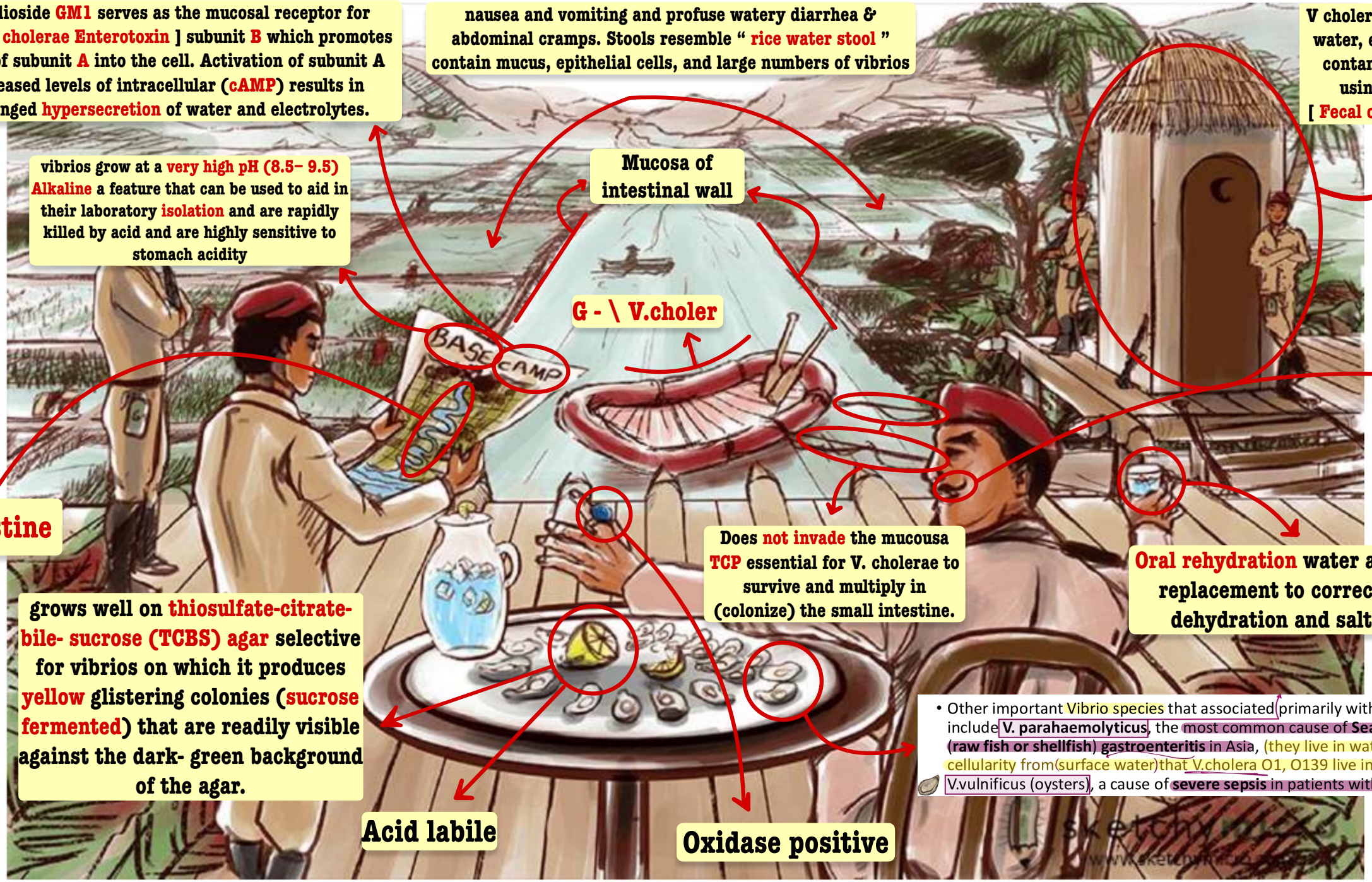

Ganglioside GM1 serves as the mucosal receptor for [Vibrio cholerae Enterotoxin] subunit B which promotes entry of subunit A into the cell. Activation of subunit A increased levels of intracellular (cAMP) results in prolonged hypersecretion of water and electrolytes.
nausea and vomiting and profuse watery diarrhea & abdominal cramps. Stools resemble " rice water stool " contain mucus, epithelial cells, and large numbers of vibrios
V cholerae transmission in water, either by drinking contaminated water or using it in cooking [Fecal oral transmission]
vibrios grow at a very high pH (8.5– 9.5) Alkaline a feature that can be used to aid in their laboratory isolation and are rapidly killed by acid and are highly sensitive to stomach acidity
Mucosa of intestinal wall
G - \ V.choler
Comma shaped not straight rods [curved]
Intestine
Does not invade the mucousa TCP essential for V. cholerae to survive and multiply in (colonize) the small intestine.
Oral rehydration water and electrolyte replacement to correct the severe dehydration and salt depletion.
grows well on thiosulfate-citrate-bile- sucrose (TCBS) agar selective for vibrios on which it produces yellow glistering colonies (sucrose fermented) that are readily visible against the dark- green background of the agar.
Acid labile
Oxidase positive
• Other important Vibrio species that associated primarily with gastrointestinal include V. parahaemolyticus, the most common cause of Sea-foodborne (raw fish or shellfish) gastroenteritis in Asia, (they live in water with higher cellularity from(surface water)that V.cholera O1, O139 live in), and V.vulnificus (oysters), a cause of severe sepsis in patients with cirrhosis and

Vibrio Cholera - Colonel Cholera's Base Camp

1. Mustache - COMMA SHAPED Gram Negative Curved rod Enteric Tract Bacilli
2. BASE in BASE cAMP – Prefers to grow in alkaline media
3. Blue Ring - Oxidase Positive - Grows on TCBS agar
4. Lemon - Grows in alkaline environments, ACID LABILE - Dies with acid
5. Rice Patties - Causes Profuse watery diarrhea **"Rice Water"** stool
6. Outhouse dumping directly into the river - Cholera is transmitted fecal oral due to poor sanitation that gets into food and is not an invasive infection
7. River walls are mucosal wall and the water is the intestinal lumen - Found in the intestines and is found in the intestinal mucosae
8. Raft that is attached to the shore - Attaches to the mucosa by fimbriae that attach to ganglioside receptors in the intestinal wall.
9. Then releases **cholera toxin - Main Virulence Factor AB type toxin**
   a. BASE cAMP map - Upregulates production of Gαs cAMP by binding to and increasing activating adenylate cyclase.
   b. GS grenade - Then it will activate the GS pathway. Activates GS, upregulates cAMP, Produces watery diarrhea through an efflux if Cl and H2O
10. Treatment
    a. Drinking some water - Oral rehydration therapy with electrolytes
11. Vibrio Vulnificus and paraliticus
    a. Oysters - Can contaminate seafood, especially oysters.
    b. Vibrio V. causes Acute Gastroenteritis
    c. Vibrio P. Causes fulminating septicemia leading to death. Marked edema and necrosis

1. Ganglioside GM1 is a mucosal receptor for subunit B, which causes toxin internalization, which promotes entry of subunit A into the cell.

2. Activation of subunit A1 >> activtes Gs >> increases levels of (cAMP) and results in prolonged hypersecretion of water and electrolytes.

- It is actively motile by means of a polar flagellum called shooting star motility

▪ V alginolyticus occasionally causes localized eye, ear, and wound (cellulitis) infections.

■ V cholerae serogroups O1 and O139 cause cholera in humans . Non-O139 strains cause mild, self-limiting cholera-like gastroenteritis

■ Most Vibrio species (like cholera) are halotolerant, and NaCl often stimulates their growth. Some vibrios (parahaemolyticus and vulnificus) are halophilic, requiring the presence (high concentration) of NaCl to grow (oceanic water).

■ Two biotypes of V. cholerae O1, classical (50% of infected mild, 50% asymptomatic) and El Tor (75% asymptomatic, 25% typical cholera symptoms), are distinguished Each biotype is further subdivided into three serotypes, termed Inaba, Ogawa, and Hikojima.

■ Prevention

1. safe water and food.

2. Patients should be isolated.

3. Three oral killed cholera vaccines:

• WC-rBS (Sweden) contains several biotypes and serotypes of V. cholerae O1 supplemented with recombinant cholera toxin B subunit.

• BiWC (India) contains several biotypes and serotypes of V. cholerae O1 and V. cholerae O139 without supplemental cholera toxin B subunit.

• Vaxchora, a live-attenuated vaccine, approved only in the USA by the FDA. It contains O1 serotype, typically Inaba strain.

Camping = Campylobacter

Comma shaped not straight rods [curved] seagull wing appearance

Guilline barre syndrome

Certain serotypes of C jejuni have been associated with post-diarrheal Guillain-Barré syndrome, a form of ascending paralytic disease. Reactive arthritis and Reiter's syndrome

Reactive arthritis

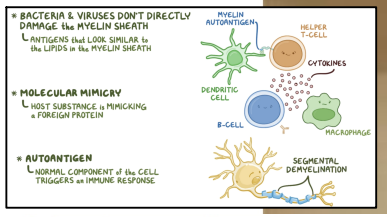
Campylobacters are found in the gastrointestinal tract of many animals used for food (poultry, cattle, sheep, and swine) and many household pets (including birds, dogs, and cats)
Main reservoir = poultry \\ Fecal oral transmission

Oxidase positive

Thermophilic bacteria =42 C°

Invasion for the intestinal mucosa

- Both the motility of the strain and its capacity to adhere to host tissues appear to favor disease, but classic enterotoxins and cytotoxins (cytolethal distending toxin, or CDT) appear not to play substantial roles in tissue injury or disease production.
- The organisms multiply in the small intestine, invade the epithelium (more specifically, Peyer's patches), and produce inflammation that results in the appearance of red and white blood cells in the stools. Occasionally, the bloodstream is invaded, and a clinical picture similar to enteric fever develops. Localized tissue invasion coupled with the toxic activity appears to be responsible for the enteritis.



- An autoimmune disease where the immune system attacks the peripheral nerves causing muscle weakness and **ascending flaccid paralysis** (in contrast to the descending paralysis caused by C. botulinum). Infections with the **O19 serotype** of **Campylobacter jejuni** can trigger Guillain-Barre. This bacterium expresses **LOS (lipooligosaccharides)** that **mimic gangliosides** on neuronal cells. The host immune system produces **anti-LOS** antibodies, which cross-react with the gangliosides present in the neuronal cells. This causes immune-mediated neuropathy commonly following **gastroenteritis** caused by Campylobacter.

Ascending paralysis

Bloody diarrhea \ stool [inflammation]

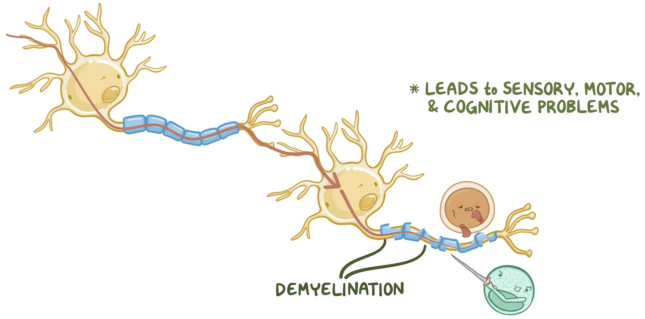
De- myelination of peripheral nerves

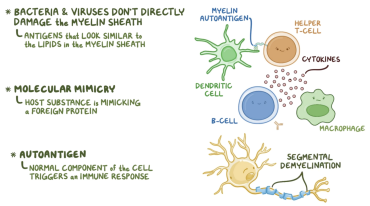
Campylobacter Jejuni - Camping Guy and the bears – guy and bears = Guillen Barre

1. Mustache is curved or comma shaped - Gram Negative Spiral / Curved Rod Bacilli - Enteric
2. Campy medium or **Skirrow Agar**
3. Microaerophilic
4. Camp Fire - Prefers warm environments around 42 deg Celsius, thermophile (Special Incubator)
5. Chicken being cooked - Main reservoir is intestinal tract of poultry and transmission is fecal oral / also contaminated water supplies or ingestion of raw milk
6. Red Stools - Bloody Stools and diarrhea
7. Blue Ring - Oxidase Positive
8. Bear cub invading the cooler - Can get Bacteremia, INVASIVE
   a. Laughing and slapping his knee - Reactive arthritis, riders syndrome
9. Bears being tripped by the sausage links on his ankle - Can cause Guillen barre syndrome due to an autoimmune response damaging myelin of peripheral nerves leading to an ascending paralysis will start at the feet then ascend.
10. Pathogenesis
    a. Bacteria Colonize intestinal Mucosa and attach to epithelial cells then replicate intracellularly causing an acute PMN response, edema of the mucosa and ulcerations. Presenting with acute enteritis and diarrhea

- C jejuni and C coli cause infections that are clinically indistinguishable (from names, we can infer C. Jejuni affect the jejunum and C. Coli the colon).

- profuse diarrhea at first that may be grossly bloody afterward.
- Local suppurative complications of infection include cholecystitis, pancreatitis, and cystitis, Hepatitis, interstitial nephritis, and hemolytic-uremic syndrome, Guillain-Barré syndrome, a form of ascending paralytic disease , reactive arthritis and Reiter's syndrome.

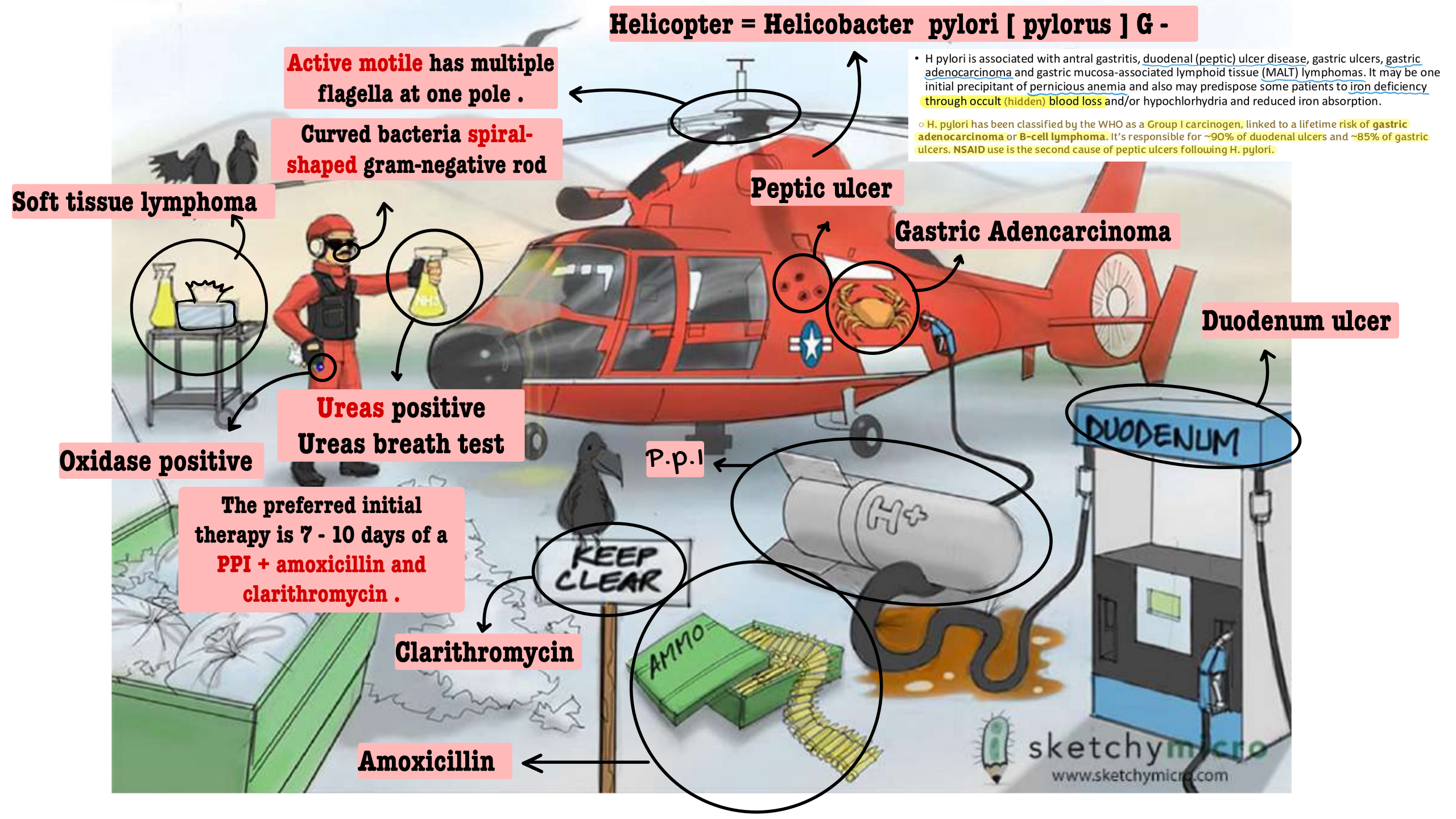




11. Treatment
    a. Supportive Care

- Fluid and electrolyte replacement.
- not all patients clearly benefit from specific antimicrobial therapy. Indications for therapy include high fever, bloody diarrhea, severe diarrhea, signs of dehydration, persistence for >1 week
>> Erythromycin.
- For systemic infections, treatment with gentamicin or imipenem or chloramphenicol should be started for C. Fetus infections.

- Domestic animals are used for food (including poultry, cattle, sheep, and swine) and many household pets (including birds, dogs, and cats).
- The infection is acquired by the oral route from food, drink, or contact with infected animals or animal products, especially poultry.



- H pylori is associated with antral gastritis, duodenal (peptic) ulcer disease, gastric ulcers, gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphomas. It may be one initial precipitant of pernicious anemia and also may predispose some patients to iron deficiency through occult (hidden) blood loss and/or hypochlorhydria and reduced iron absorption.

  - H. pylori has been classified by the WHO as a Group I carcinogen, linked to a lifetime risk of **gastric adenocarcinoma** or **B-cell lymphoma**. It's responsible for ~90% of duodenal ulcers and ~85% of gastric ulcers. **NSAID** use is the second cause of peptic ulcers following H. pylori.

Helicobacter Pylori: The helicopter Pilot

1. Red Helicopter -Curved Gram Negative rod
2. Mustache that is comma shaped - Helical slender curved rod Shape found in pylorus of the stomach
3. Not a rare infection.
4. Helicopters are motile - Motile by way of flagella
5. **Ammonia bottle - Urease positive - MAJOR VIRULENCE FACTOR** - allows to reduce the acidity of the stomach and allows Helicobacter to survive there.
6. Can be tested with Urea breath test, radioactive urea that is broken down and exhaled as CO_2 and NH_3 by urease positive organisms.
7. Blue ring - Oxidase Positive – all curved rods are oxidase positive
8. Bullet holes in helicopter, Gas pump w/duodenum - Causes 95% of all duodenal ulcers
9. Crab - Mechanism of Chronic infection causes increased acid infection. At risk of developing gastric adenocarcinoma
10. Tissues that are thrown in garbage - Patient can develop lymphoma of mucous associated lymphoid tissue.
11. Treatment
    a. Gas Pump with duodenum and H+ Bomb - Proton Pump Inhibitor
    b. Amoxicillin - ammo
    c. Crow w/ Keep Clear - Macrolide - Clarithromycin
12. Transmission
    a. Fecal Oral or Oral

Humans are the only important reservoir of H. pylori

- Urease , oxidase, and catalase positive
- microaerophilic (i.e., requires low levels of oxygen).

- if pain increases with eating, then it is a gastric ulcer, while if it decreases, it is duodenal.

- An acid-suppressing agent given for 4-6 weeks enhances ulcer healing. Proton pump inhibitors (PPIs) directly inhibit H pylori.
- The preferred initial therapy is 7-10 days of a PPI plus amoxicillin and clarithromycin or a quadruple regimen of a PPI metronidazole, tetracycline, and bismuth subsalicylate for 10 days

- Most H. pylori-colonized persons do not develop clinical sequelae. That some persons develop overt disease whereas others do not is related to a combination of factors:

a. bacterial strain differences (cag-positive , type IV secretion system, the vacuolating cytotoxin VacA)[b], host susceptibility to disease, and[c] environmental factors (the interleukin 1 gene polymorphisms, and smoking).

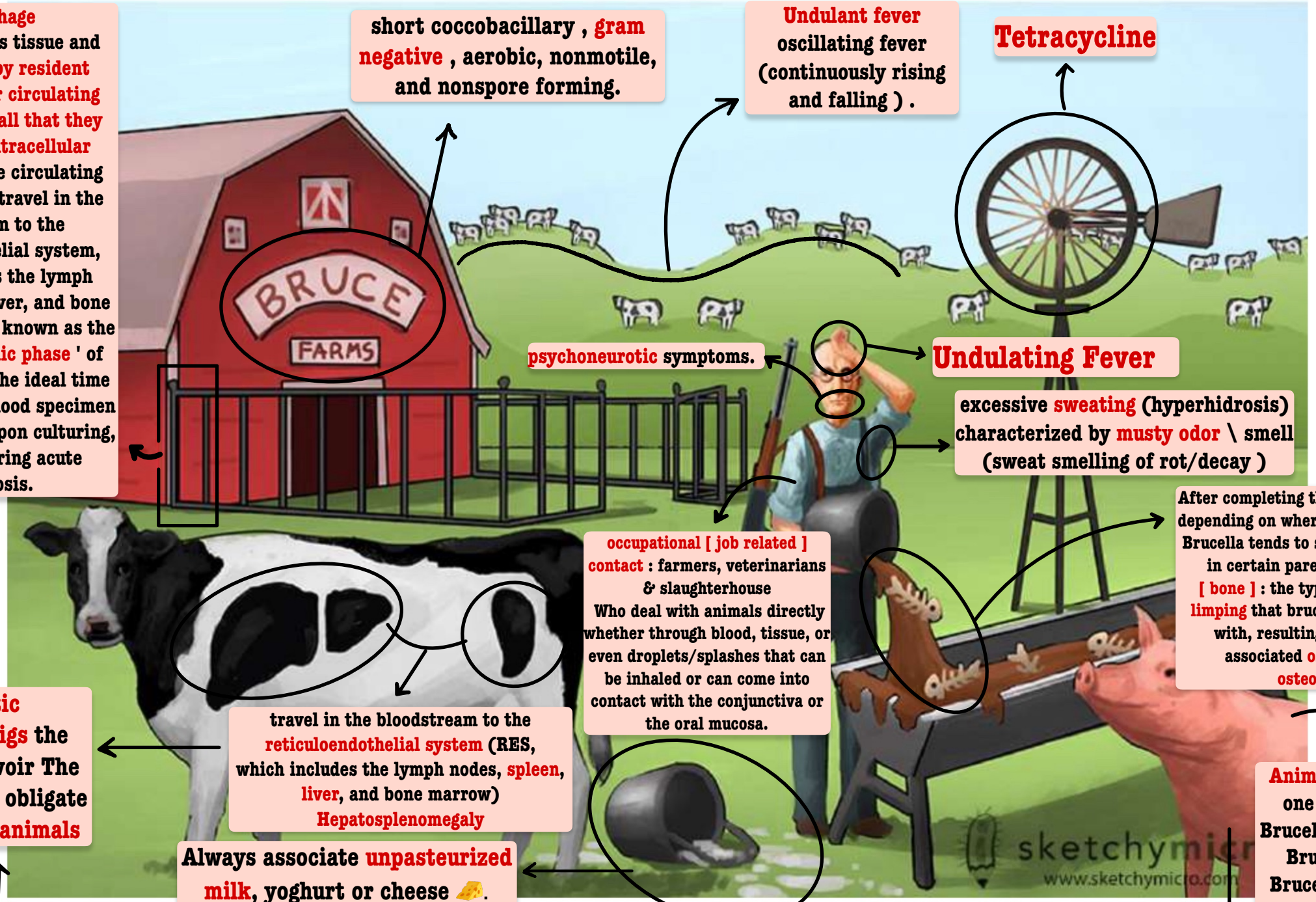
- it may be one initial precipitant of pernicious anemia and also may predispose some patients to iron deficiency .

Macrophage
Brucella crosses tissue and gets **ingulfed by resident macrophages or circulating monocytes (recall that they are obligate intracellular bacteria)**. These circulating monocytes will travel in the bloodstream to the reticuloendothelial system, which includes the lymph nodes, spleen, liver, and bone marrow). This is known as the '**acute bacteremic phase** ' of Brucella and is the ideal time for obtaining a blood specimen that is positive upon culturing, especially during acute brucellosis.

short coccobacillary , **gram negative** , aerobic, nonmotile, and nonspore forming.

Undulant fever
oscillating fever (continuously rising and falling) .

Tetracycline



psychoneurotic symptoms.

Undulating Fever

excessive **sweating** (hyperhidrosis) characterized by **musty odor** \ smell (sweat smelling of rot/decay)

occupational [job related] contact : farmers, veterinarians & slaughterhouse Who deal with animals directly whether through blood, tissue, or even droplets/splashes that can be inhaled or can come into contact with the conjunctiva or the oral mucosa.

After completing this **bacteremic trip** and depending on where the **granuloma** forms, Brucella tends to settle (**chronic stage**) in certain parenchymatous organs **[bone]** : the typical presentation of **limping** that brucellosis patients come with, resulting from brucellosis-associated **osteoarthritis** and **osteomyelitis**).

Zoonotic
Cows and pigs the mains reservoir The brucellae are obligate parasites of **animals**

travel in the bloodstream to the **reticuloendothelial system** (RES, which includes the lymph nodes, **spleen**, **liver**, and bone marrow)
Hepatosplenomegaly

Always associate **unpasteurized milk**, yoghurt or cheese 🧀.

Animal pathogens each one **Preferred host** :
Brucella **melitensis** goats
Brucella **suis** swine
Brucella **abortus** cattle
Brucella **canis** dogs

Bacteria	Characteristics	Disease	Disease management	Notes
Brucellae	**Morphology** -G –ve coccobacilli -unencapsulated -nonmotile - obligate parasites of animals & humen **Biochemical** -intracellular -aerobic except abortus (requires 5-10% CO2) -complex nutritional requirements (fastidious) -catalase & oxidase +ve -resistant to freezing & drying … but killed by boiling & pasteurization **Culture** -appear predominantly as short coccobacilli in young cultures. -smooth & transparent colony	**Brucellosis (undulant, malta, Mediterranean, Cyprus fever)** -an acute bacteremic phase followed by a chronic stage that may extend over many years & involve many tissues. **Transmission** -unpasteurized milk or milk products (goat cheese) -occupational contact (farmers, vets…) → transmitted by ingestion / inhalation (biological weapon M&S) / skin & mucosal exposure (healthcare providers vs. needle sticks) **Pathogenesis** -enters lymphatics → blood → distribution to different organs → forming granulomatous granules in reticuloendothelial system (brucella here is intracellular) → granules develop into abscesses. -granuloma: contain epithelioid & giant cells , with central necrosis & peripheral fibrosis. -spread in the blood can lead to osteomyelitis, meningitis, cholecystitis. -main histologic reaction is proliferation of PMNs, fibrosis & coagulation necrosis. **Symptoms** -incubation : 1-4 weeks -insidious onset : malaise, fever, weakness, aches, sweats -undulant fever : rises in the afternoon & falls at night متموجة -sustemic effects: -gastrointestinal& nervous symptoms -LN enlargement & palpable spleen -hepatitis & jaundice -osteomyelitis (abnormal standing & deep pain in sacroiliac joint (young age) or low back pain (older) -general symptoms subside after weeks or months ,but local lesions may continue. -a chronic stage may develop after acute infection characterized by aches, low grade fever & psychoneurotic symptoms. (miserable disease)	**Diagnosis** 1-specimens : -blood → for culture -biopsy (LN, bone…) → for culture -serum → serologic tests 2-culture : -brucella agar → specifically designed for brucella Highly inriched (since its fastidious) -trypticase soy medium -chocolate agar …. 3-serology -IgM → rises during the 1st week of acute illness + peaks at 3 months -IgG & IgA → rise after 3 weeks of onset + peaks at 6-8 weeks + remains high during chronic course A-agglutination test → IgG agglutinin titers above 1:80 in active infection . → cholera vaccine may develop false +ve B-ELISA assays → use cytoplasmic proteins as Antigens More specific & sensitive **→ culture needs a long time. So, serology is preferred** **Treatment** -G-ve antibiotics -not easily eradicated due to intracellular location -for best results: treatment must be prolonged. Combined treatment with a tetracycline (eg, doxycycline) and either streptomycin for 2–3 weeks or rifampin for 6 weeks is recommended. **Prevention** -animal vaccine: -B.abortus → live att. S19 & RB51 -B.melitensis → Rev1 -humen vaccine → still experimental -pasteurization of milk	-types : -melitensis → infects goats -suis → swines -abortus* → cattle -canis → dogs → all can infect humen by zoonotic infection (by accidental contact with feces, urine…) -B.abortus is named so bc it can cause abortion to cattles. However , it cant do so for humen (no erythrol in placenta)

occurs most commonly in the tropics and subtropics because of the climate

Kidney involvement in many animal species is chronic and results in the shedding of large numbers of leptospirae in the urine .
Renal dysfunction nephritis [nitrogen retention]

Contaminated water with animal urine Leptospira is transmitted through the urine of infected animals

swamp fever \ mud feve and sweating

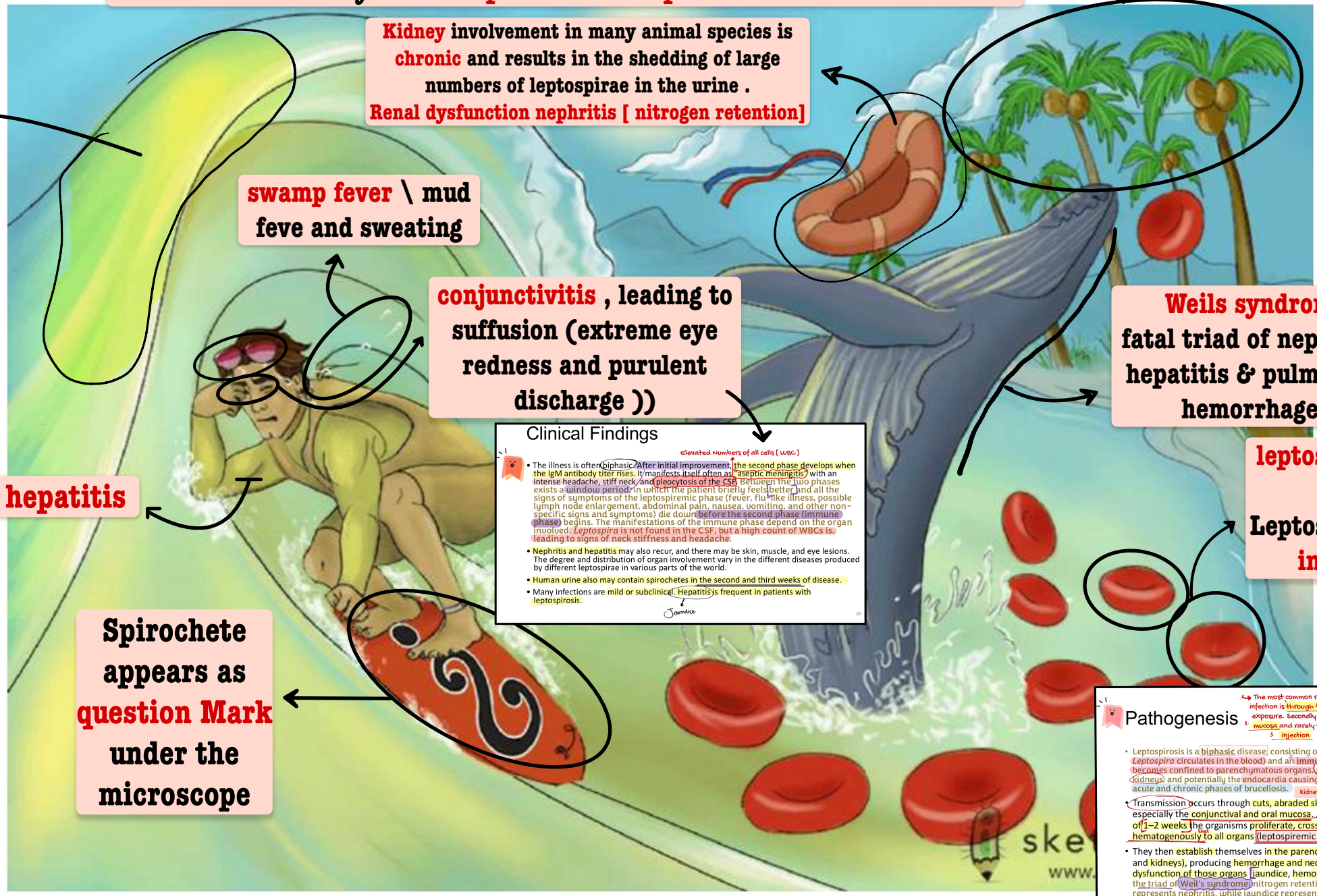
conjunctivitis , leading to suffusion (extreme eye redness and purulent discharge))

Weils syndrome
fatal triad of nephritis, hepatitis & pulmonary hemorrhage)

leptospiremic phase (in which Leptospira circulates in the blood)

Jaundice \ hepatitis

Spirochete appears as question Mark under the microscope



Clinical Findings

Elevated Numbers of all cells [WBC]

- The illness is often biphasic. After initial improvement, the second phase develops when the IgM antibody titer rises. It manifests itself often as "aseptic meningitis" with an intense headache, stiff neck, and pleocytosis of the CSF. Between the two phases exists a window period, in which the patient briefly feels better and all the signs of symptoms of the leptospiremic phase (fever, flu-like illness, possible lymph node enlargement, abdominal pain, nausea, vomiting, and other non-specific signs and symptoms) die down before the second phase (immune phase) begins. The manifestations of the immune phase depend on the organ involved. *Leptospira* is not found in the CSF, but a high count of WBCs is, leading to signs of neck stiffness and headache.
- Nephritis and hepatitis may also recur, and there may be skin, muscle, and eye lesions. The degree and distribution of organ involvement vary in the different diseases produced by different leptospirae in various parts of the world.
- Human urine also may contain spirochetes in the second and third weeks of disease.
- Many infections are mild or subclinical. Hepatitis is frequent in patients with leptospirosis.

Jaundice

Pathogenesis

The most common root of infection is through the skin exposure. Secondly threw mucosa and rarely threw injection

لما تشوفها بالدم

- Leptospirosis is a biphasic disease, consisting of a leptospiremic phase (in which *Leptospira* circulates in the blood) and an immune phase (in which Leptospira becomes confined to parenchymatous organs (most commonly the liver and the kidneys) and potentially the endocardia causing endocarditis). This is similar to the acute and chronic phases of brucellosis.

kidney بتكون بال antibodies لما تختفي من الدم و تبلش تطلع ال liver و ال

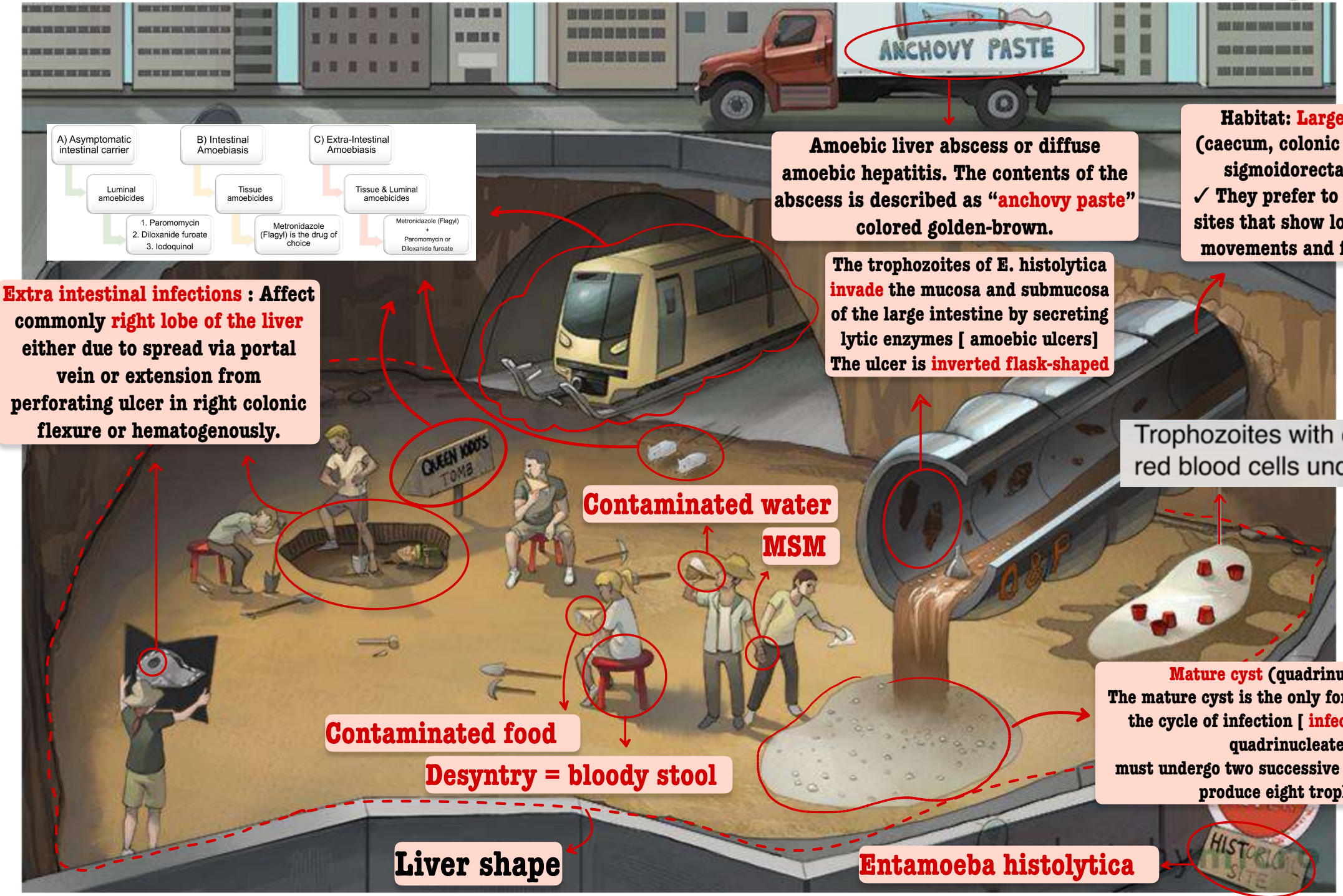
- Transmission occurs through cuts, abraded skin, or mucous membranes, especially the conjunctival and oral mucosa. After entry, and an incubation period of 1–2 weeks the organisms proliferate, cross tissue barriers, and disseminate hematogenously to all organs (leptospiremic phase) = Conjunctivitis ,musculoskeletal disorders , mialgia
- They then establish themselves in the parenchymatous organs (particularly liver and kidneys), producing hemorrhage and necrosis of tissue and resulting in dysfunction of those organs [jaundice, hemorrhage, nitrogen retention]. This is the triad of Weil's syndrome. Nitrogen retention is a sign of renal failure and represents nephritis, while jaundice represents hepatitis.

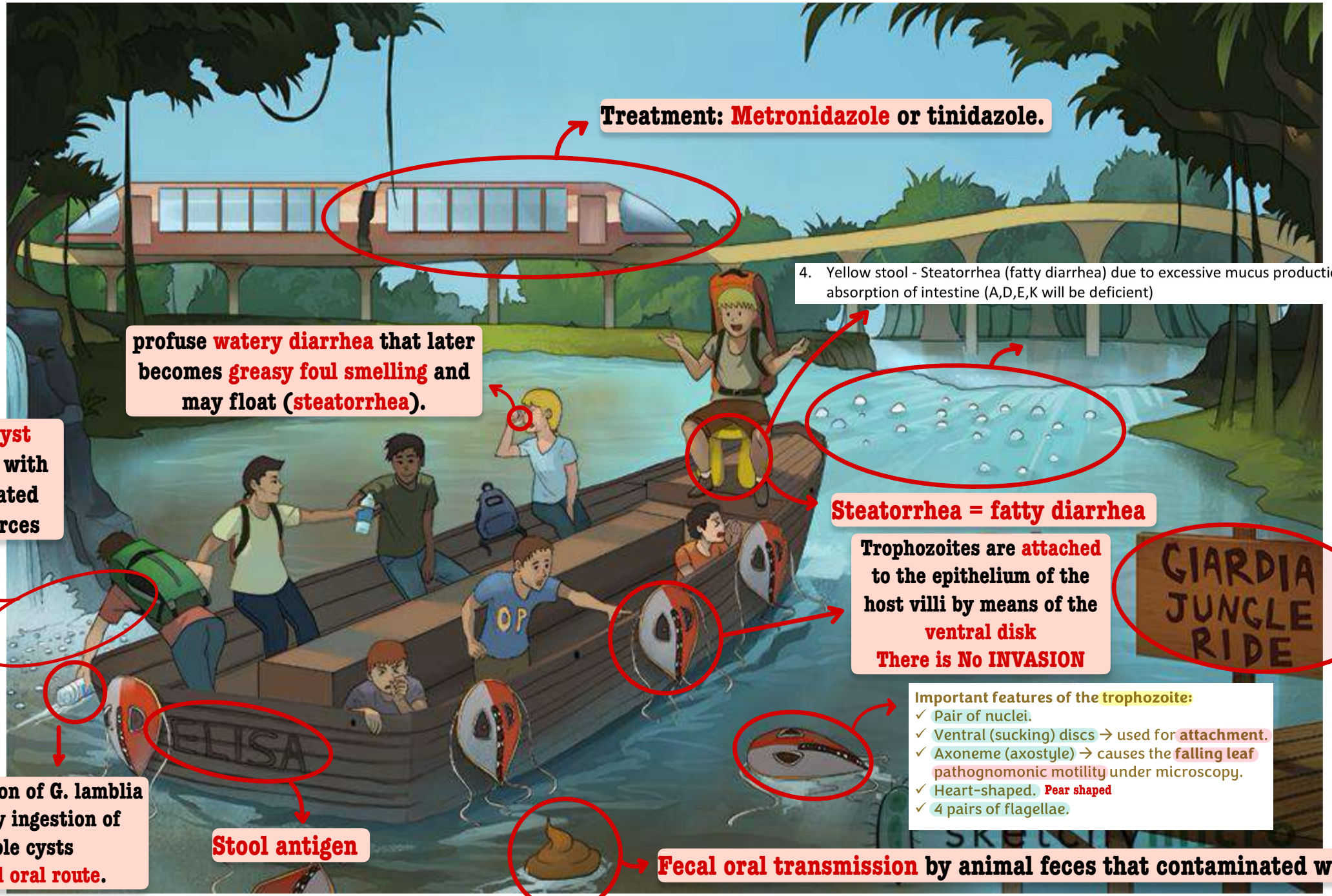
▪ It is biphasic : it has 2 phases –> leptospirosis phase (circulated in blood circulation, then they establish themselves in parenchymal organs, it also named , they prefer liver and kidneys) and parenchymatous phases .

Treatment
- mild >> oral doxycycline, ampicillin, or amoxicillin.
- Severe >> IV penicillin. why ? Because leptospira has penicillin binding proteins

Prevention : (mainly by control of rats as they are the main reservoir).
- Avoidance of exposure to urine and tissues from infected animals through proper eyewear, footwear, and other protective equipment.
- Vaccines for agricultural and companion animals are generally available.

Bacteria	Characteristics	Disease	Disease management	Notes
Leptospira	**Morphology (dark field microscope)** -G-ve spirochete (thin, tightly coiled with double membrane) -unencapsulated -motile (2 periplasmic flagella) -question mark appearance **Biochemical** -derive energy from oxidation of long chain F.A & cannot use A.A or carbs. -derive nitrogen from ammonium salts -can survive for weeks in water , particularly alkaline (urine is a great environment) -aerobic	**Leptospirosis** broad spectrum of clinical manifestations → varying from asymptomatic infection (90%) to fulminant, fatal disease (Weil's Syndrome) (10%) **transmission** -main source of infection is animal urine (kidney involvement in many animals is chronic & results in shedding of large numbers of leptospira) -humen urine may also contain leptospira in 2nd or 3rd weeks → transmitted through cuts, abraded skin, mucosa especially oral & conjunctiva. **Pathogenesis** -leptospiremic phase → invasion & hematogenous spread -immune phase → leptospira establish in the liver & kidneys mainly … producing hemorrhage & necrosis (jaundice & nitrogen retention) **Symptoms** -incubation → 1-2 weeks -second phase → starts when IgM Ab titer & manifests as: -aseptic meningitis -nephritis -hepatitis -skin, muscle, eye lesions -LN enlargement	**Diagnosis** 1-specimens: -blood -CSF -urine 2-microscopic examination -dark field -giemsa technique 3-culture -aerobic conditions at 28-30 C in semisolid medium (eg, Ellinghausen-McCullough-Johnson- Harris EMJH) in 10 mL test tubes with 0.1% agar and 5-fluorouracil. -growth is slow (you cant depend on it) 4-serology -microscopic agglutination test (MAT) -ELISA **Treatment** -mild leptospirosis → oral doxy, ampicillin or amoxicillin -severe (wiles, hepatitis, nephritis) → IV penicillin as soon as possible (leptospira has penicillin binding proteins) Immunity → serovar specific immunity after infection (but infection with other serovars is possible). **Prevention** -control of rats (main reservoir) -avoid exposure to urine (especially sewer workers) -avoid exposure to tissues of infected animals -vaccine → for animals	-types : -pathogenic → interrogans -free living → biflexa **Epidemiology** Distributed worldwide … most commonly in tropics & subtropics (climate & poor hygiene) ▪ Kidney involvement in many animal species is chronic and results in the shedding of large numbers of leptospirae in the urine; this is probably the main source of environmental contamination resulting in infection of humans. ▪ Human urine also may contain spirochetes in the second and third weeks of disease.


ANCHOVY PASTE
A) Asymptomatic intestinal carrier
Luminal amoebicides
1. Paromomycin
2. Diloxanide furoate
3. Iodoquinol
B) Intestinal Amoebiasis
Tissue amoebicides
Metronidazole (Flagyl) is the drug of choice
C) Extra-Intestinal Amoebiasis
Tissue & Luminal amoebicides
Metronidazole (Flagyl) + Paromomycin or Diloxanide furoate
Amoebic liver abscess or diffuse amoebic hepatitis. The contents of the abscess is described as "anchovy paste" colored golden-brown.
Habitat: Large intestine (caecum, colonic flexures and sigmoidorectal region).
✓ They prefer to stay at these sites that show low peristaltic movements and fecal stasis .
Extra intestinal infections : Affect commonly right lobe of the liver either due to spread via portal vein or extension from perforating ulcer in right colonic flexure or hematogenously.
The trophozoites of E. histolytica invade the mucosa and submucosa of the large intestine by secreting lytic enzymes [amoebic ulcers]
The ulcer is inverted flask-shaped
Trophozoites with endocytosed red blood cells under microscope
Contaminated water
MSM
Contaminated food
Desyntry = bloody stool
Mature cyst (quadrinucleate cyst).
The mature cyst is the only form that can continue the cycle of infection [infective stage] . This quadrinucleate cyst
must undergo two successive mitotic divisions to produce eight trophozoites.
Liver shape
Entamoeba histolytica


Treatment: Metronidazole or tinidazole.
4. Yellow stool - Steatorrhea (fatty diarrhea) due to excessive mucus production that impairs absorption of intestine (A,D,E,K will be deficient)
profuse watery diarrhea that later becomes greasy foul smelling and may float (steatorrhea).
Giardia cyst associated with contaminated water sources
Steatorrhea = fatty diarrhea
Trophozoites are attached to the epithelium of the host villi by means of the ventral disk
There is No INVASION
GIARDIA JUNGLE RIDE
Important features of the trophozoite:
✓ Pair of nuclei.
✓ Ventral (sucking) discs → used for attachment.
✓ Axoneme (axostyle) → causes the falling leaf pathognomonic motility under microscopy.
✓ Heart-shaped. Pear shaped
✓ 4 pairs of flagellae.
Transmission of G. lamblia occurs by ingestion of viable cysts by fecal oral route.
Stool antigen
Fecal oral transmission by animal feces that contaminated with cyst