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Independent Study Projects

Title

Microbiology Concept Maps

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Author

Huang, Grace

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Microbiology Concept Maps

Grace Huang (MS3)

Dr. Cannella did an amazing job of synthesizing what we covered in the microbiology lectures into systematic charts, emphasizing the high-yield facts. Sadly, he left UCSD after my year, but, with the help of the wonderful Dr. Sharon Reed, Dr. Sanjay Mehta, and Dr. David Pride, I have compiled microbiology charts based on Dr. Cannella's charts and the information covered in lecture. The maps are designed with MindMaple software and are fully editable with the free software download. The lectures cover a great deal of information, so I hope these charts will help you better sort each microbe in a systematic way.

ISP Chair: Dr. Sharon Reed

ISP Committee Members: Dr. Sanjay Mehta & Dr. David Pride

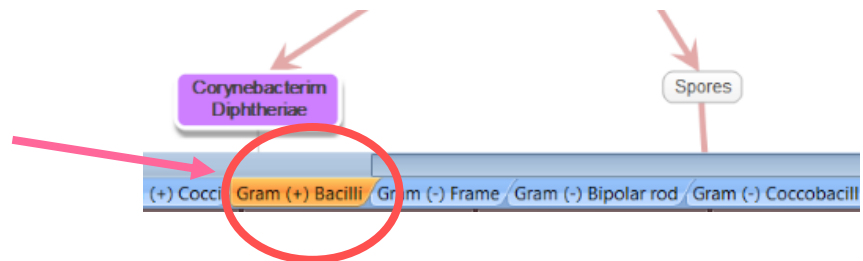
1. Download + install **MindMaple Lite** (free software):

<http://www.mindmaple.com/Downloads/Windows/>



2. Open **Micro Concept Maps** file.

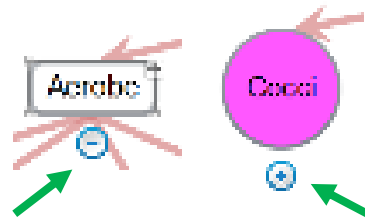
3. Toggle between the different maps using the tabs at the bottom of the screen.

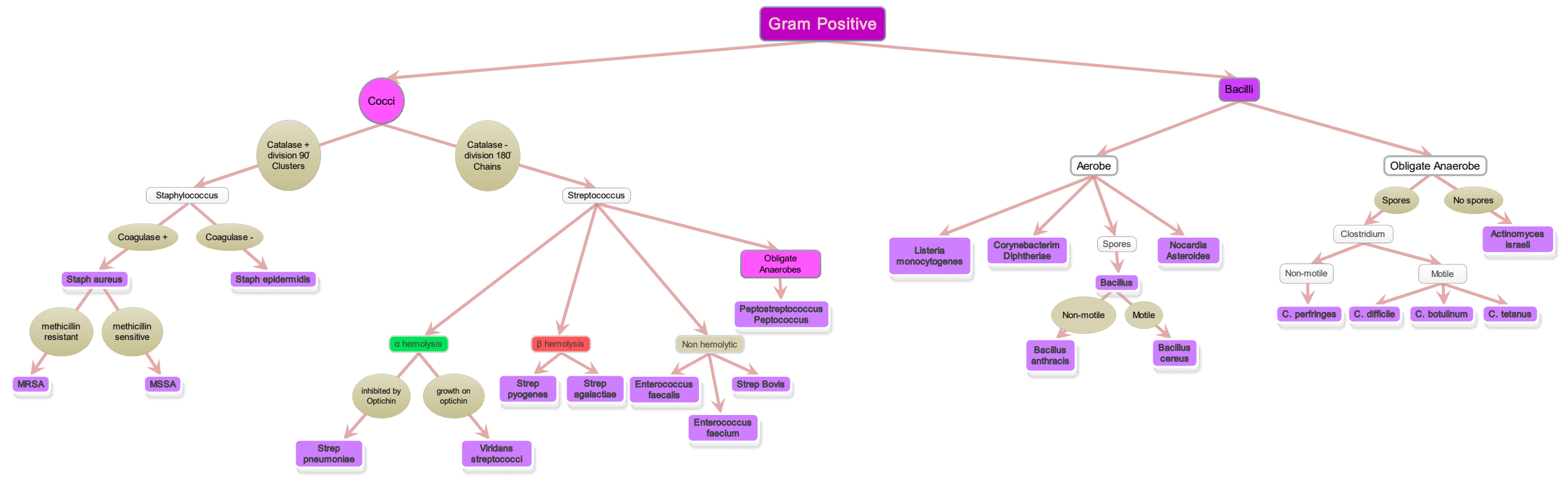


4. Two categories of maps:

- a. Frame: outline of complete map
- b. Comprehensive maps: usually focused on a subset of a particular group

5. Branches can be hidden by clicking on (-) or revealed with (+).





Gram Positive

Cocci

Bacilli

Catalase + division 90° Clusters

Catalase - division 180° Chains

Staphylococcus

Streptococcus

Coagulase +

Coagulase -

Staph aureus

Staph epidermidis

α hemolysis

β hemolysis

Non-hemolytic

Obligate Anaerobes

General:

- commonly colonizes the nose
- causes a broad range of diseases:
 - exotoxin mediated disease
 - direct organ invasion: skin infections, pneumonia, endocarditis, septic arthritis, osteomyelitis

General:

- normal skin flora
- can migrate in through Foley catheters or IV lines
- Novobiocin sensitive

inhibited by Optochin

growth on Optochin

Strep pyogenes

Strep agalactiae

Enterococcus faecalis & faecium

Strep Bovis

Peptostreptococcus Peptococcus

Pathophysiology:

Many virulence factors:

- Penicillinase: breaks down penicillin > penicillin-resistance
- Protein A - binds to Fc portion of IgG, preventing opsonization and phagocytosis
- Coagulase - activates prothrombin > fibrin clot around bacteria protects it from phagocytosis
- Hyaluronidase & proteases: enables it to tunnel through tissue

Exotoxins:

- Exfoliatin Toxin A & B: diffusible toxin that cleaves the middle epidermis > scalded skin syndrome
- Toxic Shock Syndrome Toxin (TSST-1): superantigen that binds to MHC II and T cell receptors > TNF and IL-1 stimulation > massive immune response
- Enterotoxins: preformed, heat-stable toxin in contaminated food that stimulates peristalsis of intestine > gastroenteritis

Pathophysiology:

- polysaccharide capsule allows adherence to many surfaces
- produces biofilms > able to bind to prosthetic devices + protect from attack

Presentation:

- bacteremia
- sepsis

Diagnosis:

- Gram stain: gram (+) cocci in clusters
- coagulase (-)

Treatment:

- Vancomycin (if significant infection)

General:

- lancet-shaped diplococci
- encapsulated
- IgA protease

• major cause of bacterial pneumonia, meningitis, otitis media, and sinusitis in adults

General:

- part of normal GI tract flora
- often live in nasopharynx and gingival crevices

Pathophysiology:

- Bind to teeth leading to dental infections

Subacute Bacterial Endocarditis > dental manipulation sends showers of organism into bloodstream > implant on previously damaged heart valve (rheumatic fever) > produce a dextran that allows them to cling to valve > subacute bacterial endocarditis

Presentation:

- Subacute bacterial endocarditis: low grade fevers, heart murmurs, anemia, fatigue

Presentation:

- Meningitis - nuchal rigidity, fevers, nausea
- Pneumococcal pneumonia - sudden onset of shaking chills, high fevers, chest pain, SOB. Consolidation made of WBCs, bacteria, and exudate. Cough up yellow-green phlegm
- Otitis media - middle ear infection in children

Diagnosis:

- Optochin (P disc) sensitivity helps differentiate S. pneumo from Viridans strep

Tx/Prevention:

- Pneumococcal vaccine
- Penicillins (depends on resistance pattern and type of infection)

Pathophysiology:

Many virulence factors:

- M protein - inhibits complement activation and prevents phagocytosis, but antibodies form against M protein which can then cause **rheumatic fever**
- Streptolysin O - destroys RBC and WBC, ASO antibodies develop against antigen
- Pyrogenic exotoxin - found in a few strains of group A strep, cause scarlet fever and strep toxic shock syndrome

Delayed antibody-mediated response:

- antibodies that form against M protein during pharyngitis infection cross react with antigens on the heart > damage the heart, especially the mitral valve
- antibodies formed against pharynx or skin infection > strep antigen planted on glomerular basement membrane > antibodies bind to GBM > activation of complement > acute post-streptococcal glomerulonephritis

Presentation:

Diseases caused by local invasion/exotoxin

Streptococcal pharyngitis (Strep throat):

- red swollen tonsils and pharynx
- fever, swollen lymph nodes

Streptococcal skin infection

- erysipelas - infection of upper dermis
- cellulitis - infection of dermis and subQ fat
- impetigo - vesicular, blistered eruption around mouth
- Necrotizing fasciitis - spreads between subcutaneous tissue and muscle > bullae, skin death, and myositis

Scarlet fever

- pyrogenic toxin produces fever and scarlet-red rash
- rash starts axially and spreads to extremities, sparing the face

Streptococcal toxic shock syndrome

- similar to toxic shock caused by Staph aureus

Delayed antibody mediated diseases

Rheumatic fever

- Occurs after untreated streptococcal pharyngitis
- Fever, myocarditis, migratory arthritis, Sydenham's chorea, subcutaneous nodules, erythema marginatum
- can lead to long-term damage to heart valves + murmurs

Acute post-streptococcal glomerulonephritis

- coca-cola urine (hematuria)
- periorbital edema (fluid retention)
- high blood pressure

Diagnosis:

- Throat swab for Rapid Strep and/or culture
- Serum ASO titers for chronic infection

Tx/Prevention:

- Penicillin G

General:

- Group A Strep
- pus-producing

Pathophysiology:

- babies infected during delivery > meningitis

Presentation:

- Non-specific signs in neonates
- fever, vomiting, poor feeding, irritability

Diagnosis:

- Lumbar puncture

Tx/Prevention:

- Screen pregnant women at 35-37 weeks and treat GBS+ women with penicillin

General:

- normal gut flora
- grow well in bile and 6.5% NaCl
- common nosocomial infection
- lots of drug resistance (vancomycin)

Pathophysiology:

- In hospitalized patients, commonly cause UTI, biliary tract infections, subacute endocarditis (post surgery)

Vancomycin Resistant Enterococcus (VRE):

- Chromosomal transposon vanA that changes peptidoglycan cell wall from D-ala-D-ala to D-ala-D-lactate > low affinity for vancomycin > very difficult to treat

Presentation:

- Depends on type of infection

Diagnosis:

- Culture

Tx/Prevention:

- Ampicillin or Vancomycin if sensitive
- Daptomycin/linezolid for VRE

General:

- normal gut flora
- grow well in bile
- can cause subacute endocarditis
- associated with colon cancer

General:

- part of normal flora in mouth, vagina, and intestine
- mixed with other anaerobes in abscesses
- seen in aspiration pneumonia

Tx:

methicillin resistant

methicillin sensitive

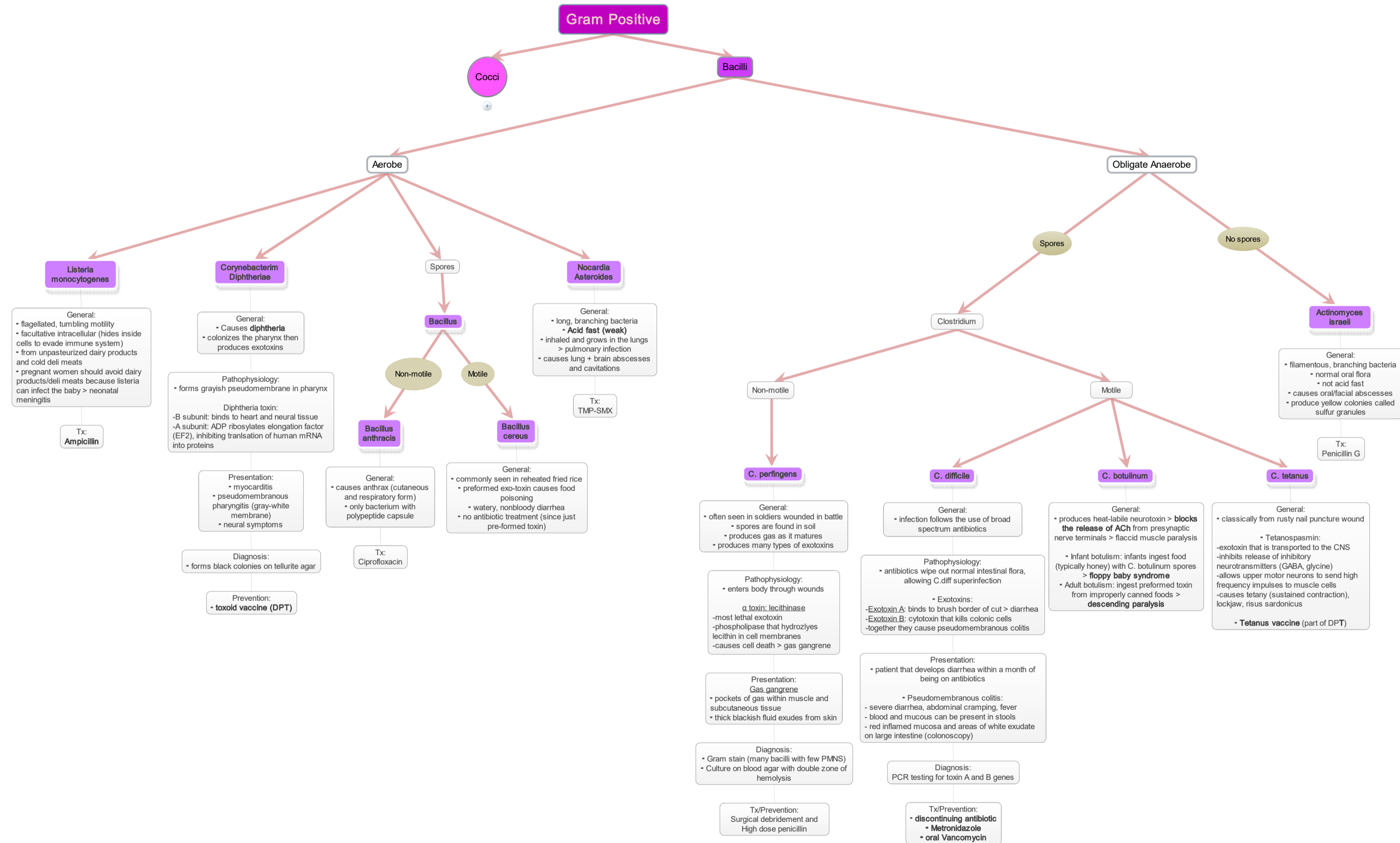
MRSA

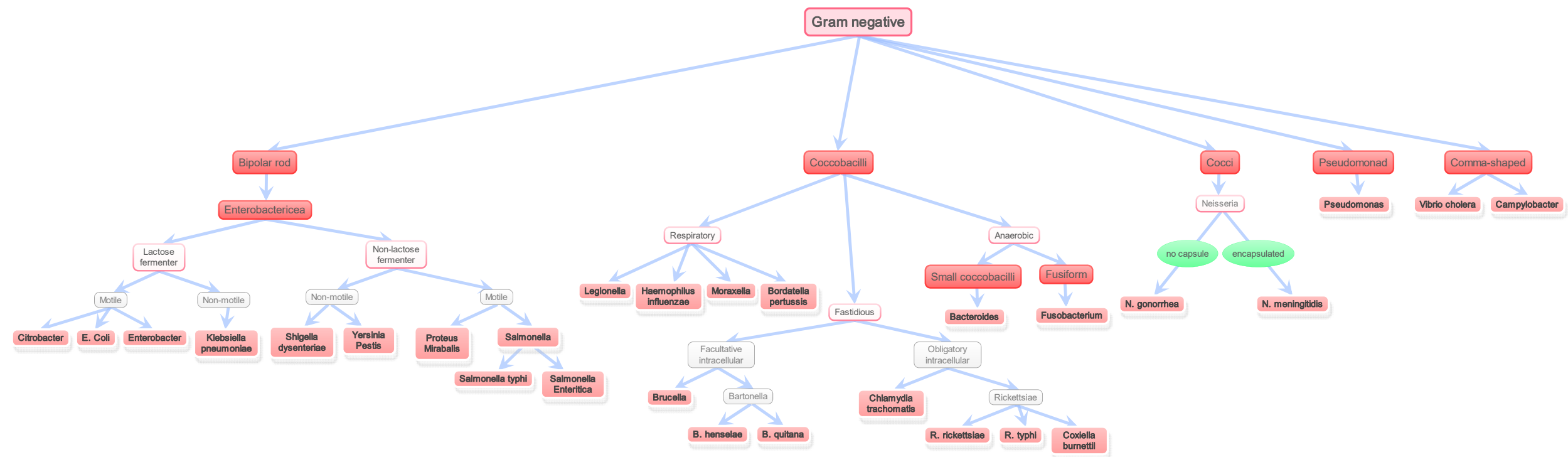
MSSA

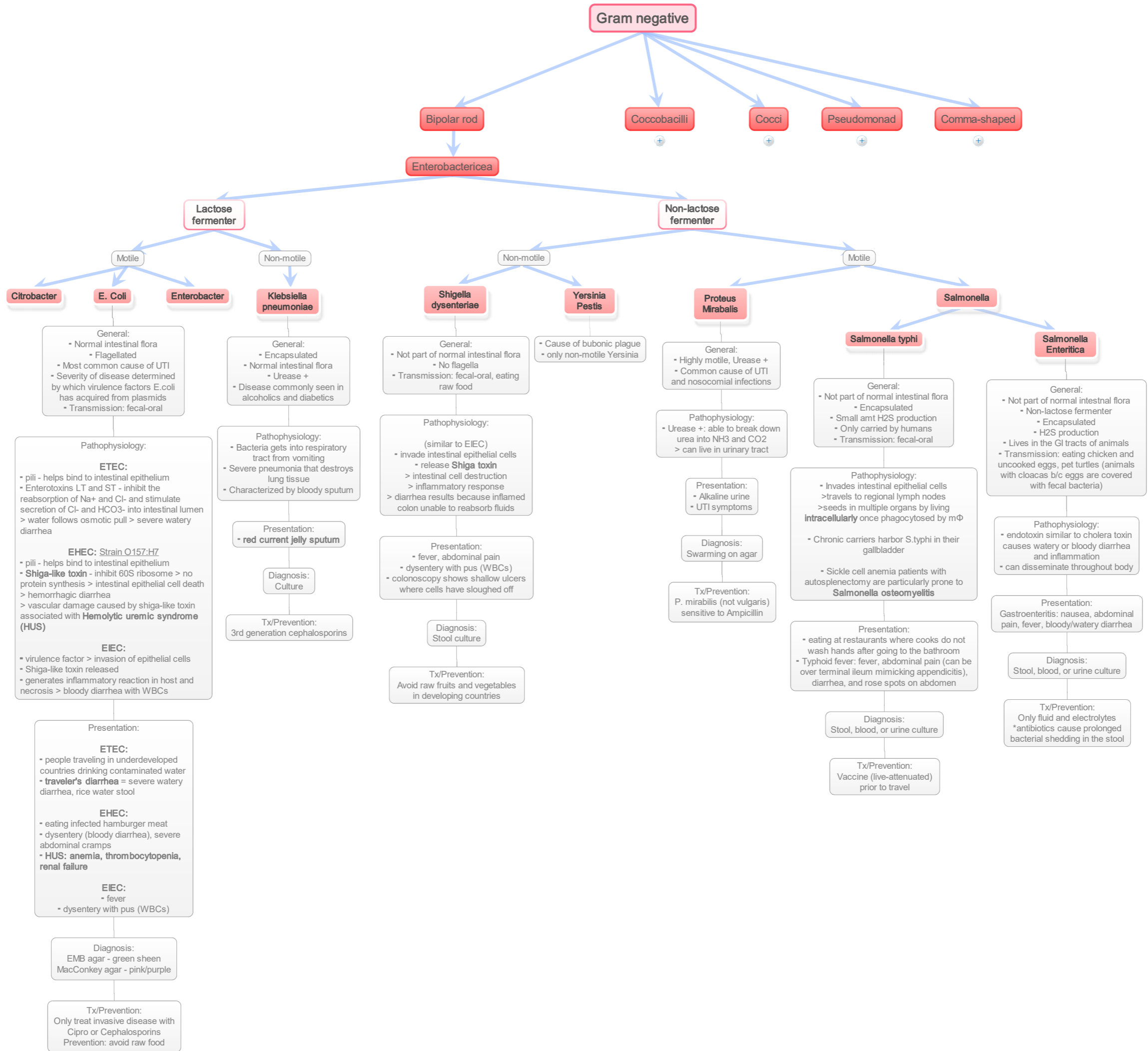
Resistant to methicillin and nafcillin

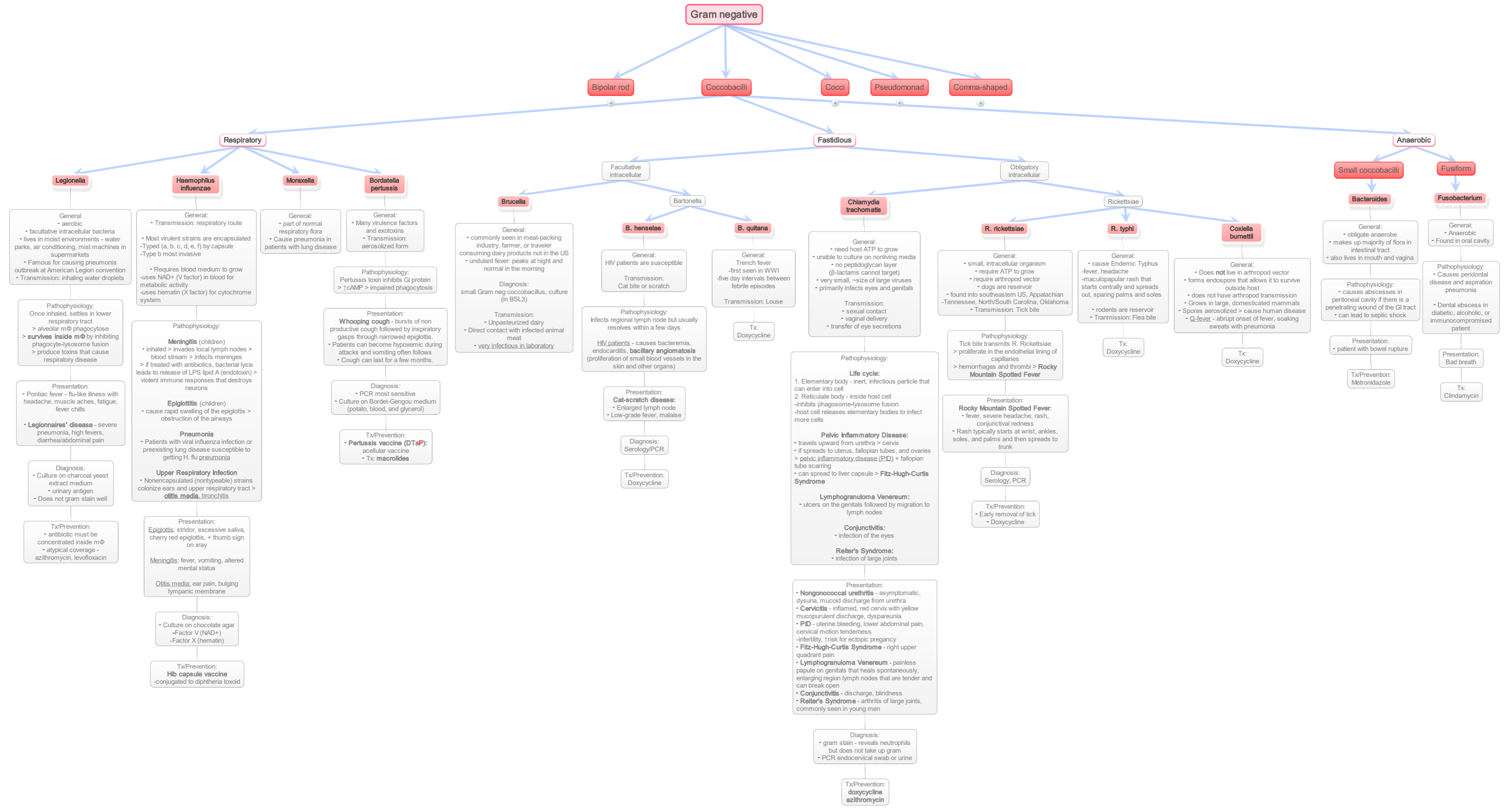
- acquired by mecA DNA segment which encodes penicillin binding protein, conferring resistance against penicillinase-resistant penicillins and cephalosporins
- treat with Vancomycin

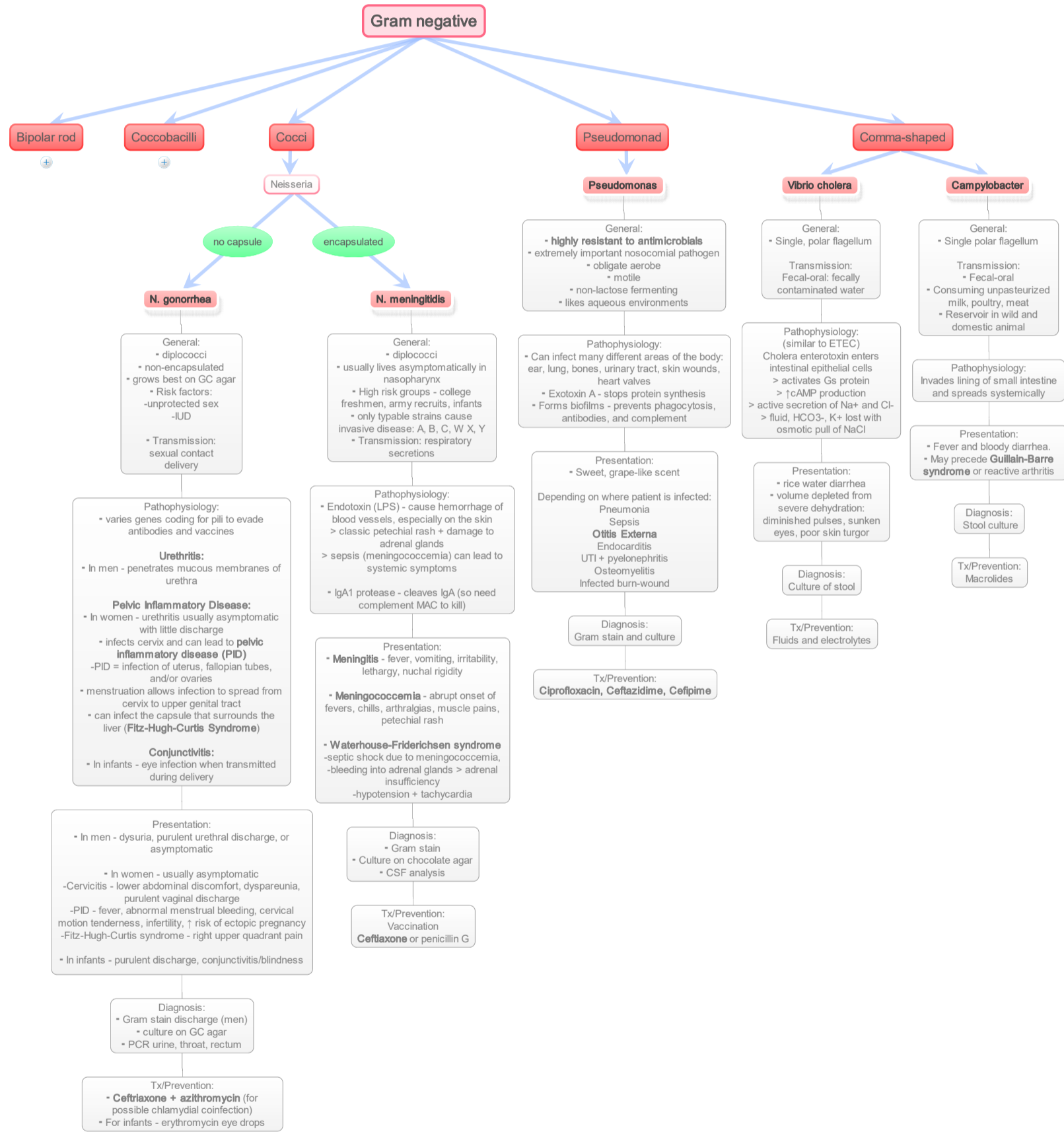
Cephazolin or Oxacillin

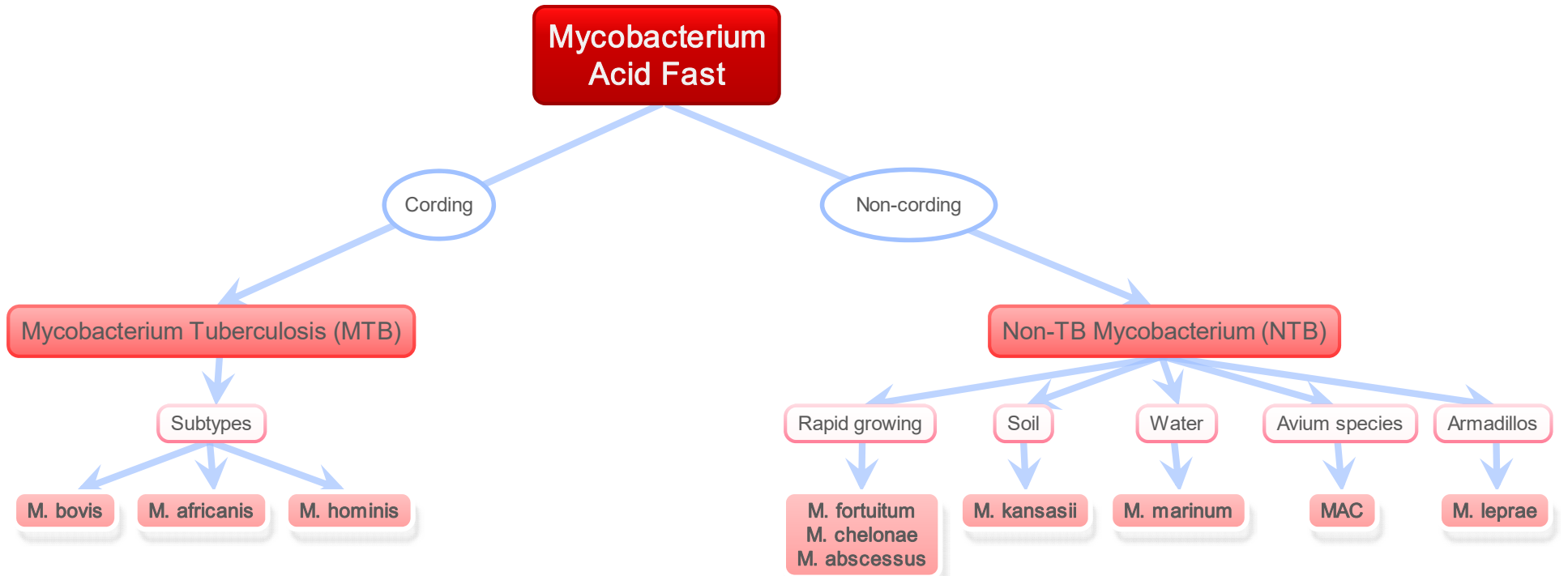












Mycobacterium Acid Fast

Cording

Non-cording

Mycobacterium Tuberculosis (MTB)

Non-TB Mycobacterium (NTB)

General:

- high lipid content in cell wall > acid fast, difficult for antibiotics to penetrate
- mycolic acid in cell wall**
 - obligate aerobe
 - facultative intracellular
- need T cell immunity to fight pathogen
- Transmission: inhalation

M. bovis

- Ingestion of unpasteurized milk causing infection of GI tract
- Infects wild animals: bison, elk, moose
- pyrazinamide resistant

M. africanis

- from Africa
- worse resistance patterns

M. hominis

- Ubiquitous, but cases are rare
- worse resistance patterns

Rapid growing

M. fortuitum
M. chelonae
M. abscessus

Outbreak from pedicures, tattoos

Soil

M. kansasii

Similar presentation to MTB, often are confused with one another

Water

M. marinum

Exposure to contaminated sea water
-aquarium owner
-angler

Avium species

MAC

General:
- common **opportunistic infection in AIDS patient** (CD4 < 100)

Presentation:
fever, weight loss, diarrhea, malaise, ↑ALP

Diagnosis:
Blood culture

Tx/Prevention:
Azithromycin for prophylaxis

Armadillos

M. leprae

General:
Causes leprosy

Pathophysiology:

Mycoside virulence factors:

- Cord factor - inhibits mΦ maturation
- sulfatides - inhibit phagosome-lysosome fusion

Acute phase:

- Inhaled into lungs and causes local inflammation, usually in **lower lobes**
- Phagocytosed and multiply in mΦ
- Can either cause systemic disease (**Primary TB**) or gets walled off in **caseous granuloma** and remain dormant in mΦ
- Primary TB can spread to lungs, kidney, bones, CNS, liver, etc.

Chronic phase (secondary TB):

- If MTB dormant, can reactivate later in host
- Lungs - infection reoccurs in **upper lobes** (highest O₂) > caseation and cavitation

Presentation:

- Fever, night sweats, weight loss, hemoptysis, swollen cervical lymph nodes
- Pott's disease** - destruction of intervertebral discs + vertebral bodies
- CNS involvement** - meningitis, granulomas in brain
- Miliary TB** - tiny millet-seed granulomas disseminated all over the body

Diagnoses:

- PPD skin test** or **gamma-interferon release assay** (blood test) - indicates exposure
 - Chest x-ray
 - Sputum acid fast stain
- Culture - faster growth (7 days) than solid media; PCR most sensitive

Tx/Prevention:

- BCG vaccine** (not used in US) - only prevents TB meningitis
- For active infection (RIPE):
 - Rifampin
 - Isoniazid
 - Pyrazinamide
 - Ethambutol

Miscellaneous bacterium

Mycoplasma pneumoniae

Spirochetes

Treponema pallidum

Leptospira

Borrelia burgdorferi

Miscellaneous bacterium

Mycoplasma pneumoniae

General:
- tiniest free living organism
- lack peptidoglycan wall (cannot use β -lactams)
- cell membrane contains cholesterol
- Transmission: inhalation

Pathophysiology:
After inhalation > attaches to respiratory epithelial cells
> 2-3 week incubation period > walking pneumonia

- patients can develop **cold agglutinins** (monoclonal IgM that bind to RBC causing them to agglutinate at 4°C)

Presentation:
Walking pneumonia: fever, sore throat, malaise, persistent dry hacking cough

Diagnosis:
Cold agglutinin test - cool sample of patient's blood and check for agglutination

Tx/Prevention:
Doxycycline

Spirochetes

- gram negative
- corkscrew movements
- axial flagella

Treponema pallidum

General:
- causes syphilis
- cannot be grown in laboratory
- Transmission: skin contact

Pathophysiology:
Penetrates intact mucous membranes by burrowing through tissue
> kills nerves so painless lesion
> can then move systemically
> infection occurs in 3 phases

1° syphilis: initial infection

2° syphilis: systemic spread

3° syphilis: slow inflammatory damage to multiple organs
-damage vasa vasorum (arteries supplying the heart)
-damage posterior columns and dorsal roots of spinal cord
-damage nerve cells of the brain > psychiatric symptoms
-rapid progression to 3° syphilis in 6 months with HIV

Presentation:

1° syphilis:
painless chancre at site of contact, highly infective
Heals after 4-6 weeks

2° syphilis:
Generalized lymphadenopathy, fever, weight loss
maculopapular rash - widespread involving palms and soles
condyloma latum - painless, wartlike lesion on genitals

3° syphilis:
Gummas- granulomas in skin and bones
Aortic aneurysm - due to damage of vasa vasorum
Tabes dorsalis: loss of all sensation (proprioception, vibratory, temp, pain) and reflexes
General paresis: mental deterioration and psychiatric symptoms
Argyll-Robertson pupil: midbrain lesion > pupils do not constrict to light but constricts for accommodation

Diagnosis:

- **RPR/VDRL test:**
-testing for antibodies against lipids that are released into the serum due to cellular damage
-non-specific tests that can be falsely positive
-titer useful to follow response to therapy

- **EIA, TPPA (treponema pallidum particle agglutination) test:**
-tests for antibodies against Treponema
-more specific test

Tx/Prevention:
Penicillin G

Leptospira

General:
- found in the urine of animals
- Transmission: fresh water contaminated with animal urine coming in contact with any mucosal membrane
- Weil's Disease: severe illness involving renal failure, hepatitis, meningoencephalitis, septic shock

Tx:
Doxycycline

Borrelia burgdorferi

General:
- causes Lyme disease
- seen in Northeast
- Reservoir: deer, small rodents
- Arthropod vector: Ixodes tick

Pathophysiology:
Infection occurs in stages
- **Early localized stage:** lesion at site of bite
- **Early disseminated stage:** travels to heart, skin, nervous system, and joints
- **Late stage:** chronic arthritis

Presentation:

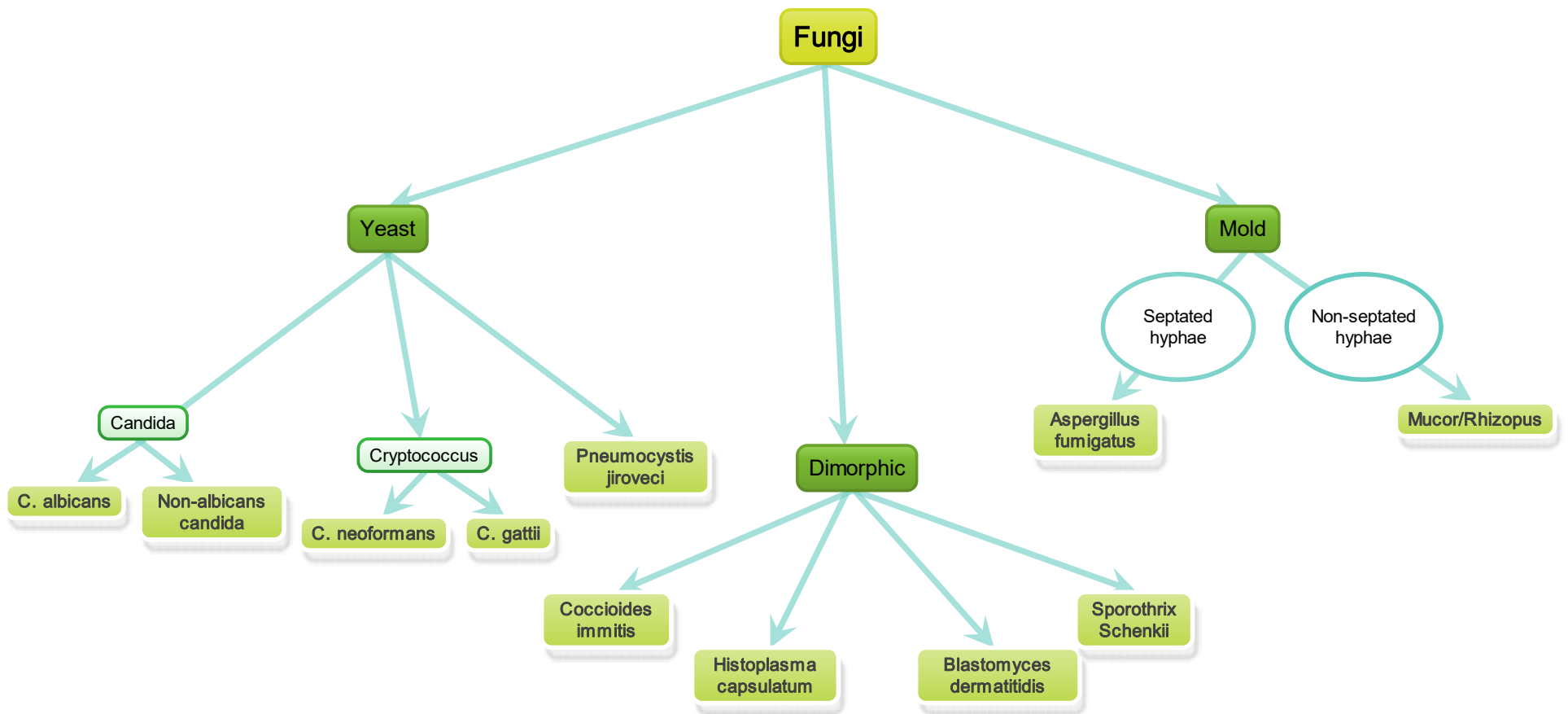
Early localized stage:
Erythema chronicum migrans - red, target-like lesion at site of tick bite

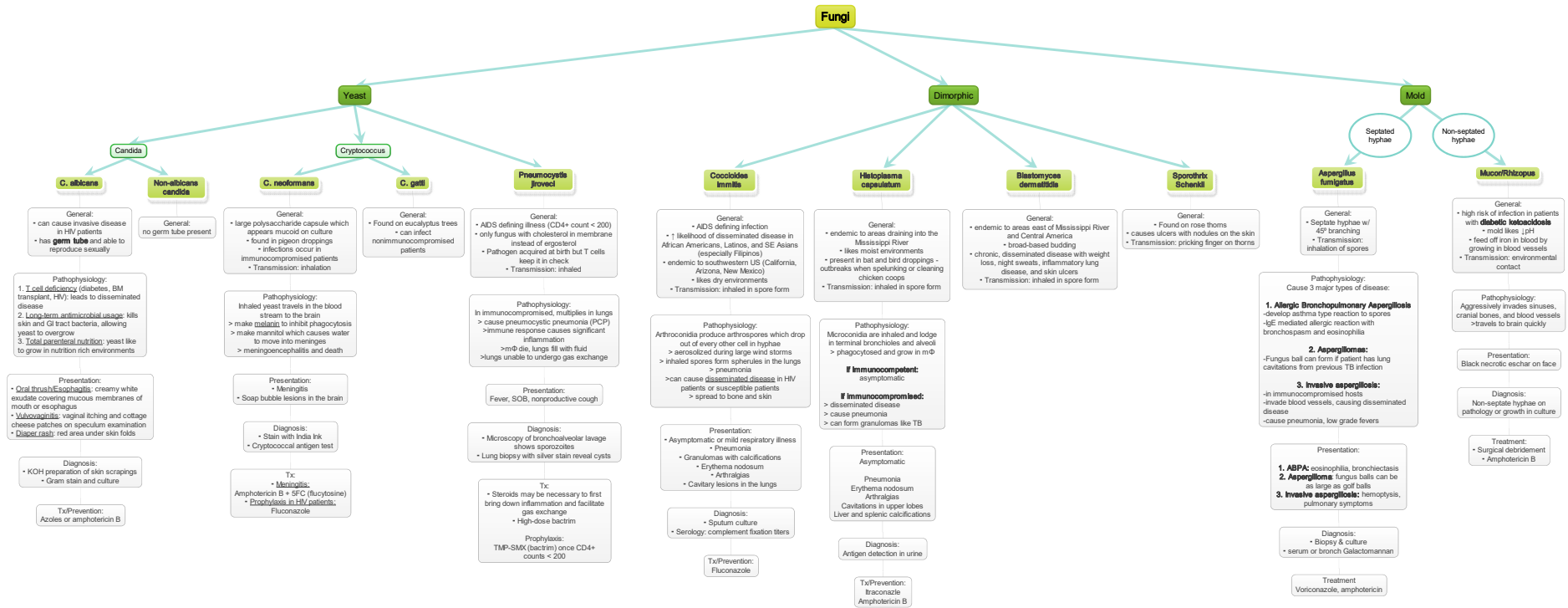
Early disseminated stage:
- CNS: facial palsy (bilateral), polyneuropathies
- Cardiac: AV heart block
- Migratory arthritis

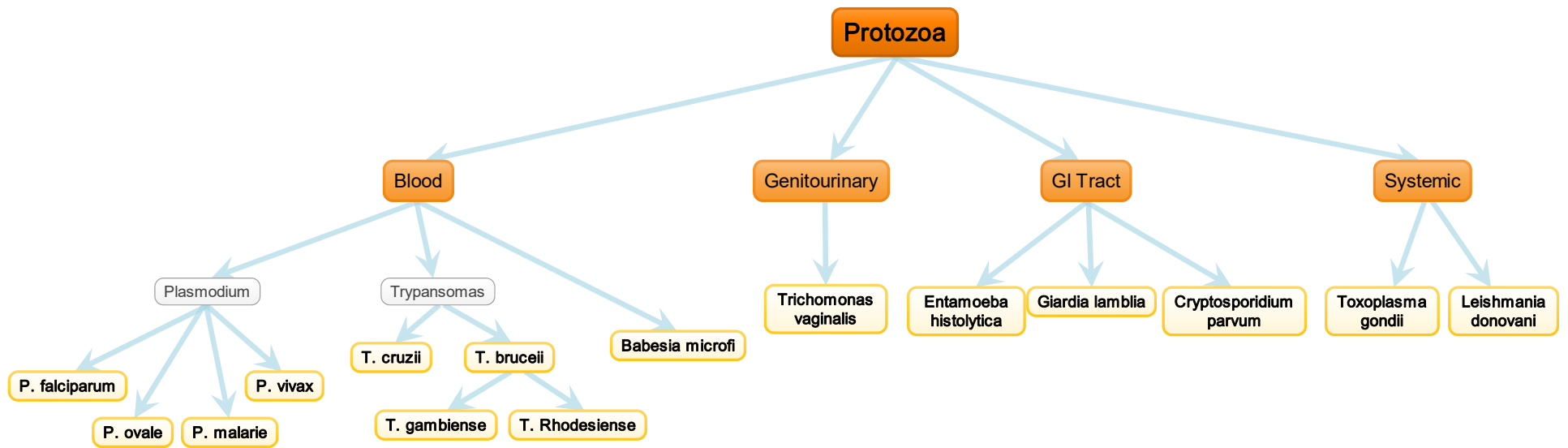
Late stage:
Chronic arthritis of large joints

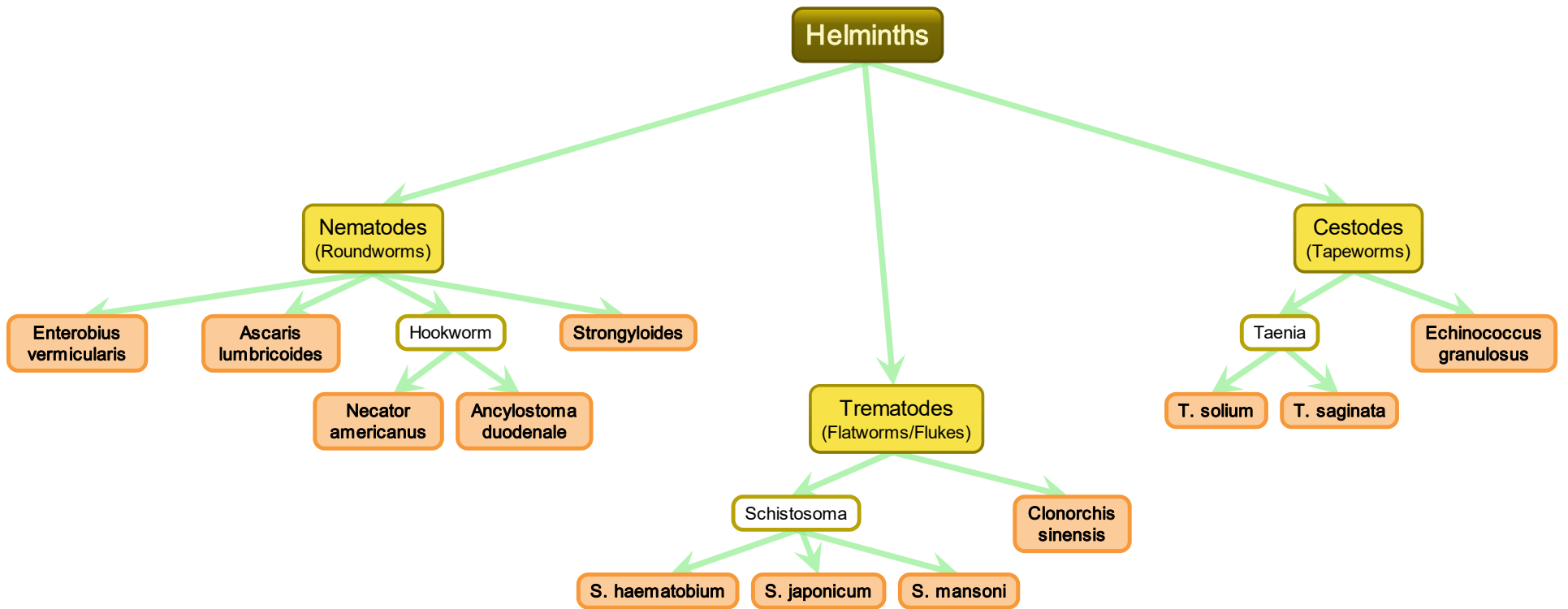
Diagnosis:
- Culture difficult
- Rely on clinical signs and serology

Tx/Prevention:
- Doxycycline
- Ceftriaxone for disseminated infection









Helminths

Nematodes (Roundworms)

Enterobius vermicularis

General:
• also known as pinworm
• Transmission: fecal-oral

Pathophysiology:
• Eggs are ingested
> mature in the large intestine
> at night, females migrates to perianal area to lay eggs
> hatch a few hours later
Intense itching
• scratching allows for host to reinfest themselves or others

Presentation:
• perianal itching, especially at night
• no eosinophilia because no tissue invasion

Diagnosis:
• scotch tape test: tape placed in perianal region at night

Treatment:
• **Mebendazole**

Ascaris lumbricoides

General:
• affects over 1 billion people
• can grow up to 1 meter long
• Transmission: contaminated food
• Can cause invasive disease b/c of developmental cycle

Pathophysiology:
• Eggs are ingested
> larvae emerge in the small intestine
> burrow through intestinal wall and travel in the blood stream to the lungs
> grow in alveoli until coughed up and swallowed
> reach small intestine and grow by consuming food in the GI tract
> adults lay eggs which are then excreted in the feces
• High worm load, can lead to swelling
• twisting of intestine

Presentation:
• **Asymptomatic**
> Pulmonary infiltrates
> High eosinophil count

• **High worm load:**
> Intussusception or volvulus of intestine
> Necrosis of bowel

Diagnosis:
Identify eggs in feces

Tx/Prevention:
• Treat acute infection with mebendazole or limit worm burden in endemic areas with yearly therapy

Necator americanus

General:
• Transmission: contact with soil

Pathophysiology:
• Larvae lives in soil
> transforms and able to penetrate human skin
> travels to alveoli and grow
> coughed up and swallowed
> move to small intestine
> attach to wall with pincers and suck blood
> sexually reproduce - release fertilized eggs in feces

Presentation:
• diarrhea, abdominal pain, weight loss
• Iron deficiency anemia
• Eosinophilia

Diagnosis:
Identify eggs in fecal sample

Tx/Prevention:
Benzodiazoles
Wear shoes at the beach in developing countries!

Ancylostoma duodenale

General:
• similar to Necator americanus but with more blood loss

Pathophysiology:
• Larvae grows in soil
> penetrate human skin
> travels to the lungs
> coughed up and swallowed
> travels to the small intestine + laye eggs
> hatched larvae can penetrate intestine and travel to the lungs to continue cycle

• Immunosuppressive agents can cause disseminated disease in infected individuals

• When penetrating gut wall, can bring gut bacteria along with it
> see gram (-) infections in the CNS

Presentation:
• Vomiting, abdominal bloating, diarrhea, anemia
• Pruritic rash, lung symptoms
• Eosinophilia

Diagnosis:
Identify larvae in feces
Eosinophilia

Treatment:
• **Ivermectin**

Strongyloides

General:
• potentially lethal disease in immunocompromised
• seen in any tropical area

Pathophysiology:
• Larvae grows in soil
> penetrate human skin
> travels to the lungs
> coughed up and swallowed
> travels to the small intestine + laye eggs
> hatched larvae can penetrate intestine and travel to the lungs to continue cycle

Presentation:
• Vomiting, abdominal bloating, diarrhea, anemia
• Pruritic rash, lung symptoms
• Eosinophilia

Diagnosis:
Identify larvae in feces
Eosinophilia

Treatment:
• **Ivermectin**

Trematodes (Flatworms/Flukes)

Schistosoma

Life cycle:
1. Eggs hatch in freshwater and infect freshwater snail
2. Released to infect humans swimming in freshwater through exposed skin
3. Travel to intrahepatic portion of portal system
4. Schistosomes mature and mate
5. Pairs will migrate either to veins around intestine or bladder (depending on species)
6. Lay eggs which are excreted through feces or urine

Clinical Manifestations
Many of the eggs that the schistosomes pairs lay do not reach the feces or urine
> flow in bloodstream to deposit in liver, lung, or brain
> cause inflammatory response + fibrosis/granuloma formation
> can get stuck in blood vessels > fibrosis > occlusion
> cirrhosis of the liver/hepatocellular carcinoma

Diagnosis:
> Identify eggs in the stool or urine
> Differentiate between species based on spine location on egg

Tx:
Praziquantel

S. haematobium

General:
• Found in Africa
• egg has **terminal spine**
• resides in veins around **bladder**
• deposits eggs in urine
• can lead to **squamous cell carcinoma** of the bladder with painless hematuria
• pulmonary hypertension

S. japonicum

General:
• found in Eastern Asia
• egg **does not have spine**
• resides in veins around **intestine**
• deposits eggs in feces

S. mansoni

General:
• Found in South America and Africa
• egg has **lateral spine**
• resides in veins around **intestine**
• deposits eggs in feces

Clonorchis sinensis

General:
• from ingesting undercooked fish
• seen in SE Asia

Pathophysiology:
Fluke travels to the biliary tract
> cause large abscess in liver or biliary tree
> relapsing cholangitis > cholangiocarcinoma
> produce eggs that are passed in stool

Presentation:
• Pigmented gallstones
• Cholangiocarcinoma

Diagnosis:
Identify eggs in stool

Tx/Prevention:
Praziquantel

Cestodes (Tapeworms)

Tapeworms

T. solium

General:
• pork tapeworm
• eating undercooked pork infected with larvae or eggs or from food workers that don't wash hands
• causes different pathologies depending on whether larvae or egg forms are ingested
• can grow to 2-8 meters
• hermaphroditic

Pathophysiology:
Larvae encysted in undercooked pork is ingested:
> larvae attaches to mucosa of intestine via hooklets
> releases eggs in feces
> minimal symptoms

Cysticercosis:
> ingest eggs instead of hatched larvae
> eggs hatch in small intestine
> larvae travel throughout the body
> penetrate tissues and encyst
> particularly likes to encyst in brain and SKM

Presentation:
• Asymptomatic
• Cysticercosis: calcified cysticerci
• **Neurocysticercosis:** cysts in the brain > seizures, obstructive hydrocephalus, focal deficits

Diagnosis:
• Eggs in fecal sample
• CT scan or biopsy of infected tissue

Tx/Prevention:
• Albendazole + steroids for cysticercosis
• Praziquantel for intestinal

T. saginata

General:
• beef tapeworm
• only causes disease when larvae form is ingested

Pathophysiology:
• same life cycle as T. solium
• no development of cysticerci so infection considered benign

Presentation:
• malnutrition and weight loss

Diagnosis:
Identify eggs in feces

Tx/Prevention:
Praziquantel

Echinococcus granulosus

General:
• ingestion of eggs from dog feces

Pathophysiology:
Eggs are ingested and larvae hatch in intestine:
> penetrate intestinal wall and disseminate throughout body
> most go to liver; some to lungs, kidney, brain
> larvae form fluid-filled hydatid cyst that undergo asexual budding
> humans highly allergic to fluid within cysts, allergic rxn to cyst bursts may be fatal

Presentation:
• Anaphylaxis from cyst burst
• Compression of organ based on location of cyst

Diagnosis:
• CT scan for cysts
• Tissue biopsy

Tx:
• **Albendazole**
• Surgical removal

Viruses

Herpes Virus

- all DNA viruses
- Has particular cell it lays dormant in

- HHV 1 = HSV 1
- HHV 2 = HSV 2
- HHV 3 = VZV
- HHV 4 = EBV
- HHV 5 = CMV
- HHV 6 = Roseola
- HHV 7 = Roseola
- HHV 8

HSV 1: Herpes simplex 1

- acquired through sexual contact or kissing
- usually infects mucosal-integument border
- associated with oral mucosa

Pathophysiology:

- Dormant cell: cell body of neuron
- reactivation at original site of infection during time of stress
- local tissue destruction > painful ulcers

Can cause **herpes encephalitis**:

- travels from CN V to specific brain regions > hemorrhagic necrosis
- temporal lobes (auditory)
- amygdala (memory loss)
- hippocampus
- fever, RBCs in CSF

Dx: culture, PCR, serology

Tx: acyclovir, valacyclovir

HSV 2: Herpes simplex 2

- acquire through sexual contact
- usually infects mucosal-integument border
- associated with genital mucosa

Pathophysiology:

- Dormant cell: cell body of neuron
- reactivation at original site of infection during time of stress
- local tissue destruction > painful ulcers

Dx: culture, PCR, serology

Tx: acyclovir, valacyclovir

VZV: Varicella zoster

- inhaled pathogen
- present with lesions at different stages of development (as opposed to small pox)
- can occur on the eye, inside the ear

Pathophysiology:

- Dormant cell: all the cell bodies within a ganglion
- Reactivation within a particular dermatome

Vaccine:

- Zoster vaccine: for younger patients
- Shingle vaccine for older patients

Tx: acyclovir, valacyclovir

EBV: Epstein Barr Virus

- direct contact - kissing, sharing drinks

Pathophysiology:

- enters oral pharynx > lymphoid tissue (tonsils)
- infects B cells and causes massive proliferation
- Causes **Mono**: lymphadenopathy, malaise, low grade fever
- T cells fight off infection, but virus remains dormant in B cells

Lymphoma:

- In the future, if infected B cell multiplies > lots of replication errors
- mutated B cells can develop into lymphoma

Tx: no good antivirals

CMV: Cytomegalovirus

- direct contact - kissing, sharing drinks

Pathophysiology:

- Dormant cell: monocytes and endothelium
- can cause especially large giant cells

In immuno-compromised patients

- only causes disease when ↓ T cells
- CMV starts to pour out of monocytes an endothelial cells
- innate immune system starts to destroy endothelial cells
- vasculitis > end organ damage
- HIV patients: see end organ damage commonly in colon and retina (vision loss)

Dx: culture, viral loads

Tx: ganciclovir, foscarnet

HHV8:

- direct contact

Pathophysiology:

- Dormant cell: monocytes and endothelium
- Enter into the genome causing mutations and uncontrolled growth
- disease typically seen in immunocompromised patients
- Causes angiosarcoma (cancer of blood vessel)

Kaposi Sarcoma

- purple, shiny lesions on the skin
- can cause bleeding into compartments of the body

Tx: antiretrovirals, local therapy tx, chemotherapy if significant lesions

Hepatitis

- viruses are not in one family
- grouped because they all cause hepatitis

- Hep A: fecal oral, vaccine
- Hep B: body fluid, vaccine, the only DNA virus
- Hep C: body fluid
- Hep D: body fluid, needs coinfection w/ Hep B
- Hep E: fecal oral, lethal in pregnant women

Hepatitis B:

- only one that is a DNA virus
- enveloped virus
- very infective (much more infective than HIV or Hep C)

Antigens

- clearing virus depends on whether or not immune system can recognize the antigens
- S protein: surface
- C protein: core
- E protein: enzymes

Pathophysiology:

- Acute phase
- Window phase
- Chronic phase

Dx: serology, PCR;

Tx: Antivirals

Hepatitis C:

- chronic process > destruction of hepatocytes
- can either progress to hepatocellular carcinoma or cirrhosis
- IL-28R: allele of receptor determines how well patient will clear infection given treatment (C allele better chance than T allele)

Dx: serology, viral loads

Tx: cure rate >90% with proteinase inhibitors and polymerase inhibitors

HIV

- RNA virus, enveloped
- Glycoprotein 120 and 41 on the surface
- Infects CD4 T cells

Pathophysiology:

1. Fusion:
 - Glycoprotein 120 and 41 binds to CCR5 on the CD4 T cell
 - Cell membranes fuse together, allowing entry
2. Reverse transcription
 - reverse transcriptase: transcribes RNA into looped DNA
 - looped DNA can then become dsDNA
 - Targeted by: NNRTI
3. Integration:
 - Integrase: integrates dsDNA into host genome
 - Targeted by: Integrase inhibitor
4. Transcribed RNA proteins
 - Protease: cleaves peptide chain to form active protein
 - Targeted by: protease inhibitor

- Common infections based on CD4 counts:
- ≥ 200: TB, Candida, viral
 - < 200: AIDS dx, pneumocystis, cryptococcus
 - < 150: toxoplasma, histoplasma, HHV8 Kaposi sarcoma
 - < 100: cryptosporidium, MAC
 - < 50: CMV

Diagnosis:

- RNA PCR:
 - measures viral load
 - can detect within days
 - main method of diagnosis
- Antibody response:
 - test if body has produced antibody against HIV

• CD4 counts used to follow patients longterm!

Respiratory Viruses

- Orthomyxovirus
- Paramyxovirus
- Respiratory Syncytial Virus (RSV)
- Metapneumovirus

Orthomyxovirus: Influenza

- RNA virus, protein coat
- enveloped
- originally in water fowl, their domestication > infection of farm animals and humans
- surface proteins H (hemagglutinin) and N (neuraminidase) are assigned #'s based on their composition (ex. H1N1)
- Influenza Type A and Influenza Type B

Pathophysiology:

- virus can mutate within each organism it infects
- Drift: virus changes its own genetic material
- Shift: two viruses exchange genetic material
- Influenza typically causes death because of secondary bacterial infection of the lungs

- Tx:
- Amantadine - only works on type A
 - Oseltamivir - works on A and B

GI/Neuro Viruses

- Fecal oral viruses: Enterovirus (Coxsackie), Poliovirus, Norovirus, Rotavirus

Coxsackie Virus:

- pericarditis

Poliovirus:

- infects the cell bodies of the lower motor neurons within the spinal cord
- paralysis occurs
- dystrophy of muscles
- if infects C3-C5, used to cause respiratory paralysis

Non-endemic areas:

- killed vaccine

Endemic areas:

- need live vaccine because need to develop stronger immune response, but will shed live vaccine

Norovirus:

- cause fever, watery diarrhea 24-48 hours after infection
- associated with cruise ships, barracks, trains, or travel to another country

Rotavirus:

- cause watery diarrhea 24-28 hours after infection
- affects children, seen in day care centers

Neuro Viruses

- West Nile Virus
- Rabies

West Nile Virus:

- Arbovirus, spread by mosquitos through birds
- fever, chills
- encephalitic symptoms > coma
- usually affects elderly patients in the summertime

Rabies:

- neurovirus that lives in salivary glands of animals
- Bat is most commonly infected animal

Pathophysiology:

- Bite > virus enters and finds closest neuron
- causes local destruction at the cell body
- begins to move toward the head
- further the bite, the better the prognosis

Presentation:

- hydrophobia
- dry mouth with copious saliva dripping out
- Negri bodies: found in nerve cells containing virus

Tx: do everything possible

- vaccinate patient
- try to find the animal responsible for the bite