

# 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

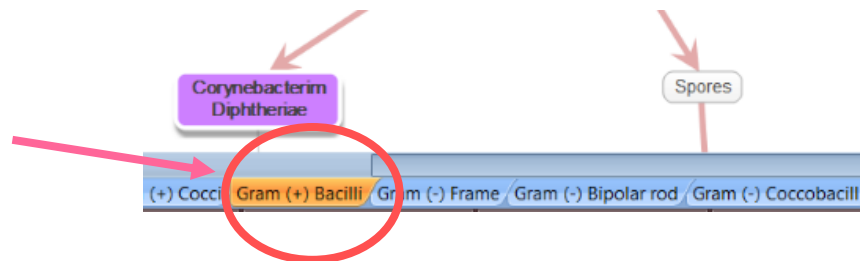
1. Download + install **MindMaple Lite** (free software):

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



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

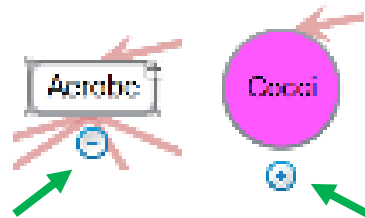
3. Toggle between the different maps using the tabs at the bottom of the screen.

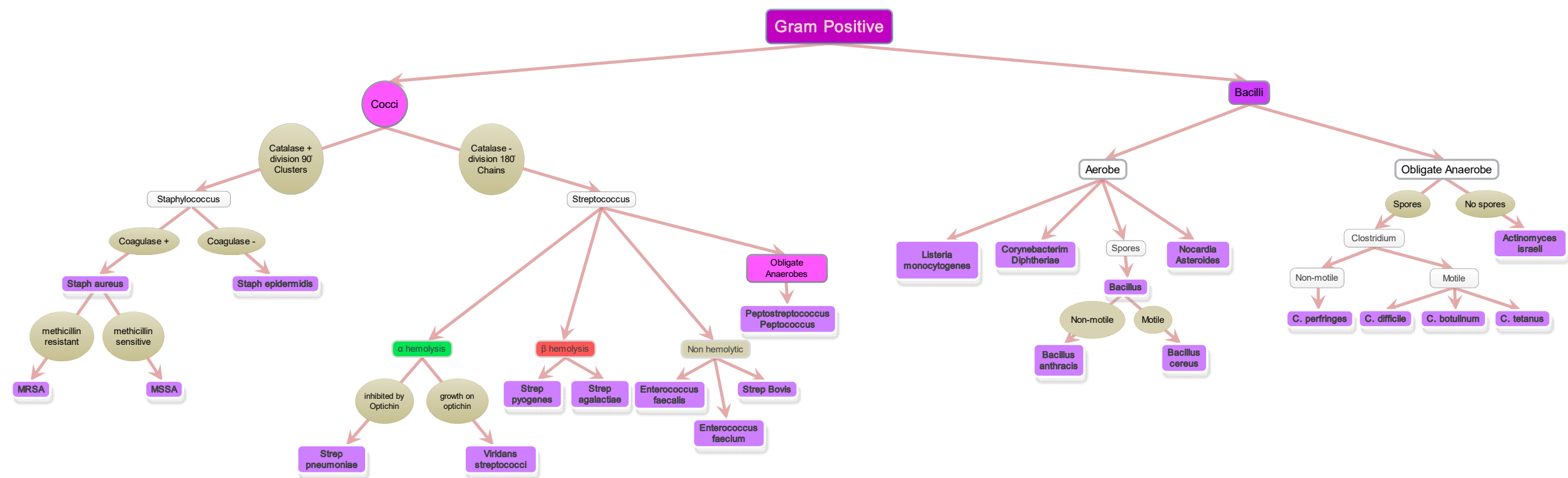


4. Two categories of maps:

- 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

#### Strep pneumoniae

#### Viridans streptococci

**General:**

- **Group A Strep**
- pus-producing

**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

**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**

**General:**

- normal gut flora
- grow well in bile
- associated with **colon cancer**

**General:**

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

**Presentation:**

- bacteremia
- sepsis

**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

**Pathophysiology:**

- babies infected during delivery > meningitis

**Pathophysiology:**

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**Diagnosis:**

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

**Pathophysiology:**

- Polysaccharide capsule protects against phagocytosis
- Asplenic patients particularly susceptible to sepsis

**Pathophysiology:**

- 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:**

- fever, vomiting, poor feeding, irritability

**Presentation:**

- Depends on type of infection

**Presentation:**

- normal gut flora
- grow well in bile
- associated with **colon cancer**

**Presentation:**

- mixed with other anaerobes in abscesses
- seen in aspiration pneumonia

**Treatment:**

- Vancomycin (if significant infection)

**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

**Presentation:**

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

**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

**Diagnosis:**

- Lumbar puncture

**Diagnosis:**

- Culture

**Diagnosis:**

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**Diagnosis:**

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**Diagnosis:**

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

**Presentation:**

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

**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

**Tx/Prevention:**

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

**Tx/Prevention:**

- Depends on type of infection

**Tx/Prevention:**

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

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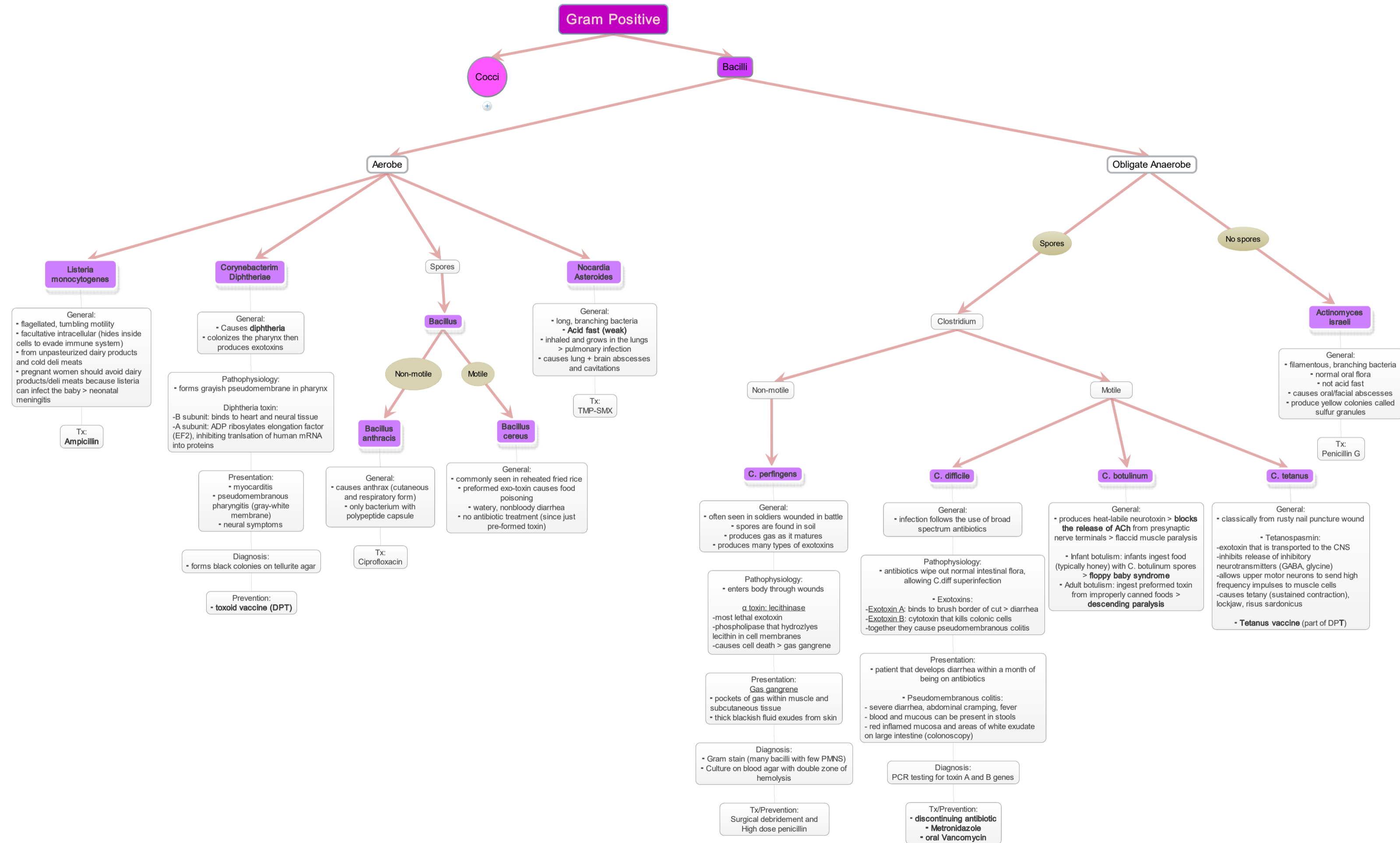
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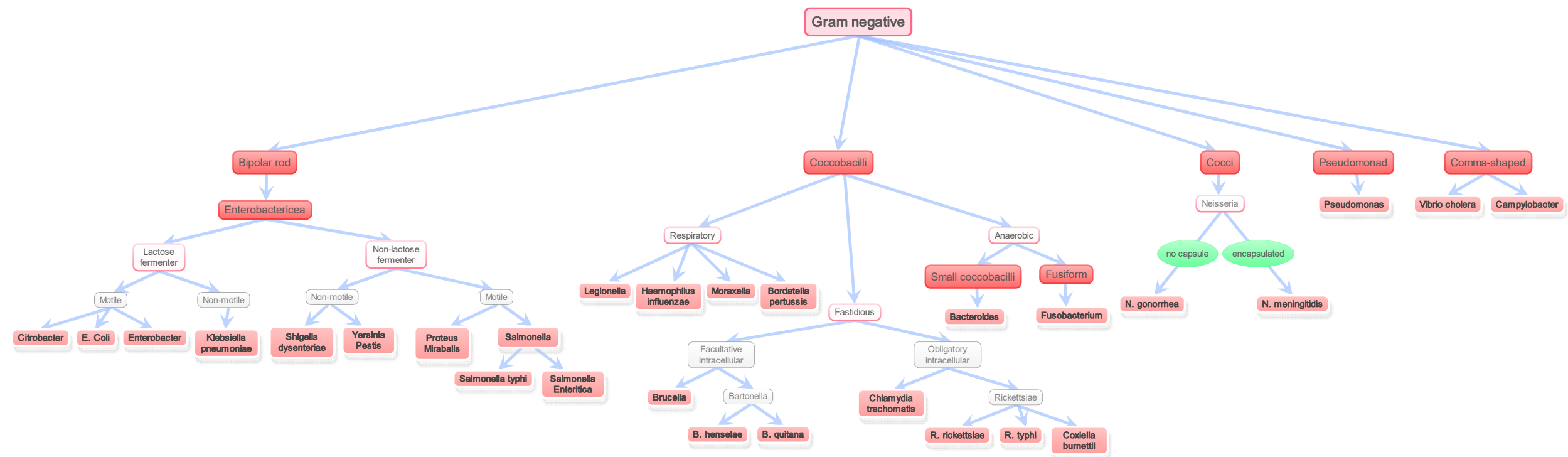
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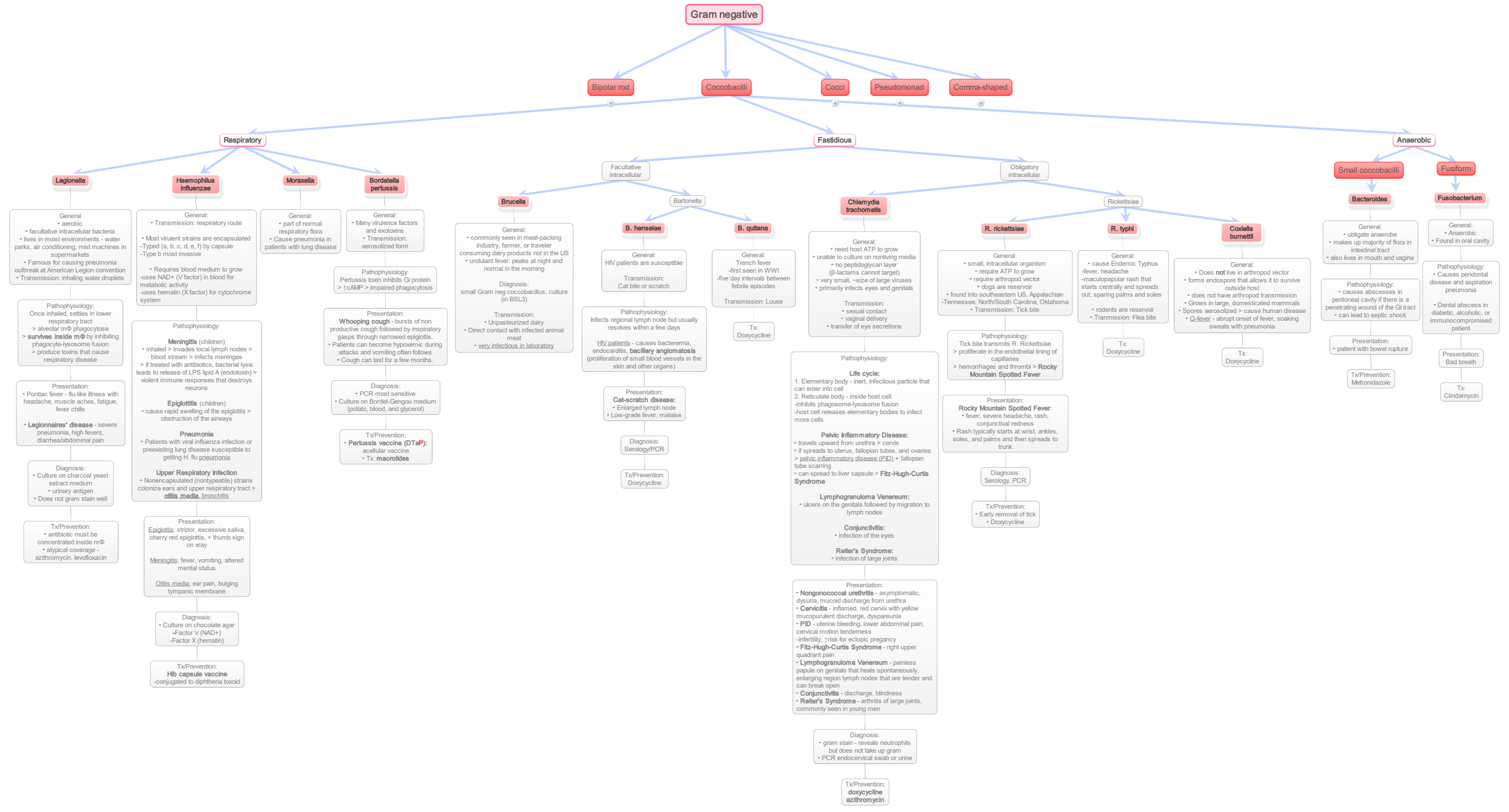
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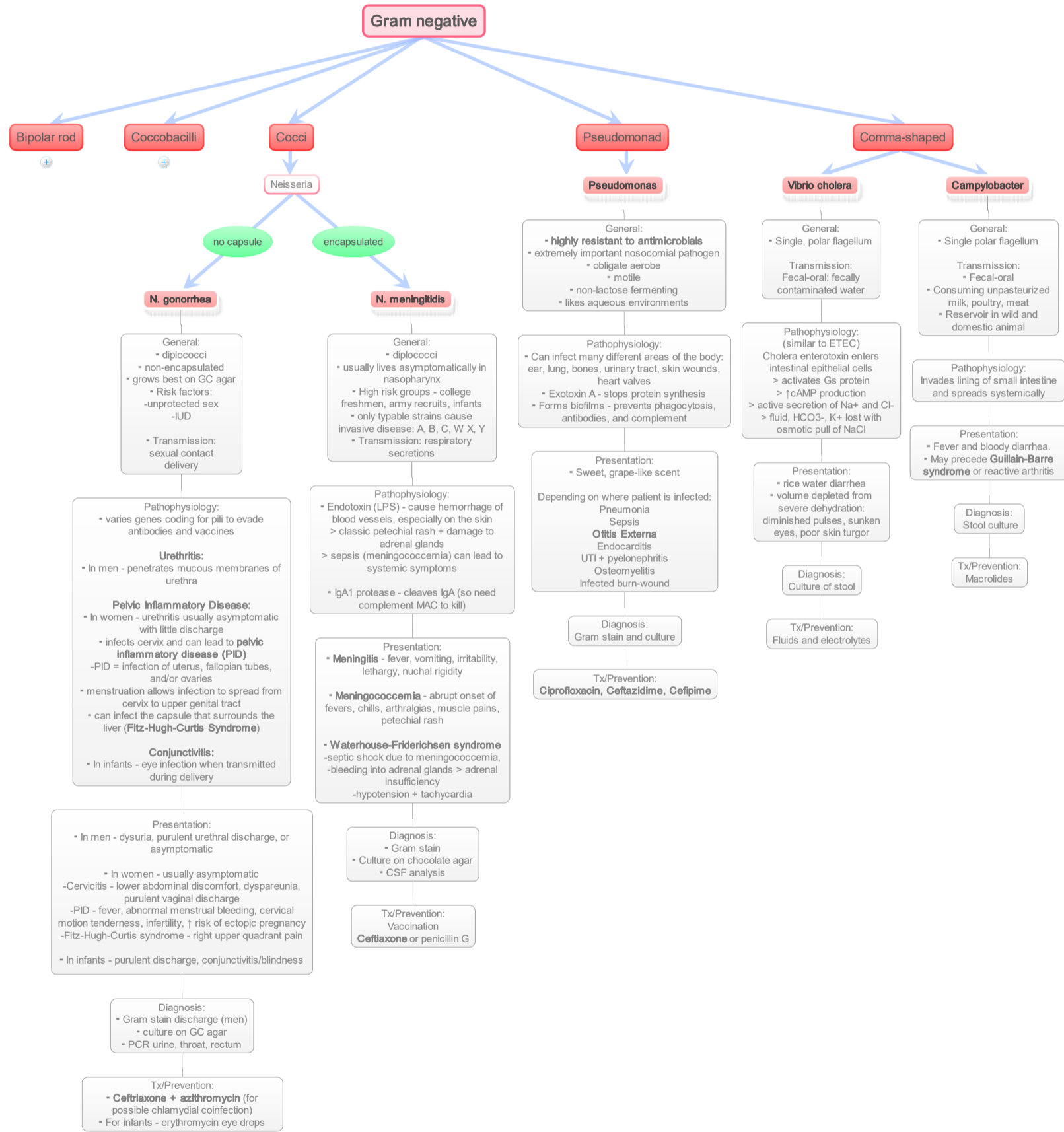


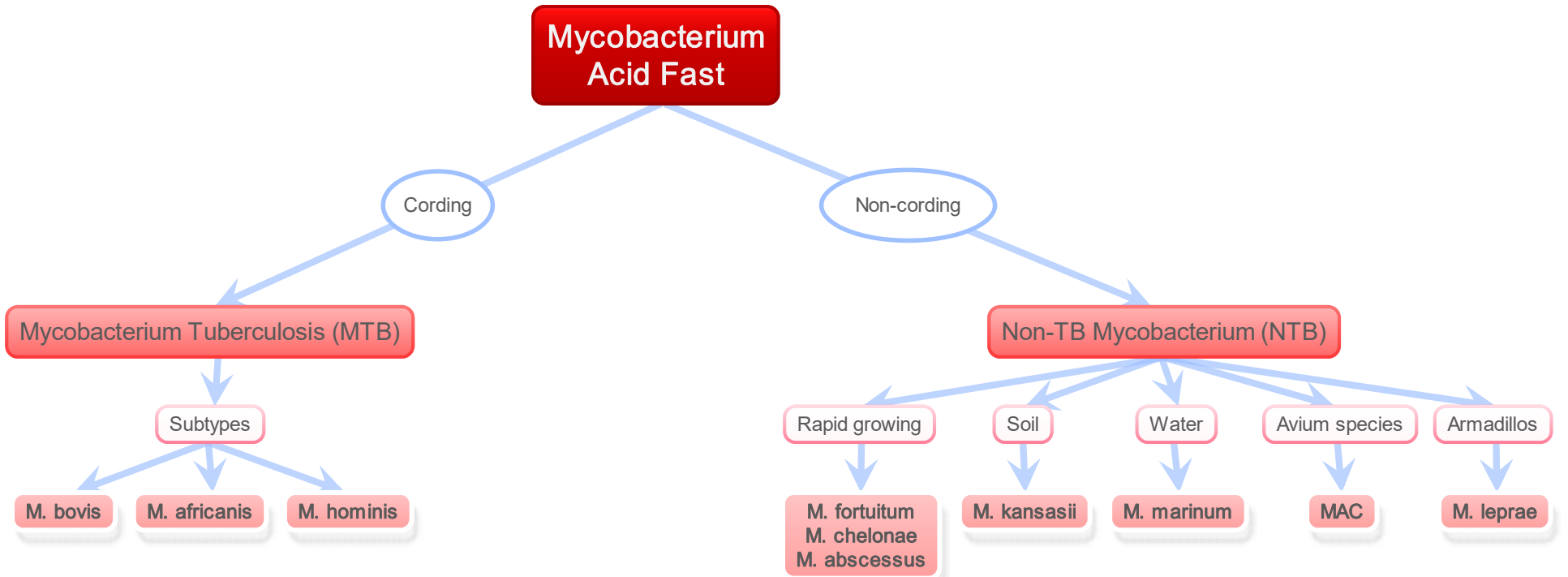












# Mycobacterium Acid Fast

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 O<sub>2</sub>) > 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  $\beta$ -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

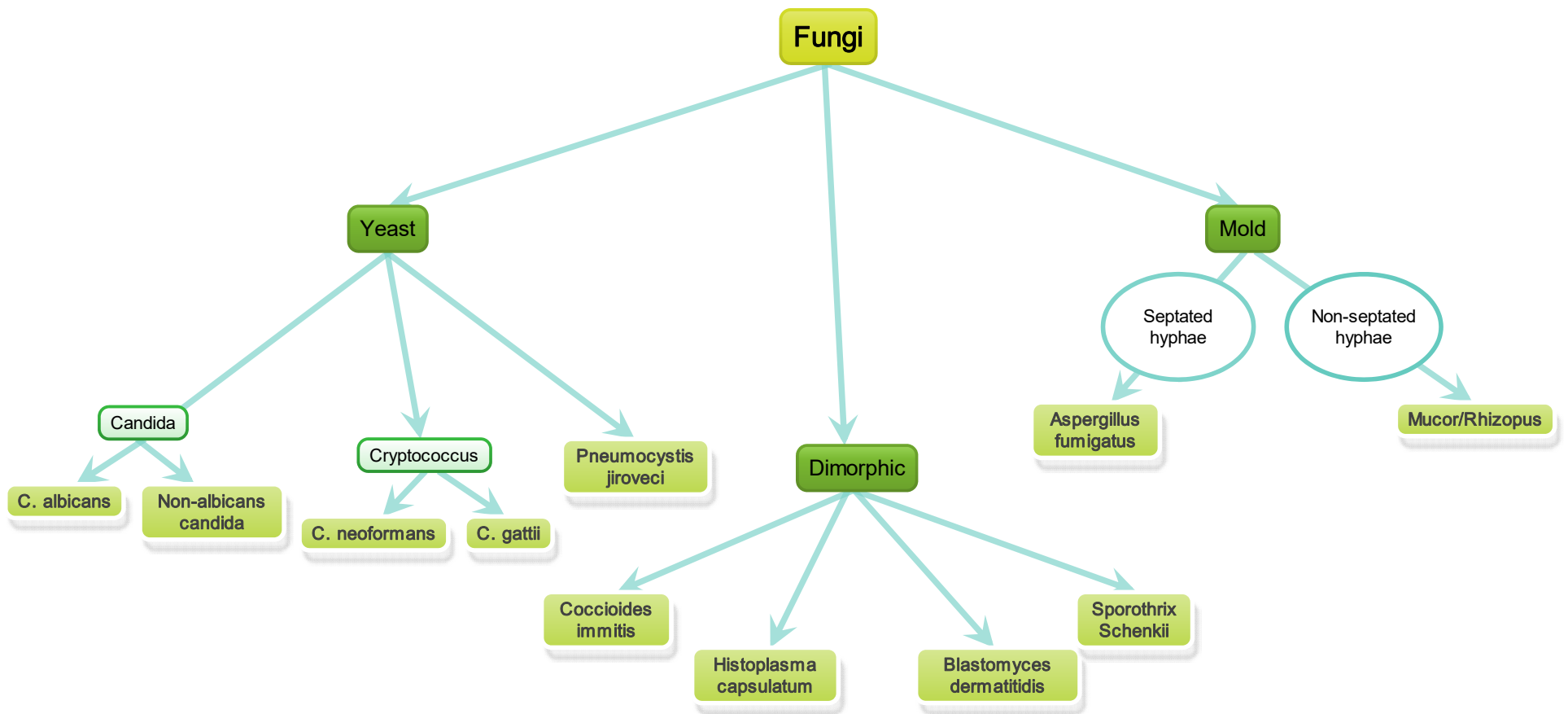
**Late stage:**  
Chronic arthritis of large joints

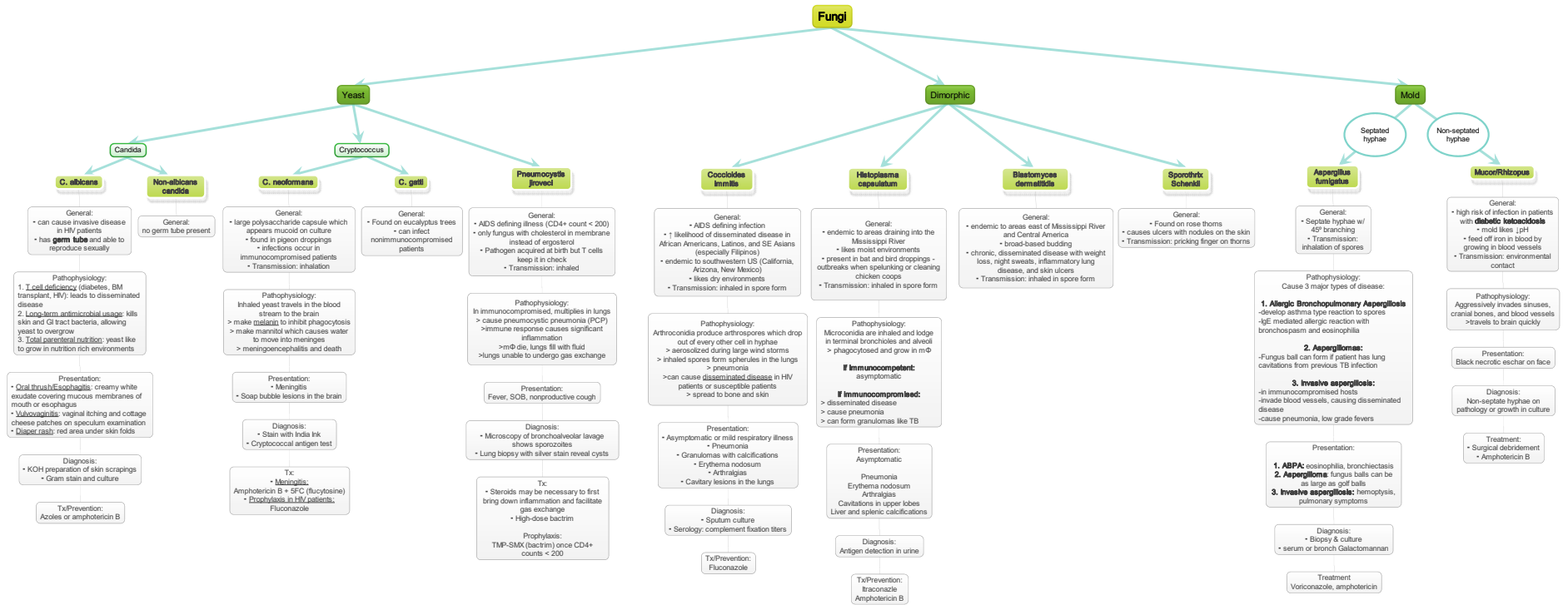
Diagnosis:

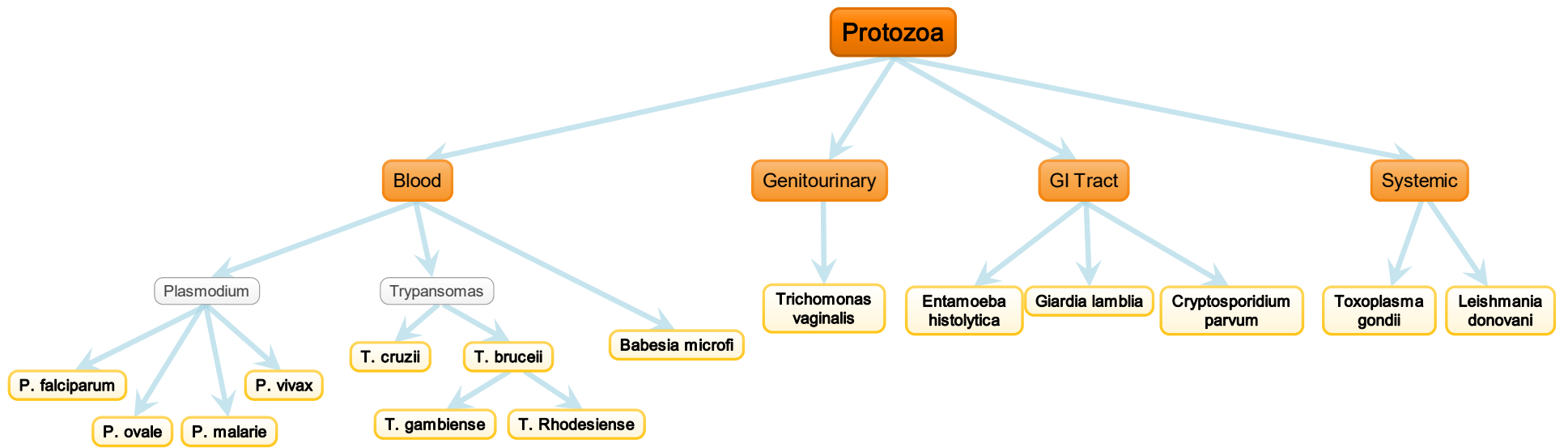
- Culture difficult
- Rely on clinical signs and serology

Tx/Prevention:

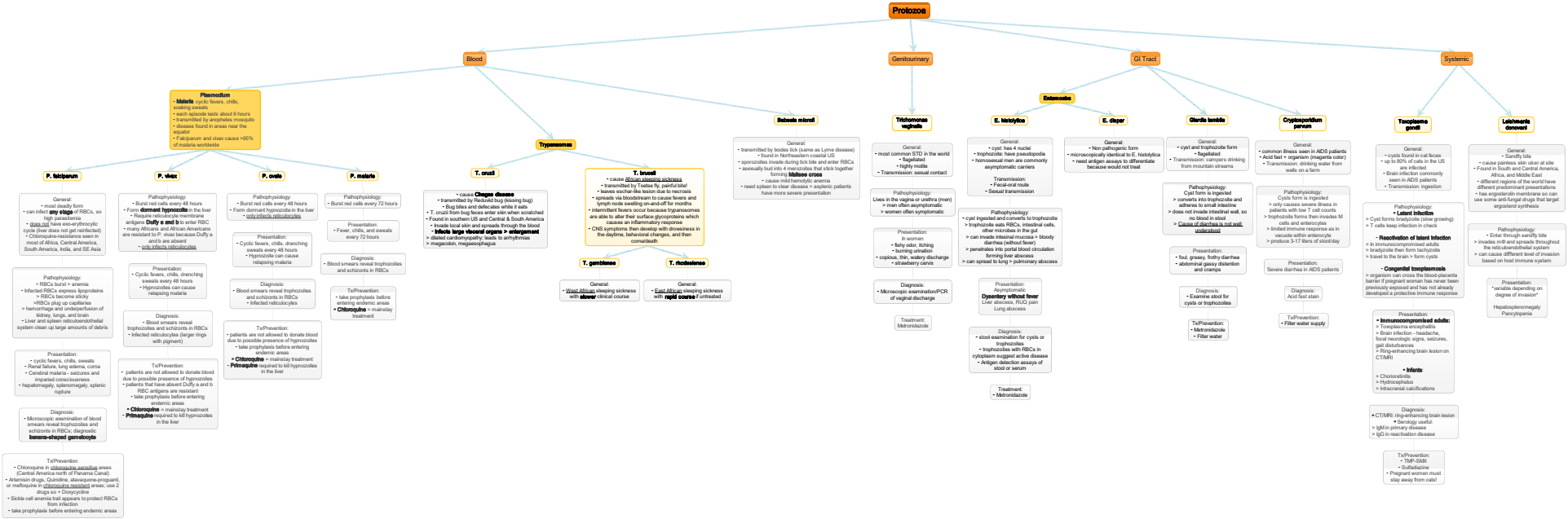
- Doxycycline
- Ceftriaxone for disseminated infection

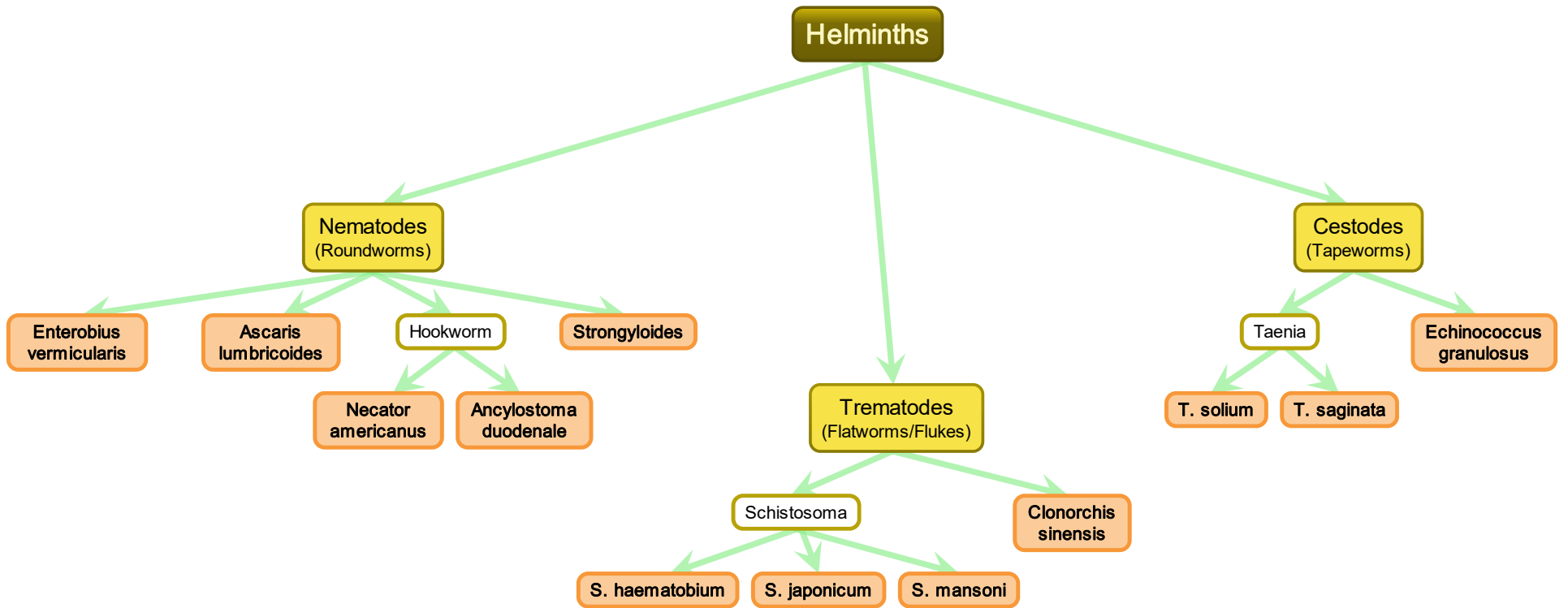


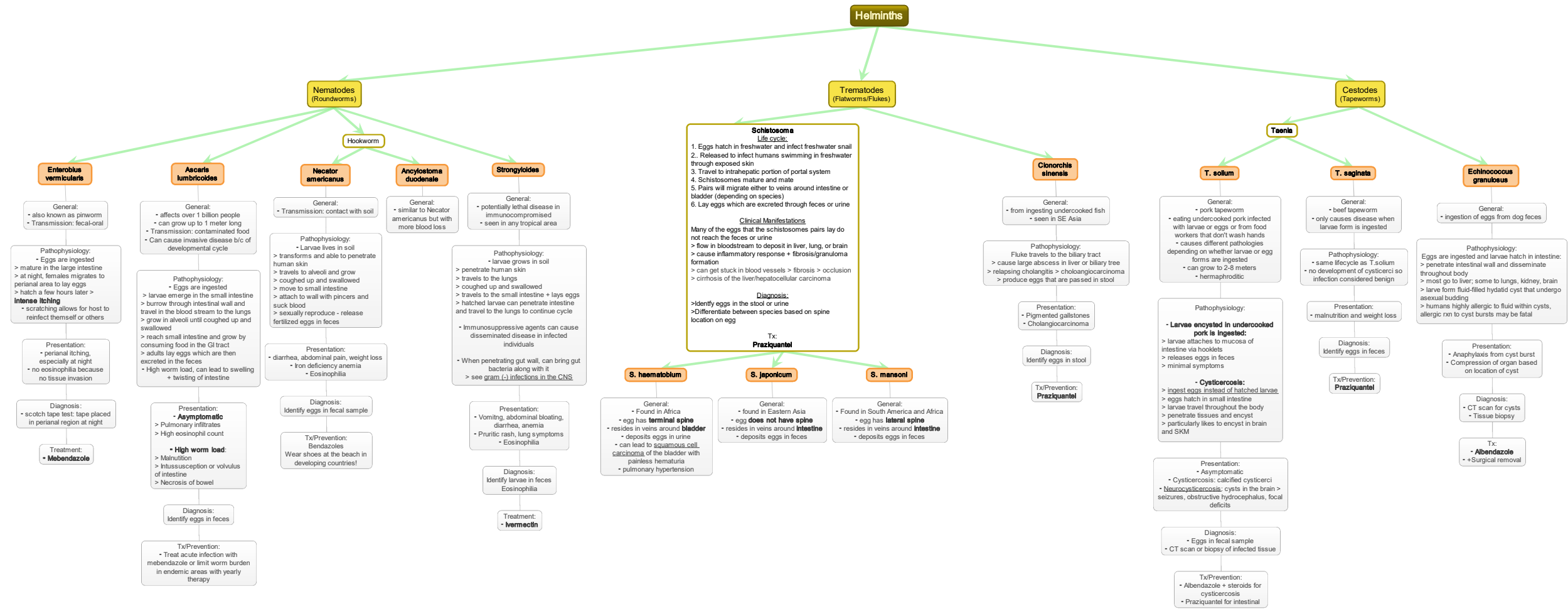












# Viruses

## 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