lion facies. Skin anesthesia, bone resorption, skin thickening, disfiguring.	Never has been grown in lab.	Brazil, India, Sudan Humans only natural hosts. Mouse footpad and armadillo growth only. Transmission by nasal secretions, skin lesions to persons with prolonged contact w/pts.	Intracellular replication (skin histiocytes, endothelial cells, Schwann cells)		Rifampin Dapsone Up to 2 years!	Prophylax exposed persons with Dapsone.
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ACTINOMYCETES

Gm- Branching Rods

A. israelii

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
Actinomycosis - hard non-tender swelling, drains pus through sinus. (abscess that spreads to neck, chest, abdomen.)	Aanerobic Gm-branching rods	Normal anaerobic flora oral cavity/GI tract. Not communicable.	Invasion after local trauma (risk factor for anaerobic growth)	Anaerobic Gm-branching rods, sulfur granules in pus	Penicillin	No vaccine

Nocardia asteroides (Acid Fast Branching)

Nocardiosis - pneumonia that progresses to abscess formation, sinus tract drainage, dissemination to brain/kidney (immunosuppressed)	Aerobic Gm-branching rods.	Soil, NOT normal flora		Acid fast branching, NO sulfur, aerobic	Bactrim (trimethoprim + sulfamethoxazole)	No vaccine
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Mycoplasma pneumoniae

(No cell wall, poor gm stain)

Small free living organism

Diseases	Characteristics	Habitat/Transmission	Pathogenesis	Diagnosis	Treatment	Prevention
“Walking pneumonia” (dry nonproductive cough, horrible CST, generally feel well) Most common pneumonia in young adults (college students).	Smallest free living organism, no cell wall so poor gm stain, resists penicillins, cephalosporins. Cell membrane has chol which are not in other bacteria. “Fried egg” colonies on Eaton’s agar. (“Eat Fried Eggs w/ chol”)	Respiratory droplets. Attaches but does NOT invade respiratory epithelium, like <i>B.pertussis</i> .	Pathogenic only for humans. Arrests cilliary motion, induces epithelial cell necrosis. Cross reactive antigens induce anti RBC autoantibodies (cold agglutinins.)	Elevated titer of cold agglutinins or specific anitbodies	Erythromycin Tetracycline	No vaccine