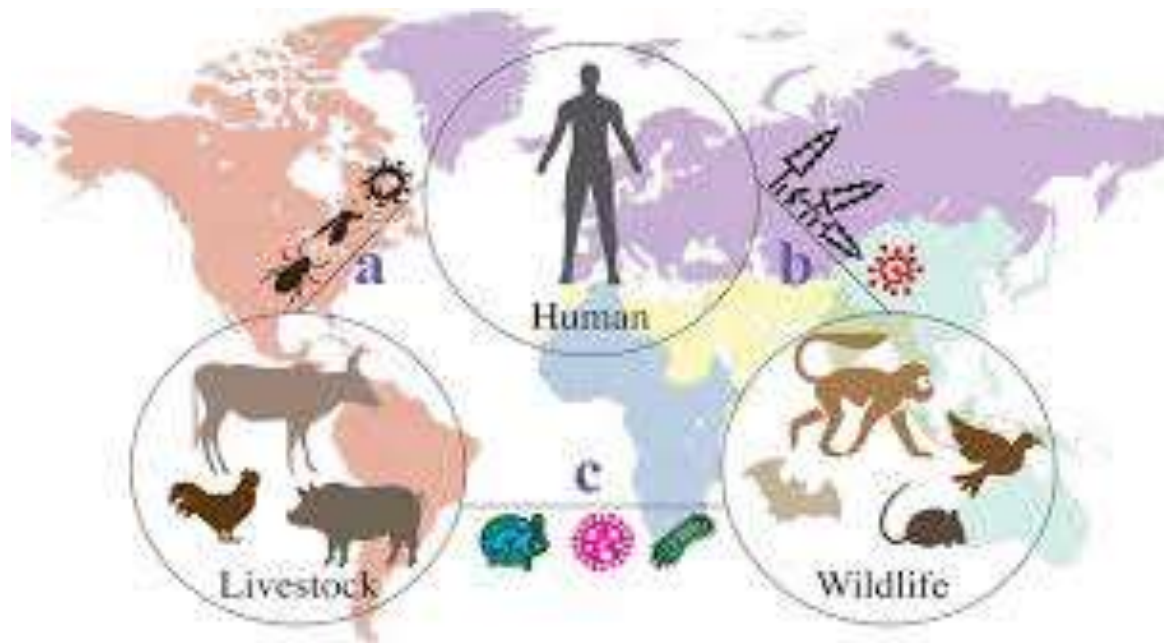




**CAUSES OF ZOONOSES AND
SEXUALLY TRANSMISSIVE DISEASES.
SPIRAL AND OBLIGATORY
INTRACELLULAR BACTERIA**

Zoonoses

- Infections and diseases of animals whose pathogens can be transmitted to humans under natural conditions.
- • **Reservoirs**: forest animals, rodents, mice, etc.
- • **Causative agents**: bacteria, viruses, parasites, fungi, rickettsiae, etc.



Zoonoses

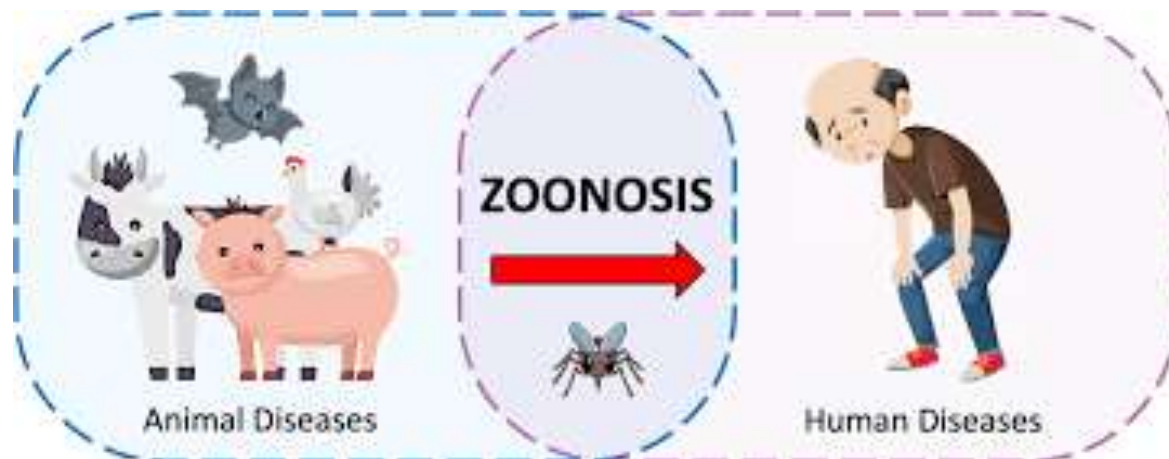
- **Transmission:**

- directly

- Direct contact with animals and their products (urine)
 - A bite or scratch from an infected animal

- indirectly


- Inhalation of infectious aerosol
 - Ingestion of infected animal products (meat, milk) that have not been sufficiently thermally processed
 - Feco-oral transmission
 - Vectors (ticks, mosquitoes)




Zoonoses

- • Polymorphic clinical picture
- • The most common diseases in our country are: salmonellosis, Lyme disease and trichinosis.
- • Zoonoses also include anthrax, rabies, brucellosis, leptospirosis, tularemia, Q fever, psittacosis etc.
- • **Prevention and treatment-multidisciplinary approach** and require joint cooperation between doctors and veterinarians.


6 WAYS TO BETTER CONTROL ZOOSES




1. Education first:
Focus on what people can do to protect themselves.




2. Vaccines are a vital tool and must be made available:
For example, it only costs \$4 to vaccinate a dog against rabies.




3. Enhanced surveillance:
Tracking data to better understand the disease.



4. One Health:
Realistic, achievable plans that bring everyone together.



5. Harness digital:
Sharing data and knowledge means effective strategies can be more widely adopted.



6. Positive feedback:
Hearing about successes inspires people to do more.

#AnimalHealthMatters

Health for Animals
global animal health association

Zoonoses

- animal diseases
- from animals to humans
- Rarely are transmitted from person to person
- bacteria, viruses, parasites

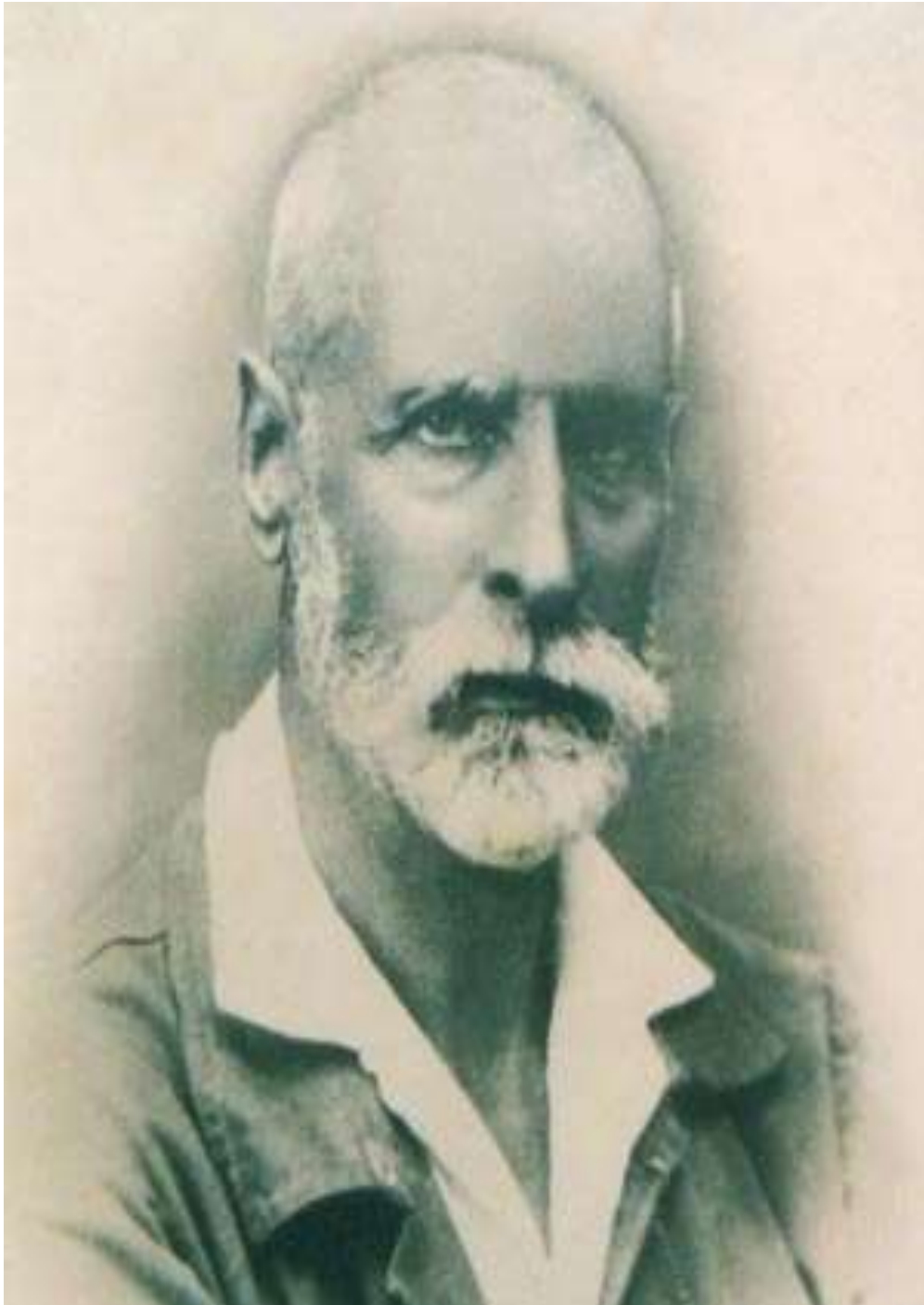
THE BLACK DEATH

THE 14TH CENTURY

Plague



Causative agent *Yersinia pestis*



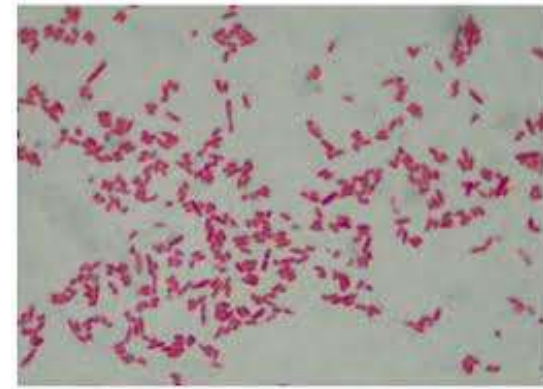
Alexander Yersin (1863-1943)

- Discovered the cause of the plague

- *Yersinia pestis* discovered in 1894 in Hong Kong

Yersinia pestis

Y. pestis is Gram- negative coccobacillus.



Virulence depends from a few factors:

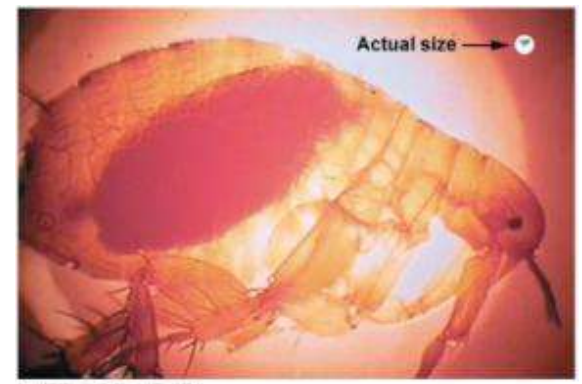
- ✓ antigen capsules FI and antiphagocytic role
- ✓ activator plasminogen degrades fibrin and facilitates expansion of bacteria
- ✓ V and W antigens have antiphagocytic property
- ✓ Yops proteins have antiphagocytic property and prevent efficient inflammatory response

Y. pestis products endotoxin(LPS),which causes most clinical manifestation of plague.

Plague -epidemiology-

Plague is a **zoonotic infection** of rodents; in urban areas the animal reservoir is usually rats, while in forested areas the disease can be transmitted by squirrels or mice.

Natural transmission between humans occurs when **infected rodent fleas feed on humans**



Xenopsylla cheopis

Pathogenesis:

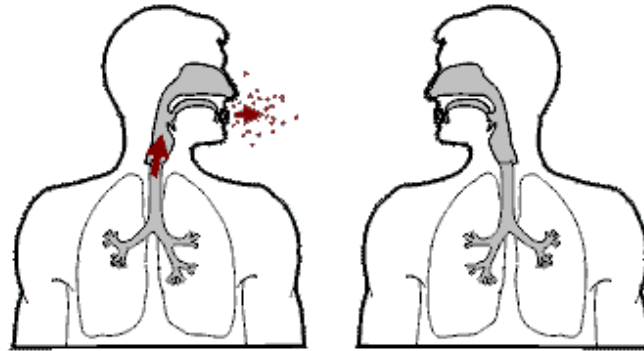
After the bite, the inoculated bacteria **migrate to the lymph nodes**, where they **multiply in mononuclear cells**. After an incubation period of 2 to 8 days from the flea bite, fever develops, and swollen, painful lymph nodes, called buboes, are the most important characteristic of **bubonic plague**.

Buboes are usually found in the groin, axillae, or cervical lymph nodes and are often so tender that they make it impossible to move the affected area of the body.



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... A biological attack with **Y. pestis** in aerosol form would most likely cause the primary pneumonic form of **plague without bubonic plague**.



Pneumonic plague can be primary or secondary.

Secondary pneumonic plague occurs due to hematogenous spread of *Y. pestis* in the case of untreated forms of bubonic or septicemic plague.

Primary pneumonic plague develops after direct inhalation of plague bacilli originating from other people or animals.

Bubonic plague



-enlarged lymph nodes in the armpit area-



-enlarged lymph nodes in the groin area

Septicemic plague is characterized by disseminated intravascular coagulation (DIC), necrosis of small blood vessels, and bleeding into the skin, which causes large purple or black lesions (purpura and ecchymoses).

- Gangrene of fingers and toes-

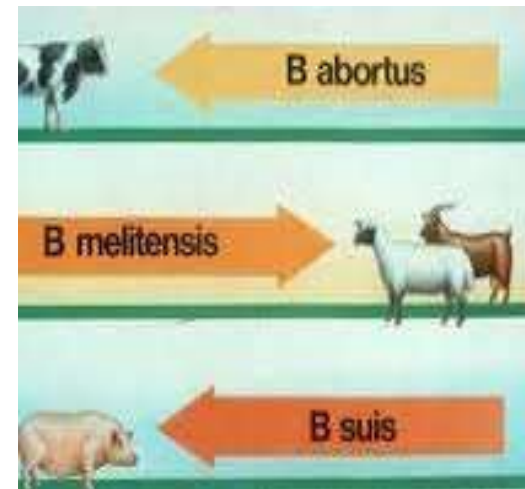
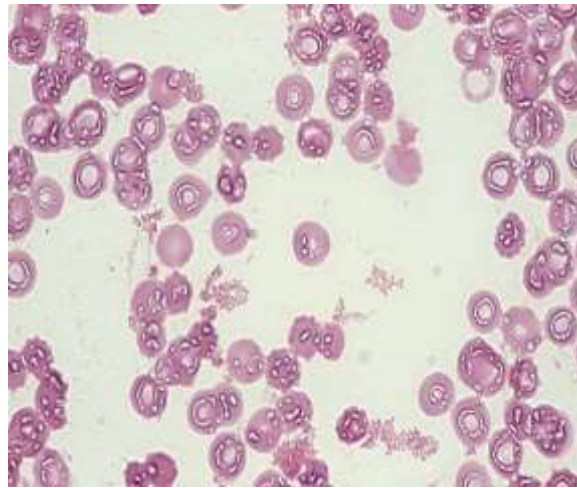
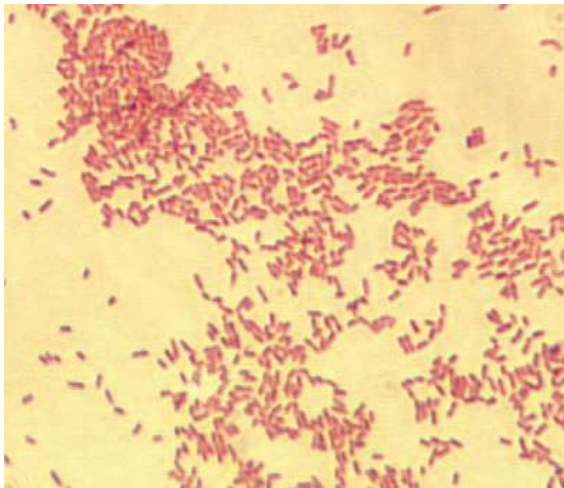
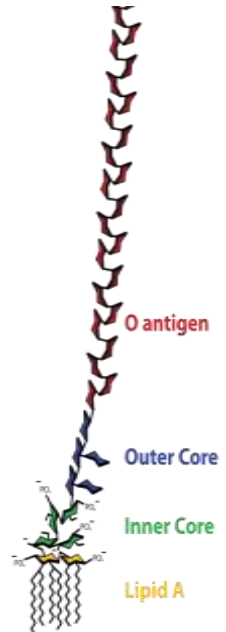
-hemorrhage on the skin-



Brucella species

–general characteristics–

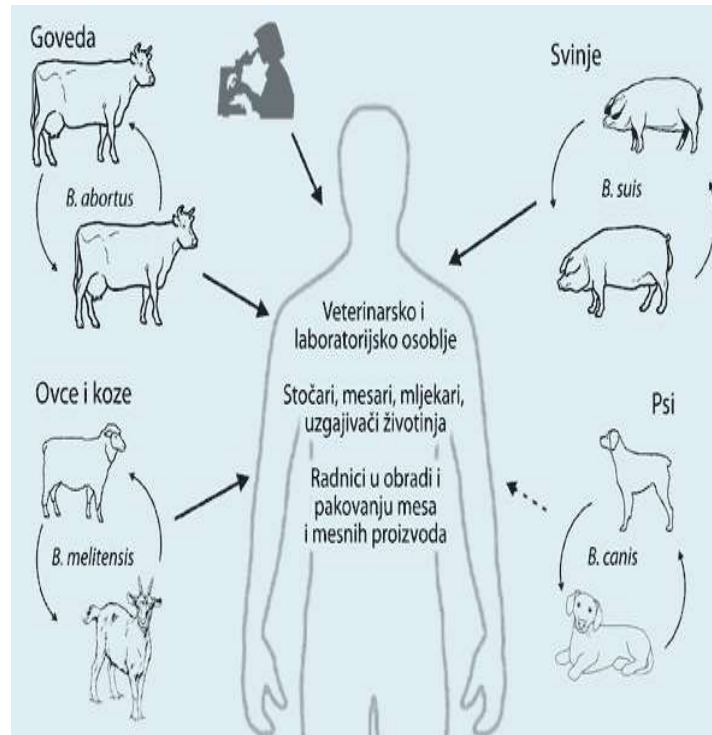
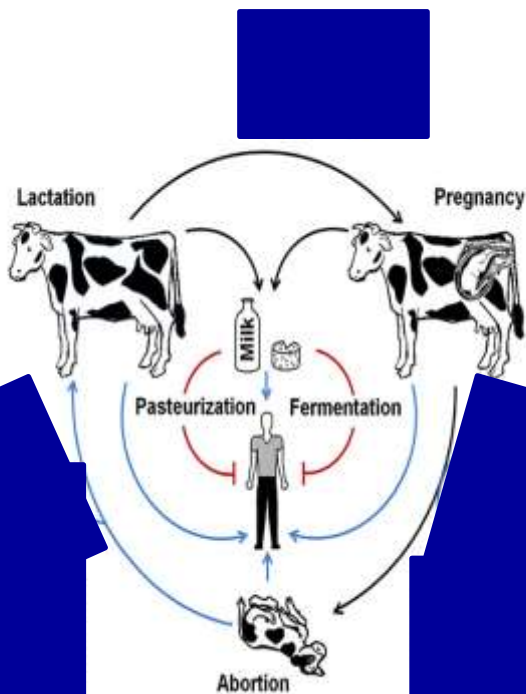
- *Brucella* are Gram-negative coccobacilli or bacilli
- It contains a **lipopolysaccharide** whose O-specific polysaccharide chain contains two major epitopes (A and M).
- The most important species: *B. abortus*, *B. melitensis*, *B. canis* and *B. suis*.
- Grows slowly under aerobic conditions



Brucellosis

–epidemiology–

- Brucellosis is **typical zoonosis**
- **In animals**, brucellosis is a genitourinary infection. Brucellae cause chronic infection of the mammary glands, uterus, and placenta.
- **In humans**, brucellosis is a chronic disease characterized by fever, night sweats, and weight loss over a period of weeks to months.

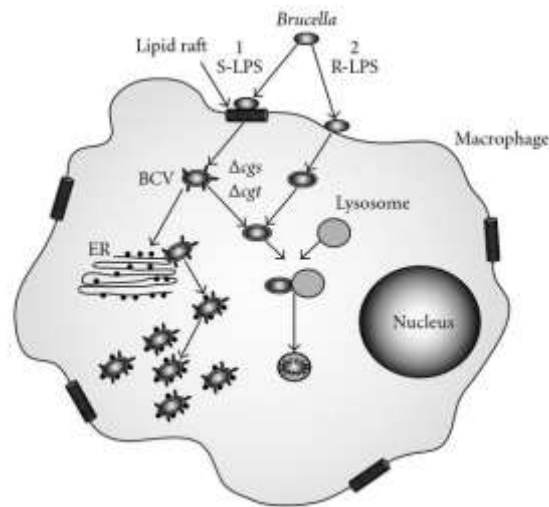




In animals, erythritol is present in the placenta and fetal membranes and significantly stimulates the growth of Brucella, which is why in pregnant animals the infection is localized precisely at this site and is accompanied by miscarriage. The human placenta does not contain erythritol.



Brucellae are facultative intracellular bacteria that infect epithelial cells and cells of the mononuclear phagocytic system...

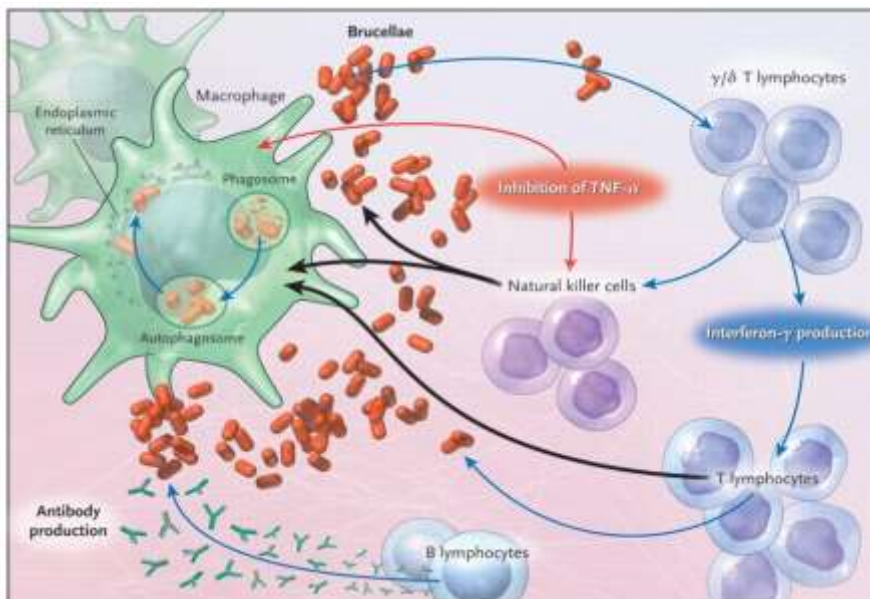
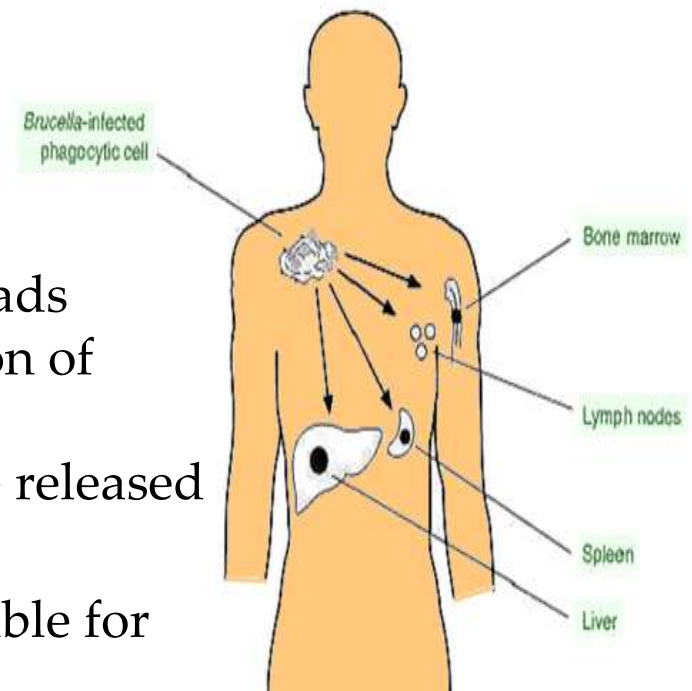


...They **survive within macrophages** by suppressing myeloperoxidase, inhibiting phagosome-lysosome fusion, and disrupting cytokine production.

Brucellosis

-pathogenesis-

- If the local infection is uncontrolled, it spreads further and is accompanied by the formation of **small granulomas** at the site of *Brucella* proliferation, from where these bacteria are released into the systemic circulation.
- Episodes of bacteremia are mainly responsible for **recurrent chills and fever**.

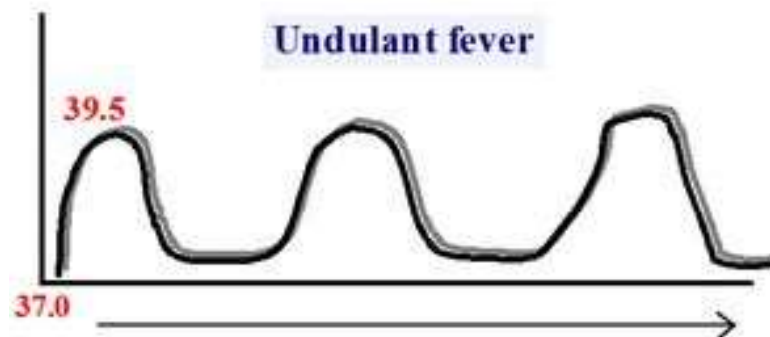


The T-cell immune response, especially helper T lymphocytes that produce TNF- α , TNF- β , IL-1 and IL-2, is responsible for controlling the infection and eliminating *Brucella* from macrophages.

Brucellosis

-clinical manifestations-

- Brucellosis (Bang's disease, Febris undulens - undulating fever, Malta fever, Mediterranean relapsing fever...)
- Periodic night fever (undulant fever) lasts for weeks, months, and even from 1 to 2 years. Sweating, lymphadenopathy and hepatosplenomegaly are present. In the case of prolonged brucellosis, weight loss also occurs
- Occasionally, localized infection develops in the lungs, bones, brain, heart or in the genitourinary system.



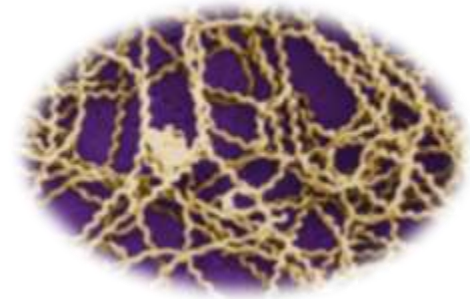
Prevention and therapy brucellosis

- ✓ Prevention involves **control and immunization of animals**: live, attenuated *B. abortus* and *B. melitensis* vaccines
- ✓ Prevention of laboratory infections
- ✓ Tetracyclines are primarily used in the treatment of brucellosis. In severe forms of the disease, streptomycin, gentamicin and rifampin are also used.



Leptospira species

-general characteristics-



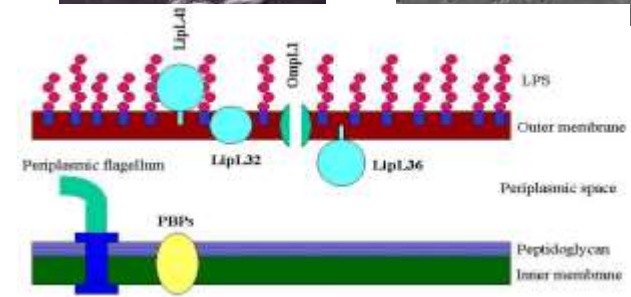
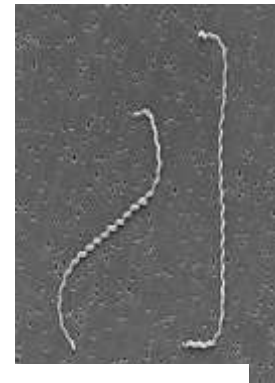
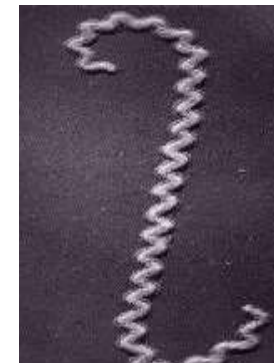
- Gram-negative spiral bacilli.

The genus *Leptospira* includes two species:

- ...*L. interrogans* (pathogenic species)
- *L. biflexa* (saprophytic species)

They are sensitive to...

- ... desiccation
- ... extreme pH values (e.g. gastric acid)
- ... the action of natural antibiotics
- ... low chlorine concentrations
- ... temperatures of 40-60°C.



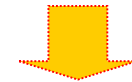
Leptospirosis -epidemiology-



zoonosis



pathogenic leptospires: renal tubules
of infected animals



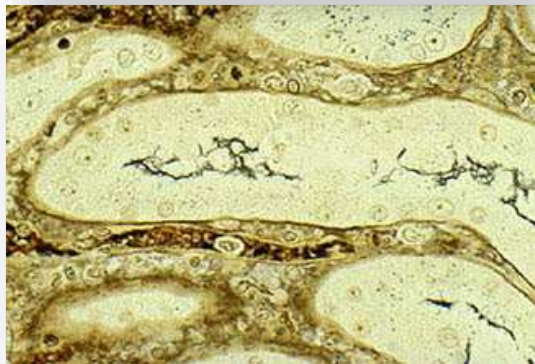
urinary excretion

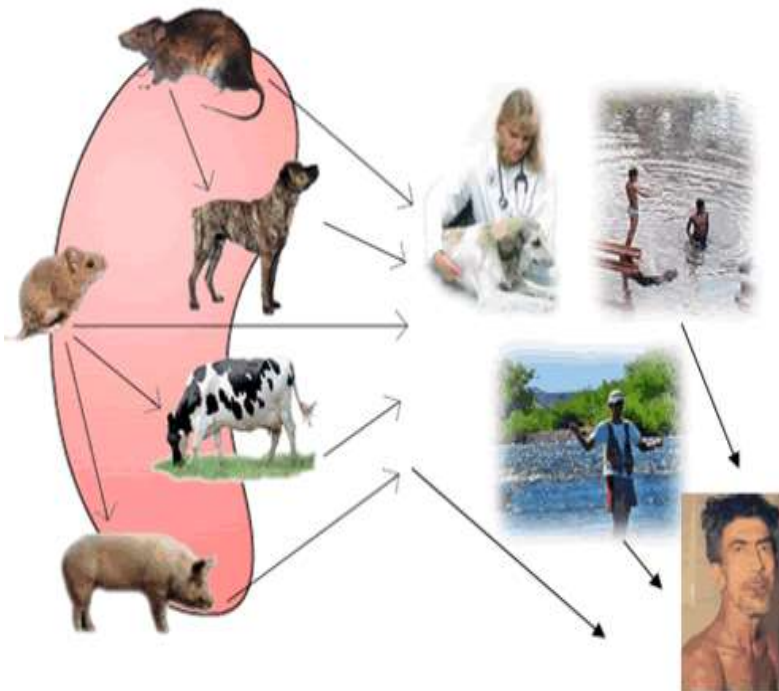


water and soil contamination



direct contact with urine or tissues of
infected animals, water
contaminated with urine of
infected animals

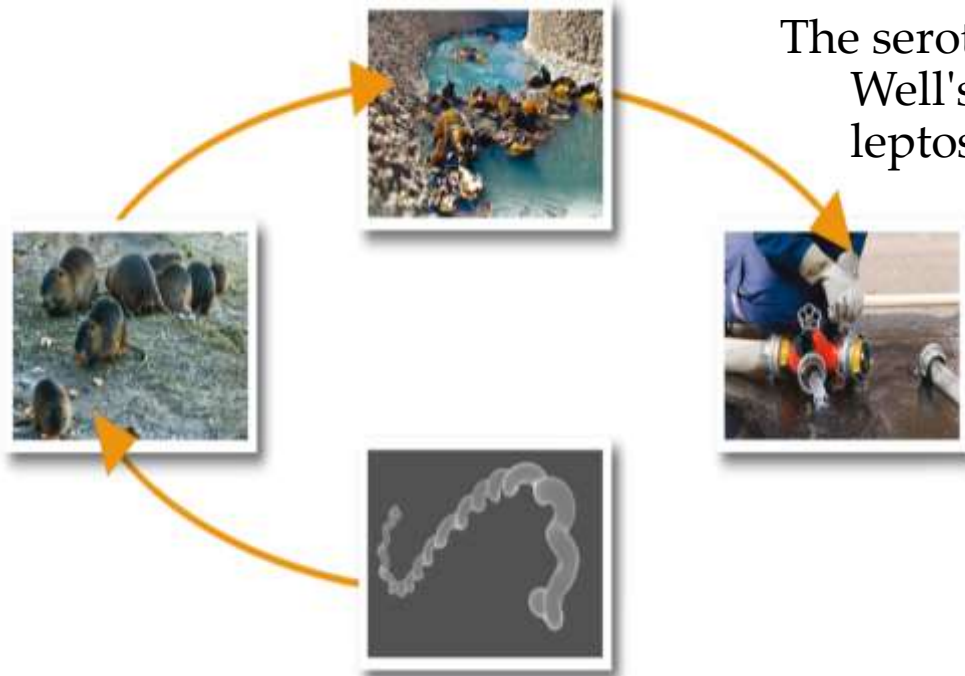




Leptospirosis is an occupational disease of butchers, veterinary surgeons, and animal breeders, as well as hunters and pet owners who have direct contact with infected animals.

Humans can also become infected by working in stagnant and canalized water contaminated with leptospira.

Interhuman transmission is extremely rare.



The serotype *L. icterohaemorrhagica* often causes Weil's disease, one of the most severe forms of leptospirosis.



Leptospirosis -pathogenesis-

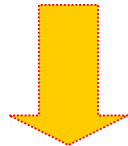
Leptospire enter the body through minor lesions on the skin, mucous membranes (conjunctiva)



hematogenous dissemination



Vasculitis and damage to the endothelium of small blood vessels



possible consequences: renal tubular necrosis, hepatocellular damage, meningitis, myositis, jaundice, bleeding



Leptospirosis

-clinical manifestations-

Leptospirosis can occur as a mild subclinical infection. Mild forms of leptospirosis are described as anicteric leptospirosis.

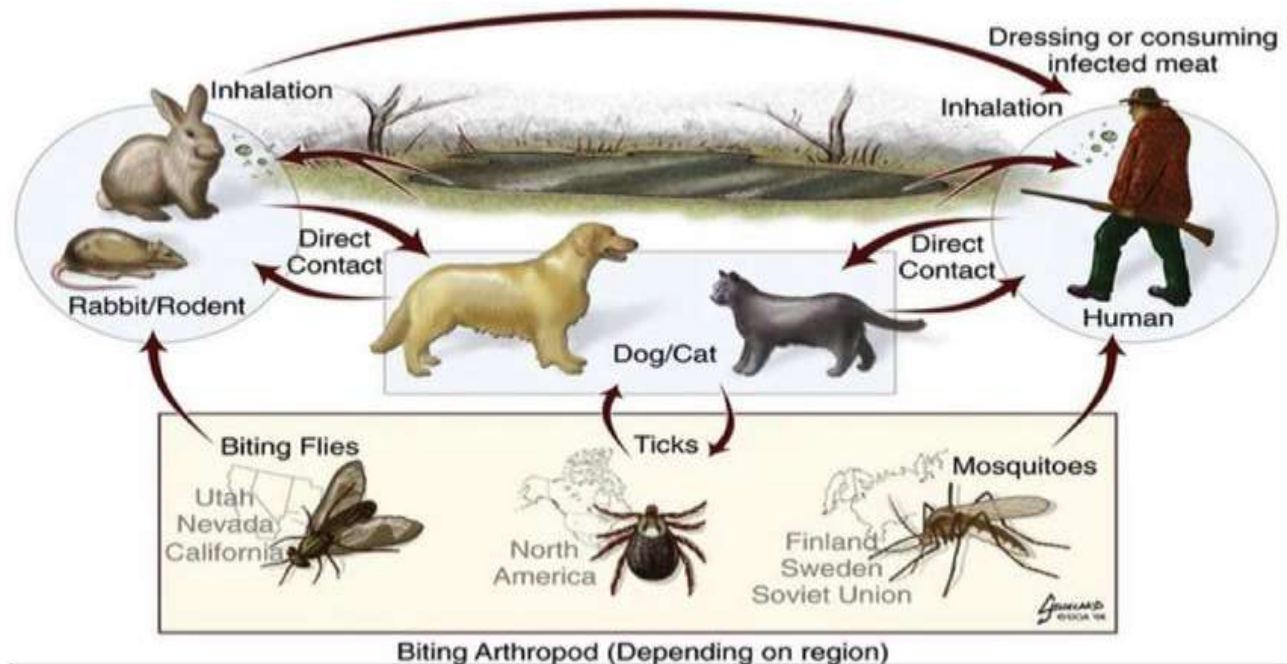


Severe forms are described as icteric leptospirosis. There is a bacteremia phase and an immune phase characterized by antibody production and temporary disappearance of leptospires from the blood.

Severe icteric form of the disease (Weil's disease)

Francisella tularensis

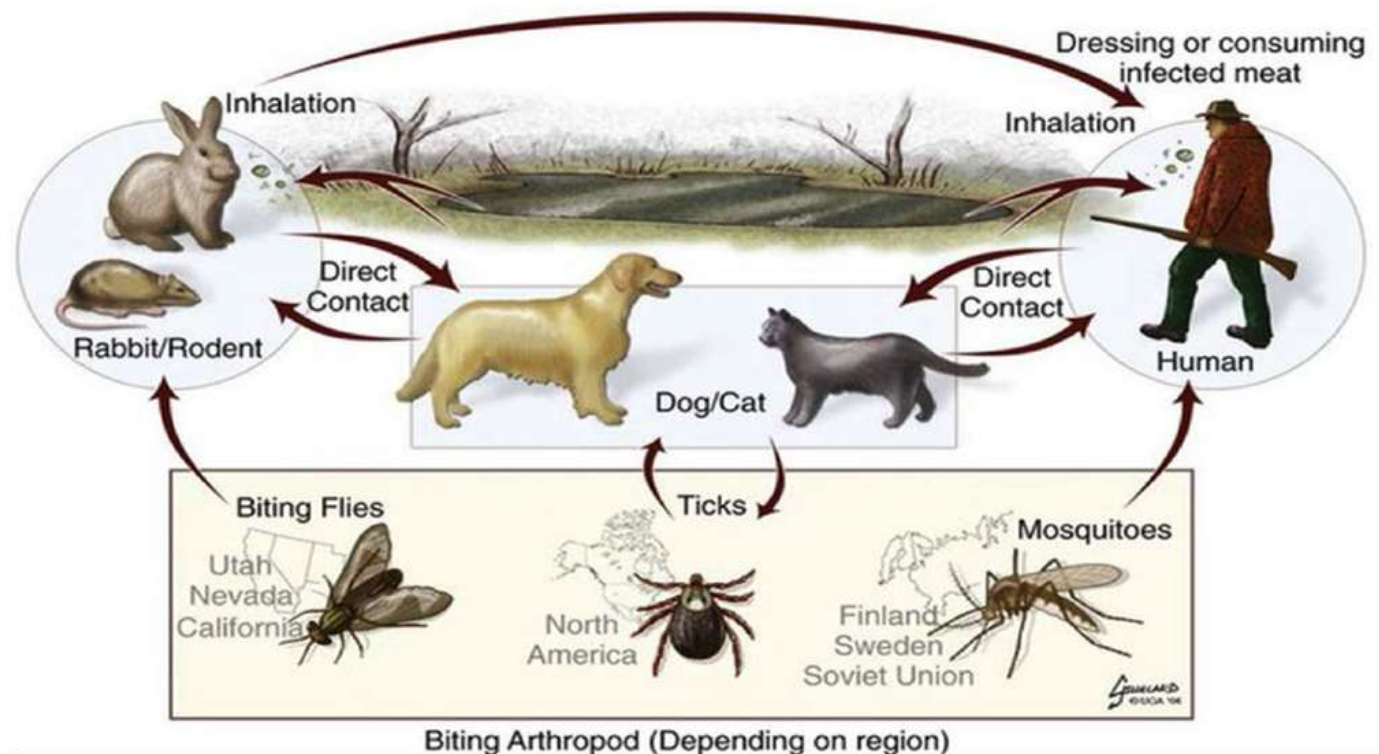
- gram-negative bacteria
- throughout North America and Eurasia.
- **Tularemia**
- Animal **reservoir**- rabbits, squirrels.
- **transmission of infection** among these animals – bite tick, fly and mosquitoes.



Francisella tularensis

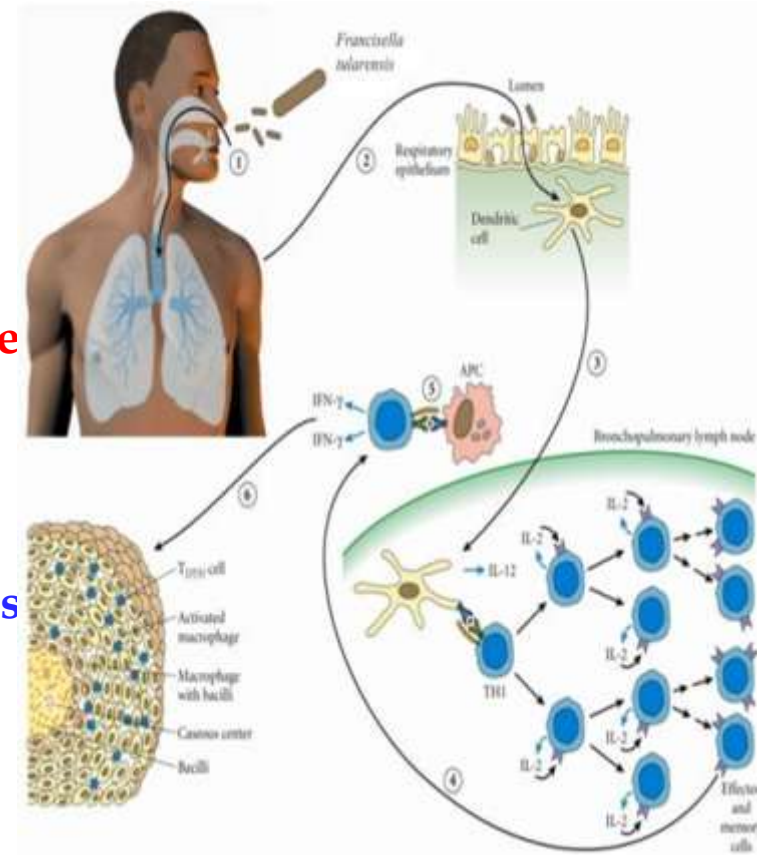
People get infected:

- arthropod bite
- direct contact with infected animal tissue,
- contaminated food and water
- by inhalation of aerosols



Francisella tularensis

- intracellular pathogen
- Infects **macrophages**, **hepatocytes** and **epithelioid cells**.
- Virulence factors include **antiphagocytic capsule** and
- Enzyme **citrulline ureidase**.
- **LPS** endotoxin, but it is **activity a thousand times weaker** from activity of LPS of *Escherichia coli*.
- **Cellular immune response** is the main host response to infection -**granulomatous infiltrate** in lymph nodes, liver, lungs, spleen and bone marrow.
- Tularemia can be **misdiagnosed** as tuberculosis if the granulomas are caseous.



Francisella tularensis

- Tularemia also has a history **as an agent of bioterrorism**.
- The Japanese studied the disease in their biological weapons program in Manchuria (1932 to 1945), and the Russian Biopreparat program developed aerosolized forms. *F. tularensis* are engineered to be resistant to antibiotics and vaccines.
- A bioterrorist attack would most likely involve aerosolized *F. Tularensis*
- One or two organisms are enough to cause infection.
- The mortality rate from untreated pneumonic or septic tularemia is as high as 60%.
- With treatment, the mortality rate is less than 2%.

Francisella tularensis

- **Diagnosis** (bioterrorism) is difficult to establish – non-specific clinical picture
- Lymphadenopathy - a hallmark of naturally occurring tularemia
- standard microbiological testing of sputum
- **Treatment**- pneumonic, septic or typhoid tularemia, the drugs of choice are aminoglycosides.
- Ciprofloxacin and doxycycline are also effective and would be preferable in mass casualty settings.
- **Live, attenuated vaccine** is available to laboratory workers exposed to tularemia;
- In volunteer studies, the vaccine did not protect all subjects from aerosolized challenge.



Cat scratch disease and bacillary angiomatosis

Causative agent *Bartonella
henselae*

Bartonella species

-general characteristics-

Species of the genus *Bartonella* – cause severe infections in immunodeficient and immunocompetent individuals with different consequences depending on the host's defenses.

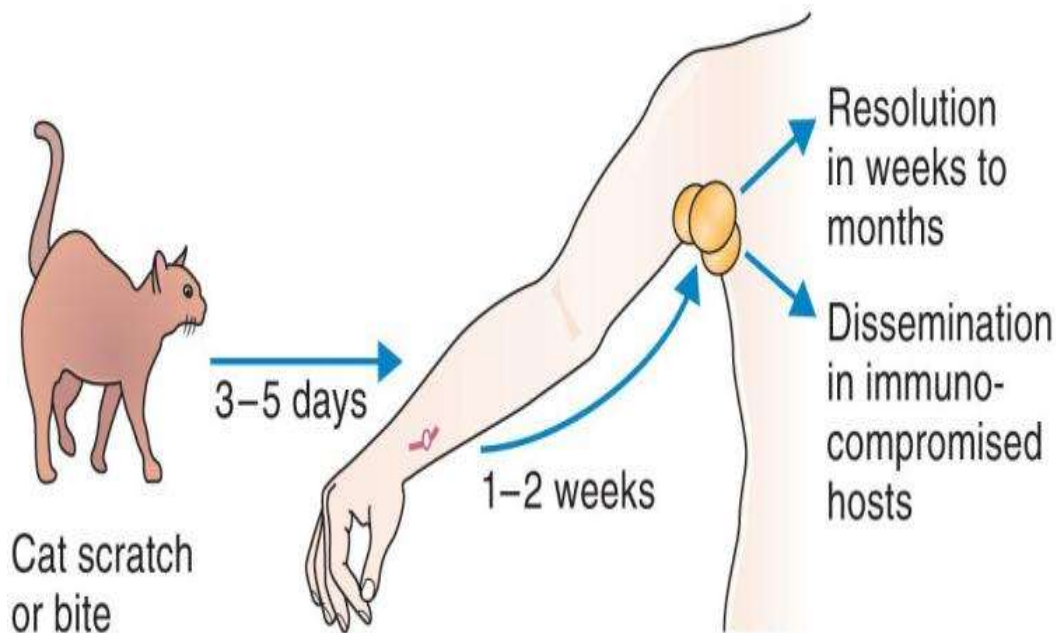
B. henselae – the causative agent of two diseases...

...**cat scratch disease**, a self-limiting infectious lymphadenitis transmitted by a scratch or bite from an apparently healthy but infected cat

...**bacillary angiomatosis**, cutaneous and visceral tumor-like proliferations of blood vessels, primarily in AIDS patients..

Cat scratch -pathogenesis-

B. henselae causes a small papule at the site of inoculation. It then spreads through the lymphatic vessels to the draining lymph nodes, where the bacteria multiply and a strong mixed granulomatous and suppurative reaction occurs.



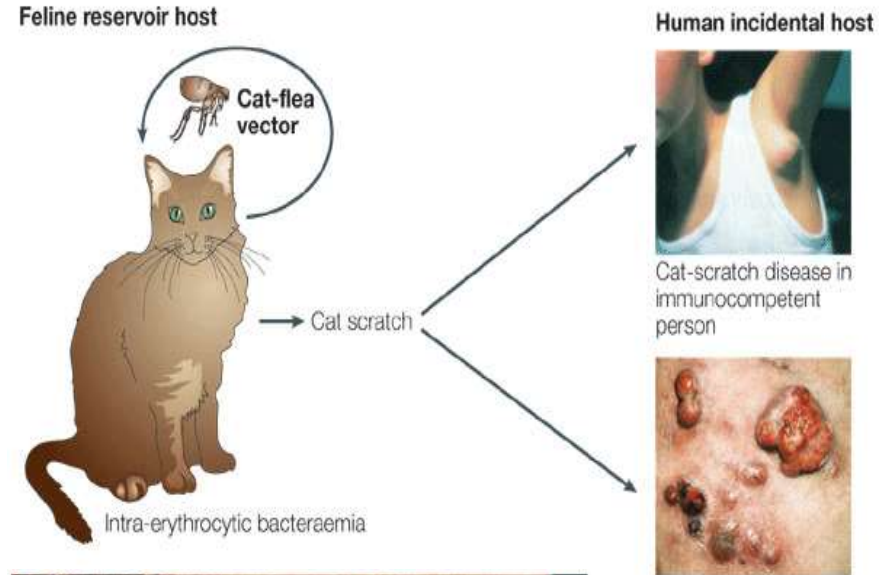
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Bacylar angiomatosis -pathogenesis-

In some patients, the host response is not sufficient to control the infection. The microorganisms **replicate freely and disseminate in large numbers** through the bloodstream, which often manifests clinically as sepsis or **localized infection** of the skin, liver, and other internal organs.

The bacteria **produce substances** that **stimulate cytokine production**, which results in the **proliferation of small blood vessels** and, consequently, the **lesions** of bacillary angiomatosis



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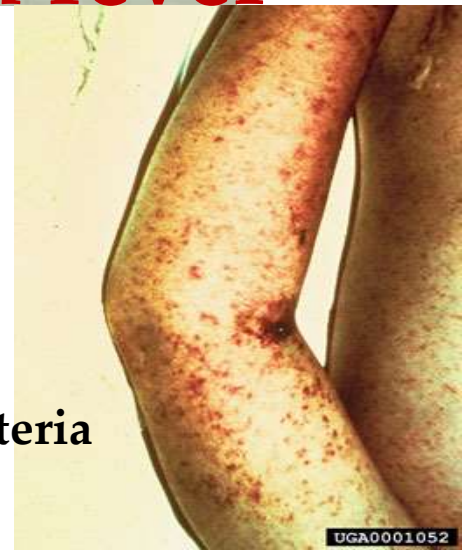
HUNGER TYPHUS IN RUSSIA—THE INTERIOR OF A FEVER HOSPITAL AT MOSCOW

Rickettsia species

Causative agent of **spotted fever**



Rickettsiae are obligate intracellular bacteria

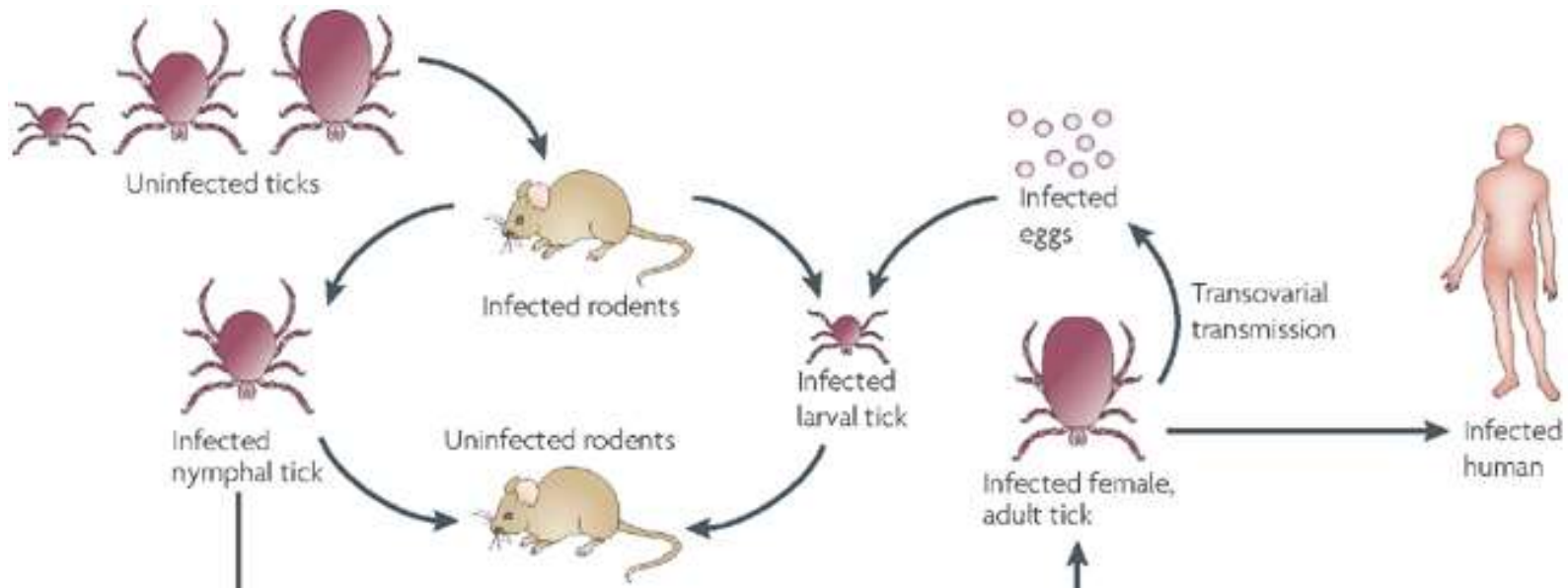


Rocky Mountain Spotted Typhus

- *Rickettsia rickettsii*-

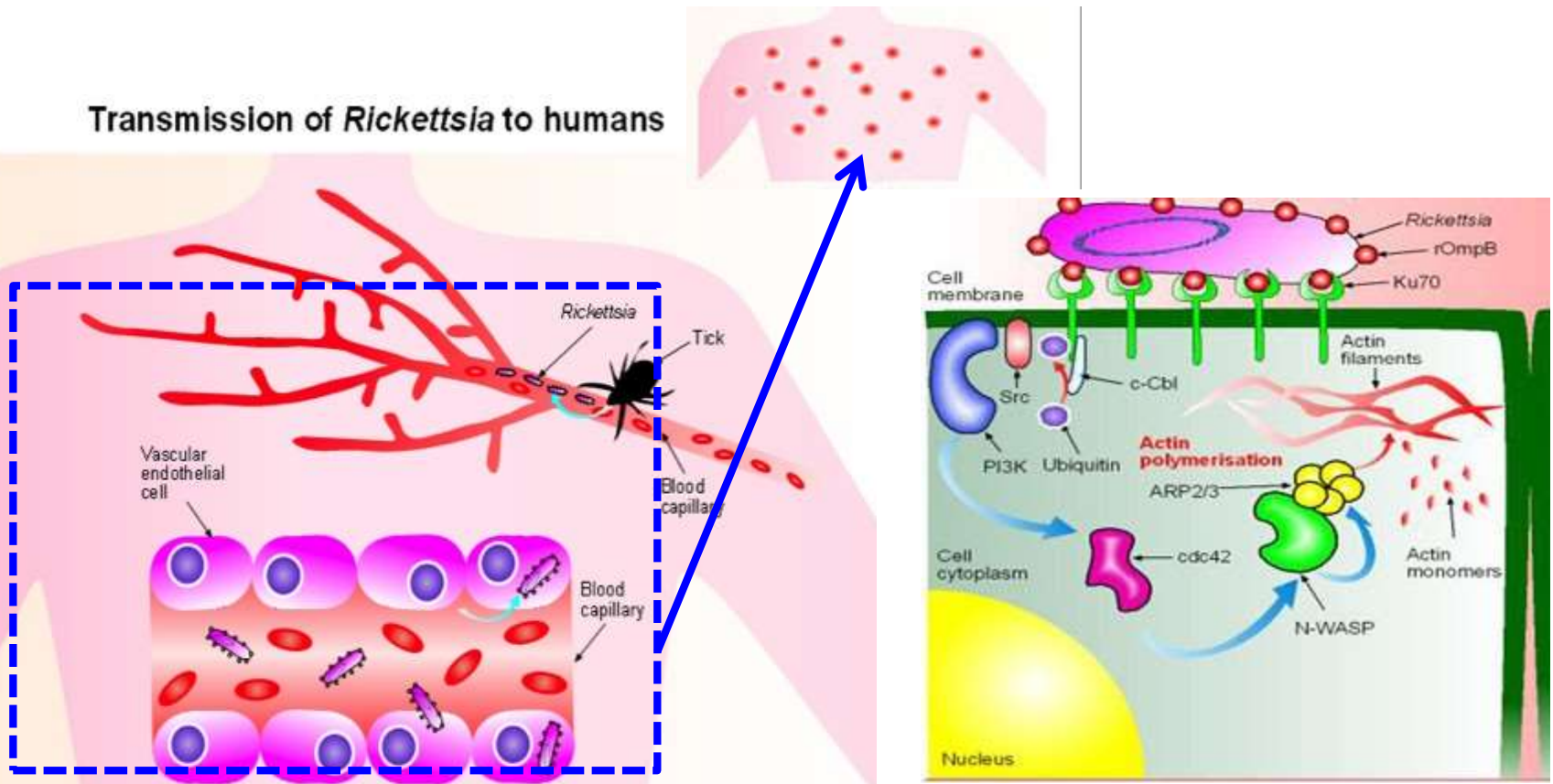


Rocky Mountain spotted fever is caused by *R. rickettsii*. It is transmitted from tick to tick transovarially and usually causes minor lesions in the tick. **Ticks** (species **Dermacentor**) are involved in transmission.



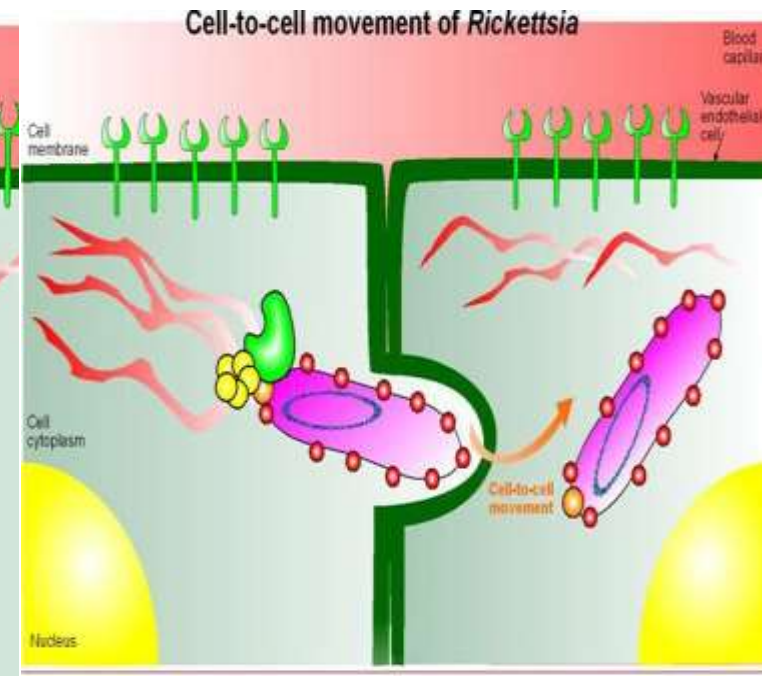
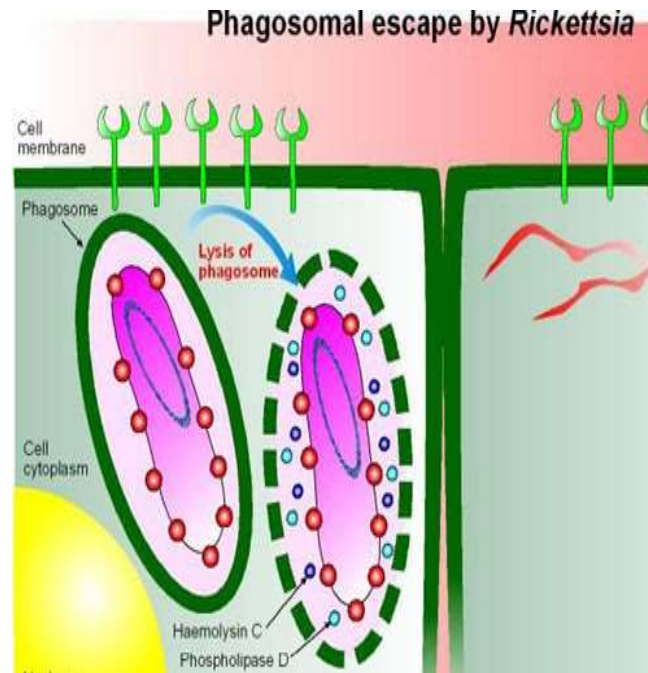
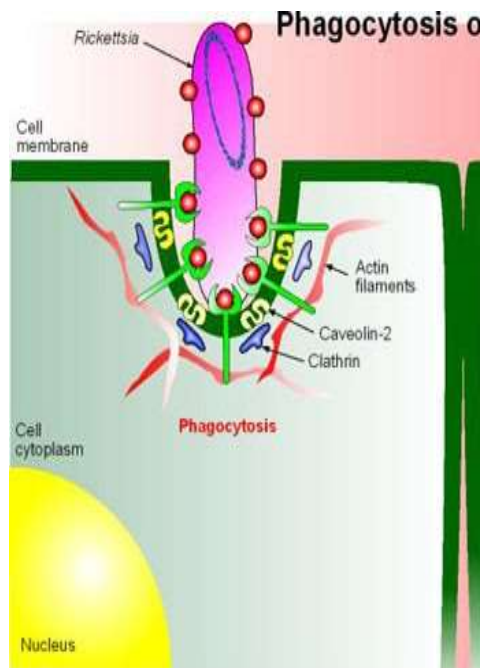
Rickettsiae **spread** through the **blood** throughout the body.
They bind to **vascular endothelial cells** and induce their
own endocytosis into these cells...

Transmission of *Rickettsia* to humans



... Inside cells, rickettsiae rapidly escape from the phagosome into the cytoplasm by lysing the phagosome membrane with phagolipase...

...*R. rickettsii* replicate **in the cytoplasm**, and **move like a propeller** through the cytoplasm by inducing polymerization of host cell actin at one pole of the bacteria. The bacteria then spread to other endothelial cells via long extensions of the cell membrane.



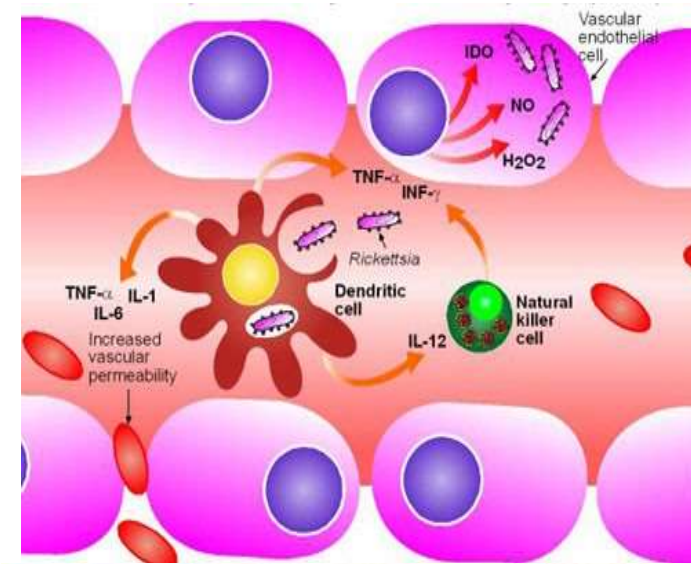
Rocky Mountain spotted fever

-tissue damage-

The target of rickettsia is the **endothelial cell** membrane and the mechanism of damage to this structure is the result of the action of free radicals induced by lipid peroxidation of the membrane.

The effects of endothelial cell damage are visible on the skin, where a **pink rash** first appears due to dilation of the blood vessels. The release of erythrocytes results in pinpoint hemorrhages (**petechiae**, which is why the disease got its name.

In the blood vessels of the brain, lungs, heart, liver, as well as other internal organs, these pinpoint hemorrhages cause encephalitis, pneumonitis, cardiac arrhythmia, nausea, vomiting, and abdominal pain.



Epidemic typhus



Epidemic typhus, a classical rickettsiosis, was one of the most significant infectious diseases in history, influencing the outcome of most European wars. During and immediately after World War II and the Russian Revolution, 30 million people fell ill with typhus, of whom 3 million died.

The causative agent of epidemic typhus is *Rickettsia prowazekii* and is transmitted by **body lice**, with the causative agent being deposited in the feces of the louse on human skin.

With the discovery of effective insecticides, transmission of epidemic typhus was interrupted and the incidence of this disease was greatly reduced.



FIG. 2. TYPHUS VICTIMS AWAITING BURIAL

Epidemic typhus (Spotted typhus)

-*R. prowazekii* -



Pediculosis human body

Epidemic typhus - is transmitted between people by **body fluids**.

- ... Epidemics begin with a single case of **late relapse of typhus (Brill-Zinsser disease)**, a reactivation of *R. prowazekii*. The causative agent may remain latent for years or even decades after recovery from an epidemic of spotted typhus or zoonotic typhus transmitted by fleas from flying squirrels...
- ... The disease is associated with overcrowding, poverty, and poor hygiene.

Murine typhus

- *R.typhoid*-



A more common and widespread rickettsiosis is murine typhus. This endemic disease is caused by **R. typhi**, which is transmitted in a **natural cycle between rats and rat fleas**. Humans are infected by the deposition of feces of infected fleas on the skin.

Murine typhus occurs in tropical and subtropical regions.

Coxiella burnetii

- Q fever is an abbreviation for "Query fever" and this name refers to the unknown etiology when the disease was first described
- *C. burnetii*, stands out from other rickettsiae, and the disease also differs from other rickettsioses in clinical manifestations, pathological lesions, and epidemiology.
- **significantly more resistant** to numerous harmful effects of the external environment compared to other rickettsiae
- formation of **structures resembling spores** that are observed by electron microscopy



Coxiella burnetii

- does not require protection from a live vector, but is **transmitted by aerosol**
- typical zoonosis
- **main reservoirs** are infected sheep, cattle, cats, rabbits and other animals
- milk, urine, feces, placenta
- The disease most commonly occurs **in sheep farmers, veterinarians...**

Coxiella burnetii

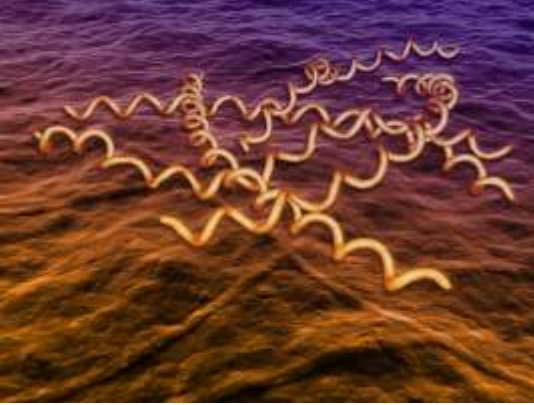
- Q fever differs significantly from typhus and spotted fever in that it has **acute form**,
- Asymptomatic or is associated with nonspecific febrile illness, atypical pneumonia, or granulomatous hepatitis
- **chronic form**-usually affected heart valves.
- Grows **in macrophages** in the lungs, liver, bone marrow, and spleen, survives in the phagolysosome and stimulates granuloma formation.
- Diagnostic method - Elisa test

Spiral bacteria



Treponema pallidum

Borrelia burgdorferi



Treponema pallidum

Causative agent of **syphilis**
(**lues**)



Fritz Schaudinn

*The causative agent of syphilis was
discovered in 1905.
(Fritz Schaudinn)*

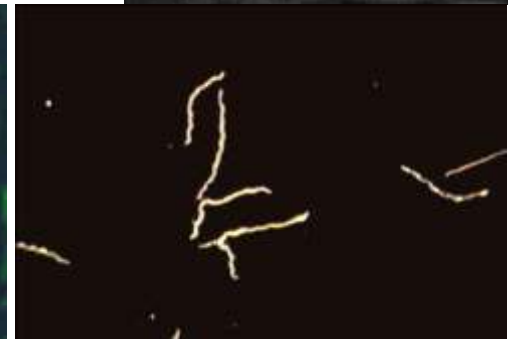
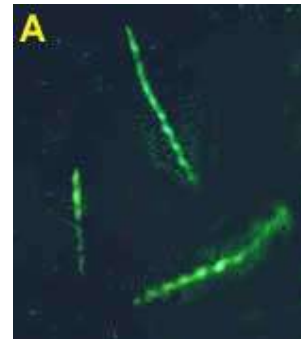
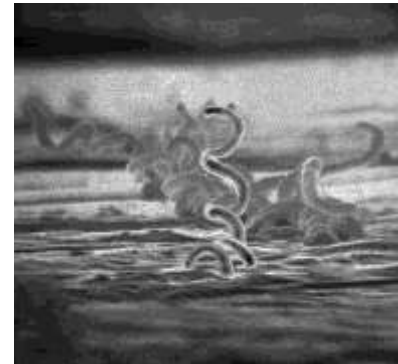


Treponema pallidum

–general and epidemiological characteristics–

Treponema pallidum is **Gram-** negative and **thin** spirochete ...

... The flagella are within the periplasm, resulting in the characteristic movement of this spirochete.



It cannot be grown on artificial media and does not synthesize toxins.

It is sensitive to drying, disinfectants and heat.

T. pallidum is transmitted exclusively by **direct contact**, and the two main routes of transmission are **sexual and transplacental**.

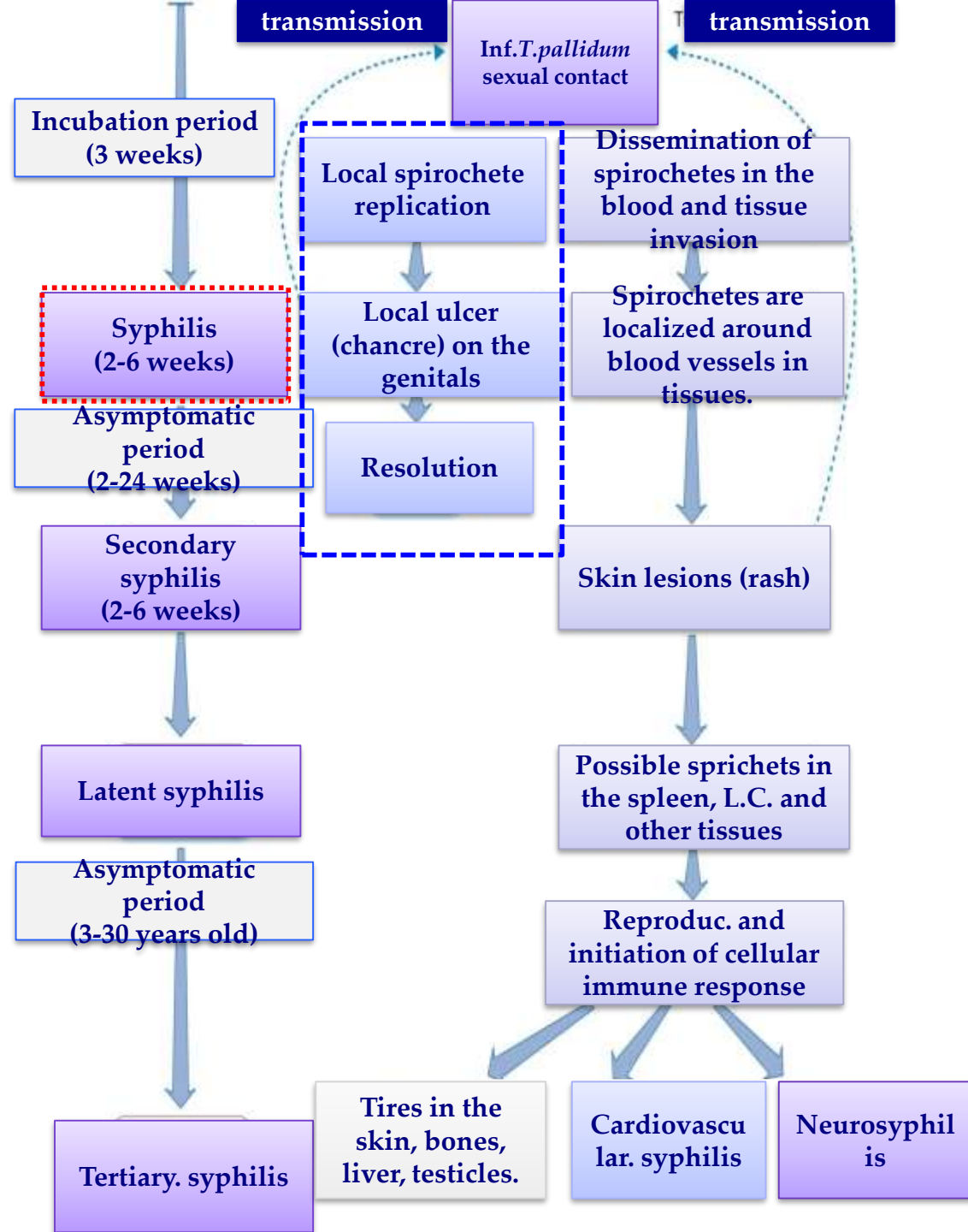
Tissue damage and disease

1. Primary syphilis

A **hard chancre (Ulcus durum)** forms at the site of treponema entry.

The basis of a hard chancre is **endarteritis**. The endothelium of small arterioles proliferates and obstructs blood flow, causing necrotic ulcerations. A granulomatous infiltration develops around the arterioles, i.e. periarteritis.

A hard chancre is **painless**.



Primary syphilis



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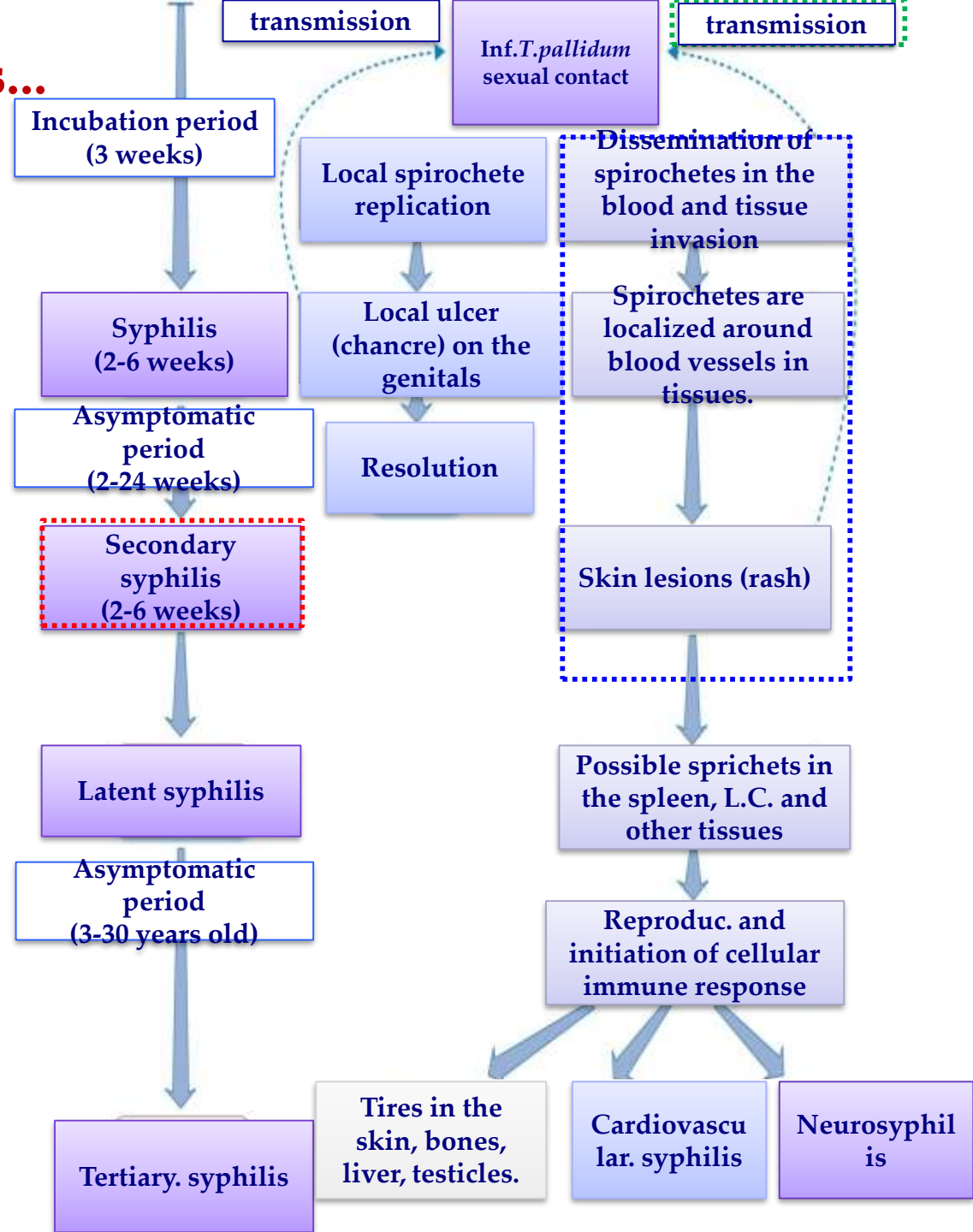


Ulcus durum

2.Secondary syphilis...

... is the result of **systemic spread of infection**, when treponemas disseminate via the blood to the liver, joints, muscles, as well as to the skin and mucous membranes at sites distant from the primary chancre.

Mucocutaneous rash and other manifestations of secondary syphilis resolve within weeks to months.



Secondary syphilis



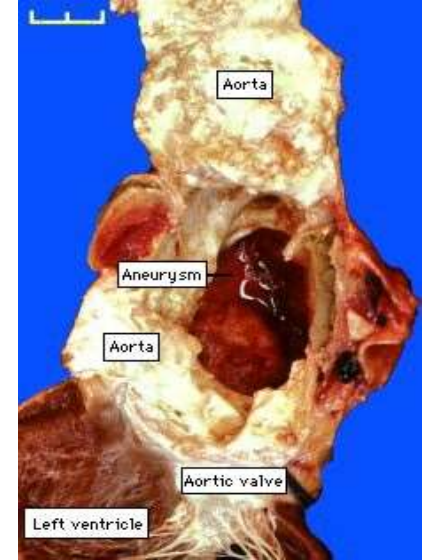
Tertiary syphilis



The hallmark of tertiary syphilis is **tissue destruction caused by an immune response**, manifested by the development of **gummas**, or granulomatous lesions that affect the bones and mucocutaneous tissues. Gummas are **not infectious** and are not a direct result of treponema, but rather a **delayed hypersensitivity reaction**.

In **cardiovascular syphilis**, vasculitis occurs that affects those arteries that flow into the aorta.

The destruction of elastic tissue in the media of the **aorta causes dilatation of the wall** and **aortic valve insufficiency**, or the **formation of an aortic aneurysm** with subsequent **aortic rupture**.



The clinical findings of **neurosyphilis** can be discrete. Involvement of the dorsal parts of the spinal cord induces **tabes dorsalis**, which manifests as a staggering or **ataxic gait**.



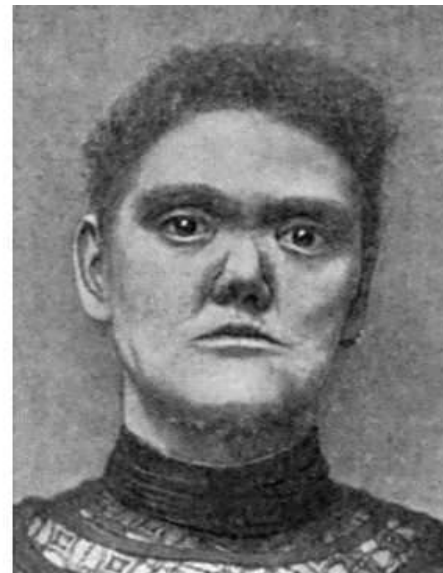
The physical sign of neurosyphilis is the **Argyll Robertson pupil**



Fig. 9.38 Argyll Robertson pupil. The patient's right pupil was small, slightly irregular and fixed to light though reacting to a near stimulus. The left eye is artificial.

4. Congenital syphilis

The most common manifestations of congenital syphilis become visible during the first 2 years of life, and these are facial and dental deformities. Other less common manifestations are deafness, arthritis, and "saber" bones.



" One night with Venus, the rest of your life with Mercury "

Syphilis therapy

The drug of choice is **penicillin**.

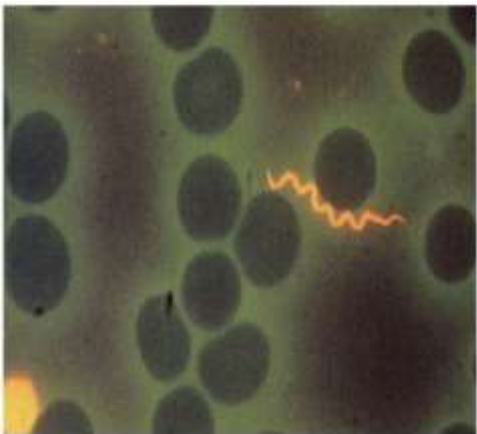
Patients with primary and secondary syphilis are treated with penicillin injections, while patients with late latency or tertiary syphilis are treated with penicillin injections but with repeated treatment. Neurosyphilis should be treated with high-dose penicillin, intravenously, for 10 days



Lyme disease

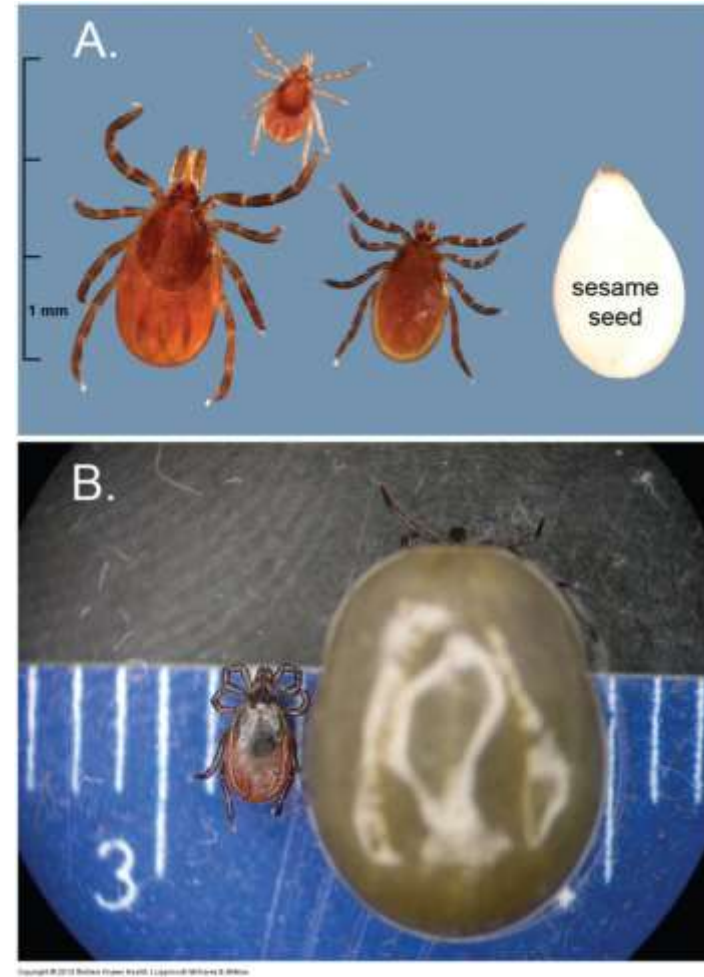
ADAM

Borrelia Burgdorferi



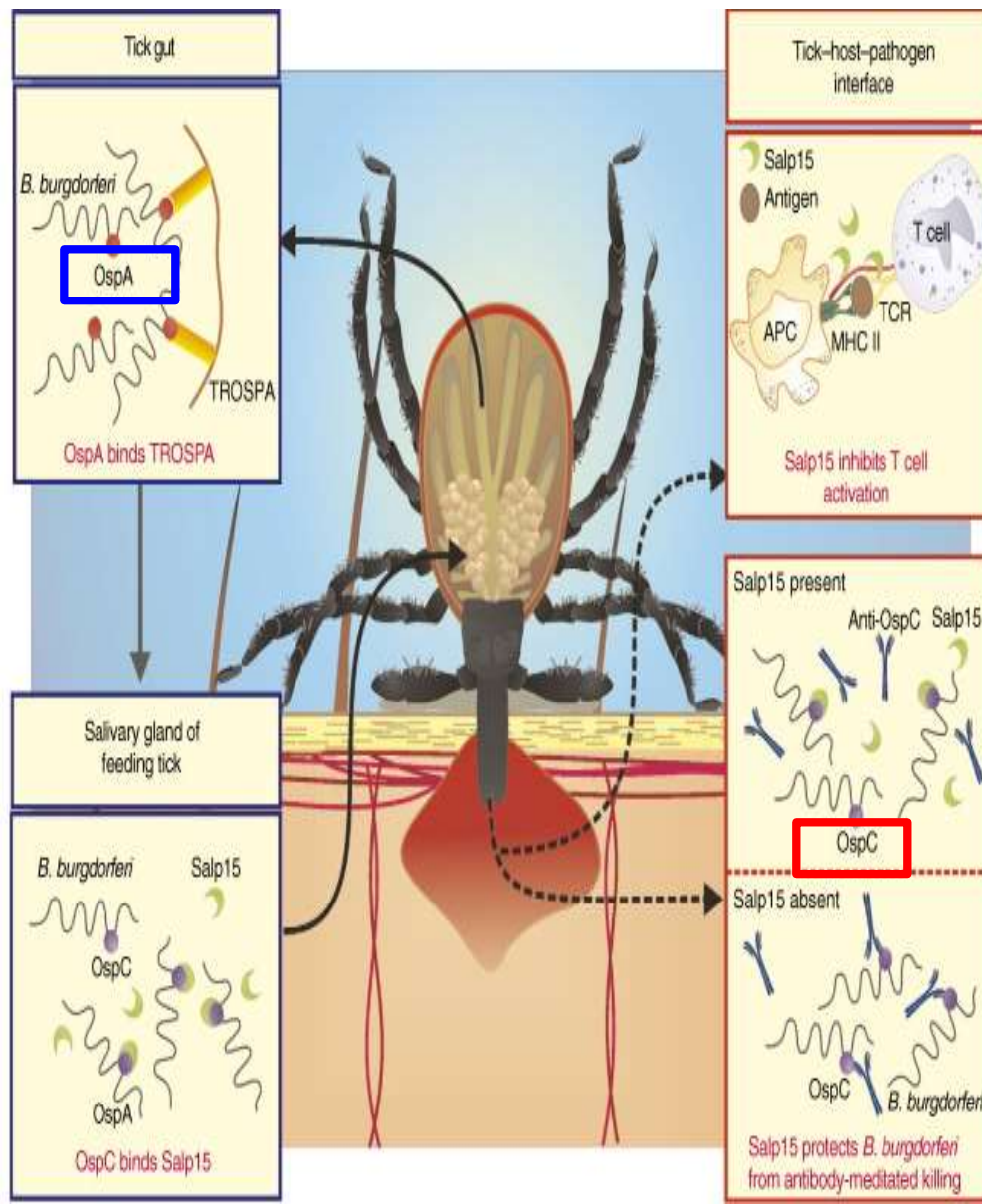
Causative agent of **Lyme disease**

Humans are infected with *B. burgdorferi* through **vectors, ticks (*Ixodes* sp.)**



In Europe, Lyme disease is caused by: *B. burgdorferi*, *B. garinii* and *B. afzelii*.

Entry of *B. Burgdorferi*

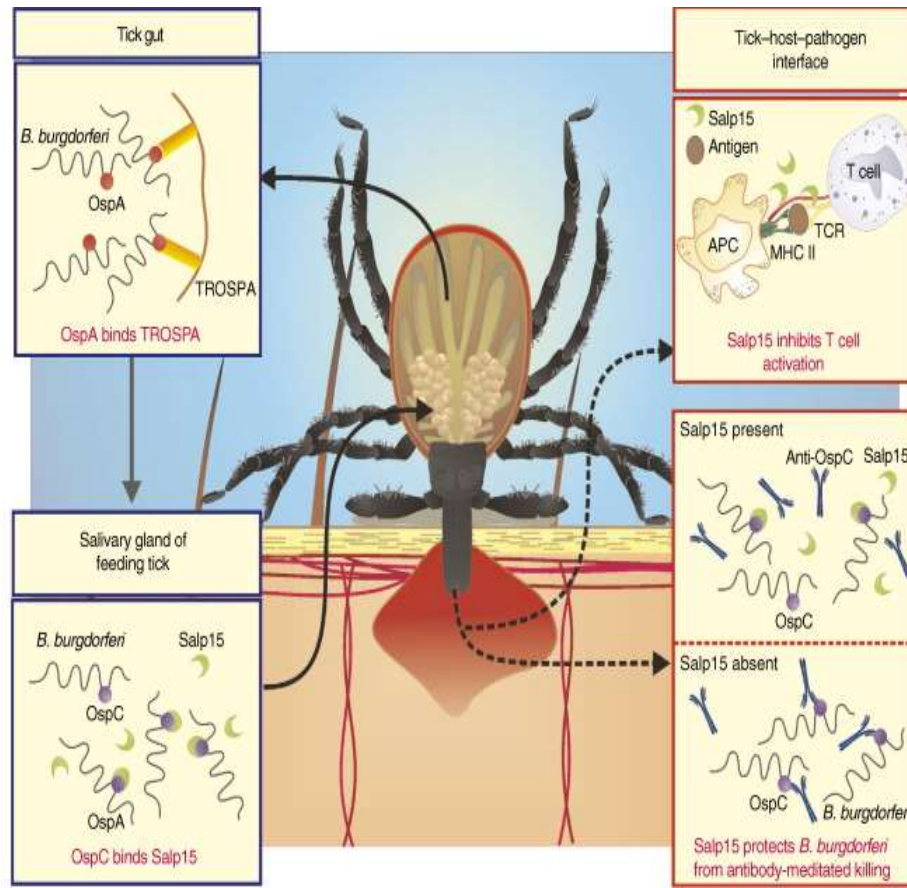


In the Ixodes tick, between feeding periods, the spirochetes are attached to epithelial cells and are dormant. They express a major surface protein (**OspA**)..

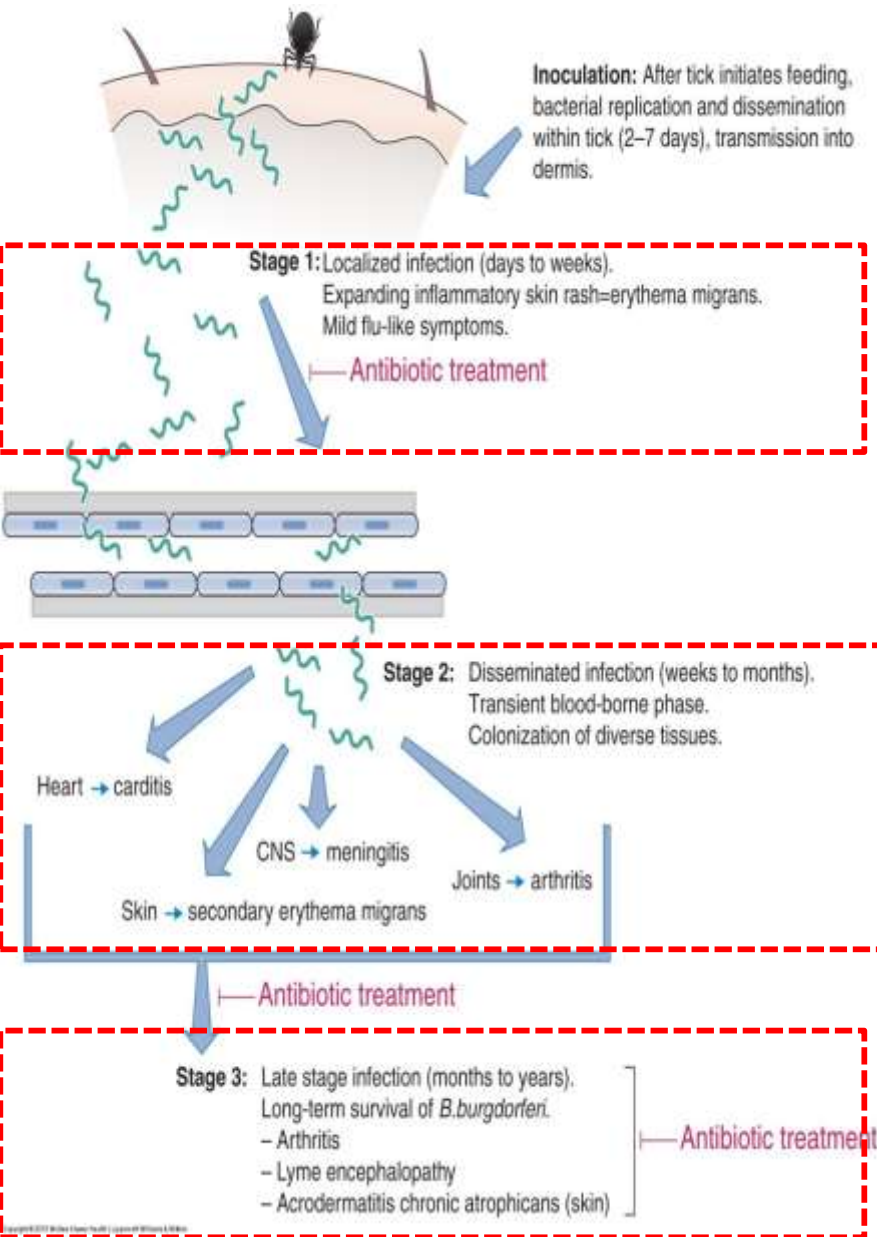
...when the tick feeds on a mammal, the spirochetes are exposed to higher temperatures and blood components. The spirochetes undergo phenotypic changes over the next 2 or 3 days. **OspA expression decreases**, while **OspC expression increases**, which is necessary for **successful establishment of infection in mammals**.

The transition from OspA to OspS is essential.

The transmission of *B. burgdorferi* from ticks to mammals is also facilitated by **increased expression of some genes that encode proteins with immunosuppressive effects.**



Spread and reproduction of *B. burgdorferi*



B. burgdorferi multiplies and establishes an infection in the skin, causing the development of a skin lesion, **erythema migrans**, which is the hallmark of **localized infection or stage 1 Lyme disease**.

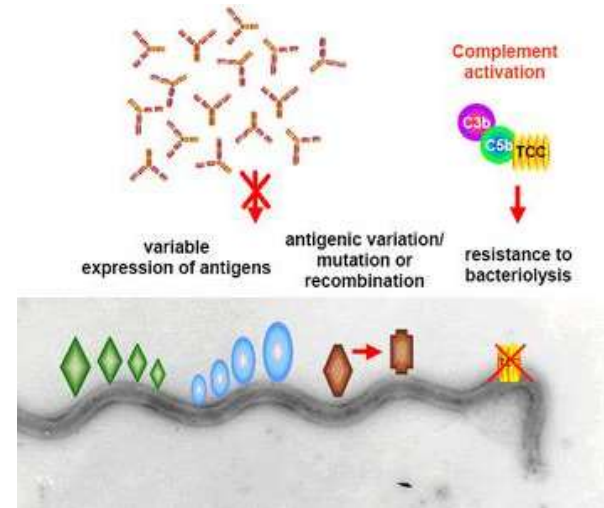
Some strains multiply further and spread to multiple tissues (nervous system, muscle tissue, and heart). This phase is known as **early disseminated infection or stage 2 Lyme disease**.

In the late stage, or stage 3 Lyme disease, the rate of bacterial multiplication is significantly reduced or is controlled by the host's defenses, so that very few bacteria are present in the tissues.

B.Burgdorferi evades the immune system....

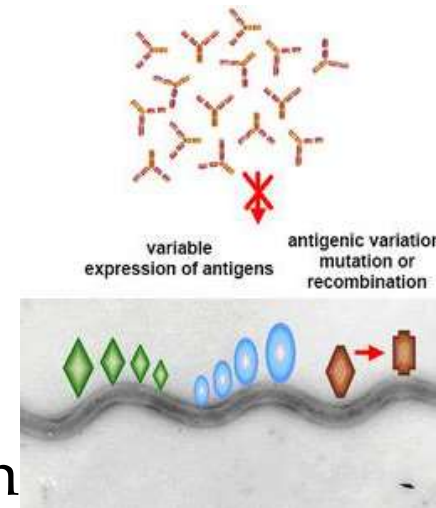
... ..bind to **regulatory proteins** and thus become resistant to complement-mediated lysis and opsonization.

.



...turns on and off the **expression of many genes** encoding **surface proteins** or changes its surface structure ("mobile" targets) and thus evades the immune system.

...On the eighth day after infection, the **reduction of gene expression for the OspC molecule** is essential for the persistence of *B. burgdorferi* (the **production of anti-OspC antibodies decreases**)...



.... simultaneously with the decrease in **production** of the surface protein **VlsE** increases. VlsE **undergoes constant antigenic variation** during active infection, which is a mechanism for evading the immune response (theoretically up to 10^{30} protein variants can be generated).

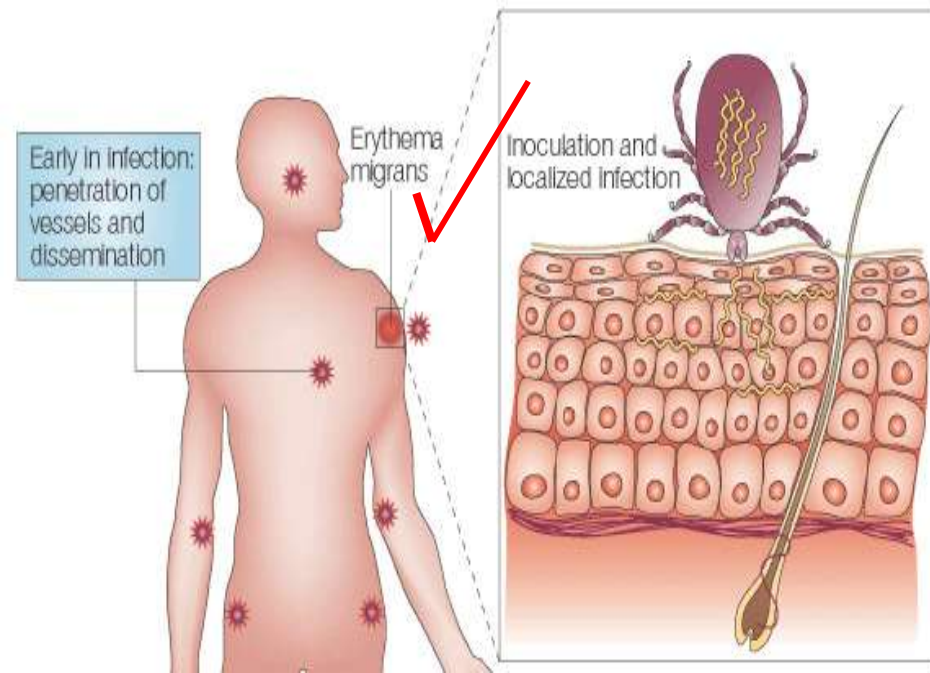
Tissue damage caused by *B. Burgdorferi*

Local skin infection

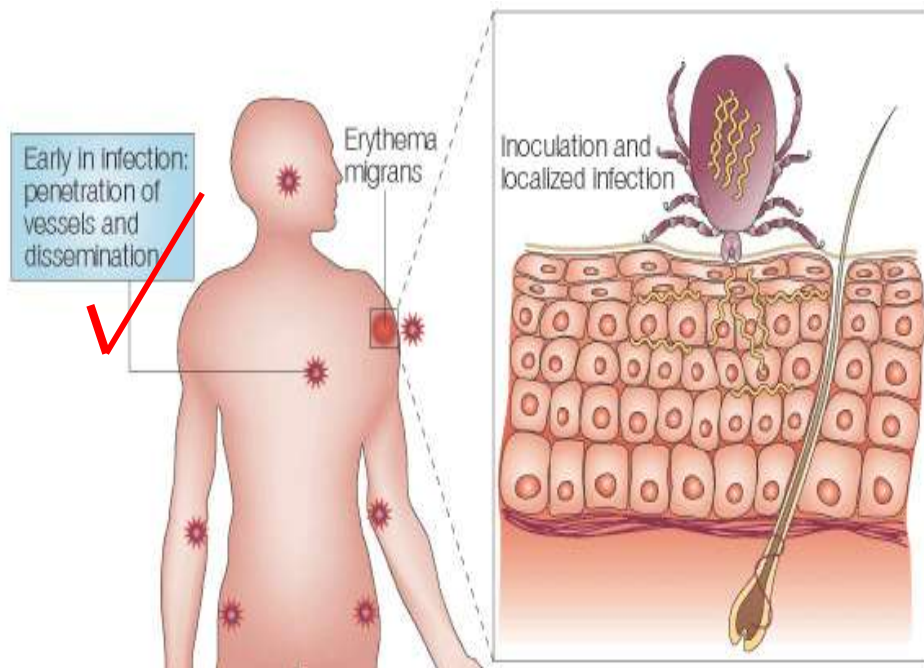
(stage 1 of the disease),

at the site of the tick bite,

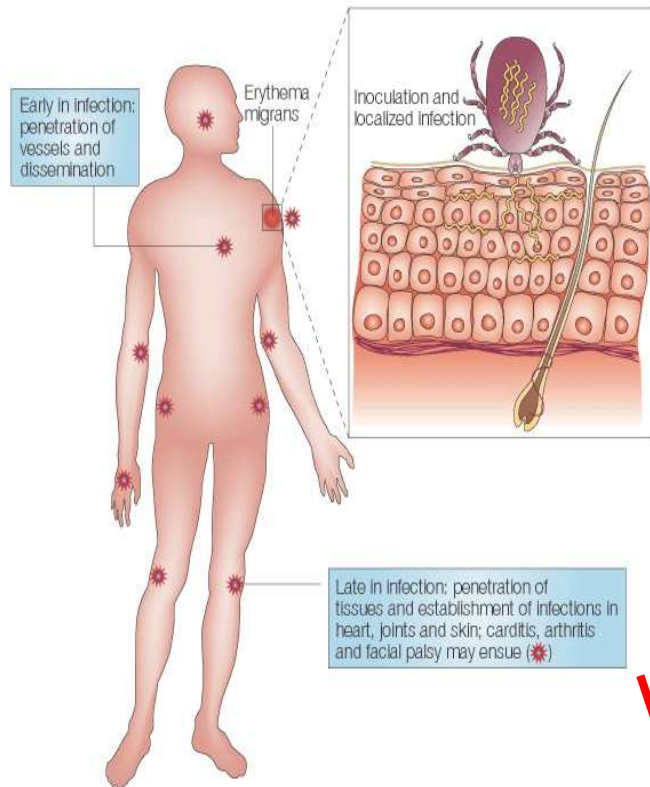
induces the development of **erythema migrans** (infiltration of lymphocytes and macrophages).



... **Disseminated infection (stage 2)** occurs due to **transient invasion of the bloodstream** by spirochetes. Some degree of vascular damage including **mild vasculitis** or **hypercellular vascular occlusion** may be seen at **various locations**, suggesting that spirochetes are colonizing the blood vessel wall.



In untreated patients, various manifestations of **late-stage Lyme disease (stage 3)** can occur several months and years after the onset of the disease, and are a consequence of **chronic infection** that is established in many tissues.



- recurrent episodes of arthritis (multiple joints)
- Lyme encephalopathy
- acrodermatitis chronica atrophicans



Nature Reviews | Microbiology

Lyme disease treatment

In the first stages - **doxycycline** in adults, and **amoxicillin** in children

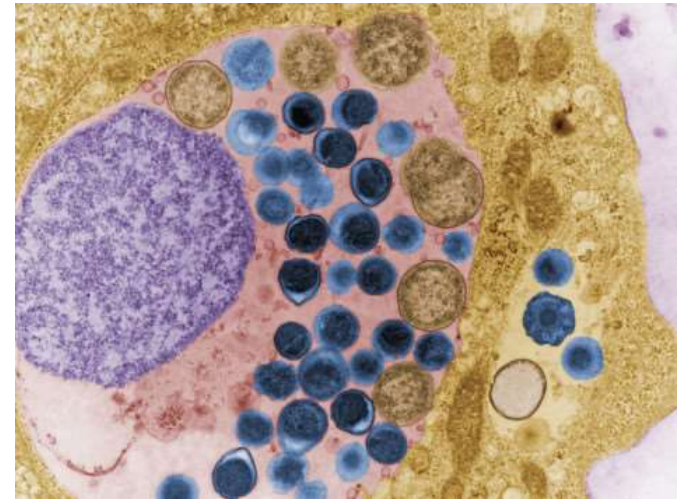
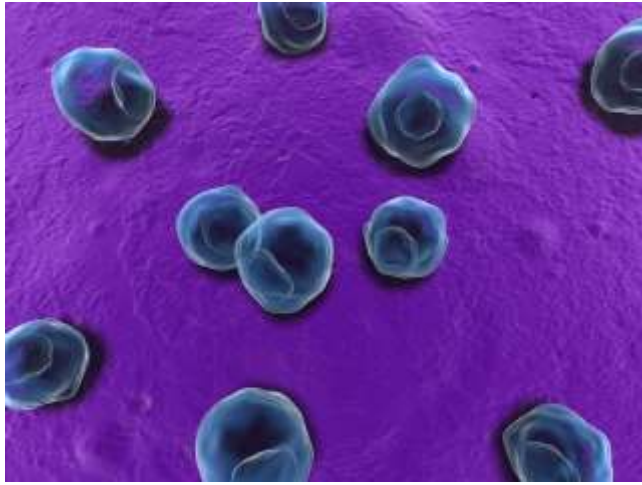
Lyme meningitis - 3rd generation cephalosporin drugs (**ceftriaxone** or **cefotaxime**) intravenously.
Myocarditis - ceftriaxone

A detailed microscopic illustration of a host cell, likely an epithelial cell, showing its internal organelles such as the nucleus, mitochondria, and endoplasmic reticulum. Numerous small, green, oval-shaped bacteria are depicted within the cytoplasm, some appearing to be in the process of budding or dividing. A large, circular, yellowish structure is visible in the upper left corner. The text 'Intracellular bacteria' is overlaid in a large, bold, dark blue font. Below it, the text '*Chlamydia species*' and '*Mycoplasma species*' are written in a smaller, red, italicized font. A large, dark blue arrow points from the bottom right towards the center of the cell.

Intracellular bacteria

Chlamydia species

Mycoplasma species



Chlamydia species

Genital and ocular infections

-Epidemiological characteristics-

Chlamydia- **mandatory intracellular** bacteria

C. Trachomatis serovary DK is leading bacterial causative agent full portable diseases.

Lymphogranuloma venerum biotype (LGV, serovars L1-L3) causes a sexually transmitted disease with lymphadenopathy.

Trachoma is an eye disease caused by *C. trachomatis* serovars A-C. It occurs mainly in poor communities in tropical regions.

They are transmitted by direct contact with the mucous membrane or through abrasions on the skin, by sexual contact or by direct inoculation into the eye, in the case of trachoma or neonatal conjunctivitis.

C. Pneumoniae and *C. psittaci* are usually transmitted by **respiratory route**.

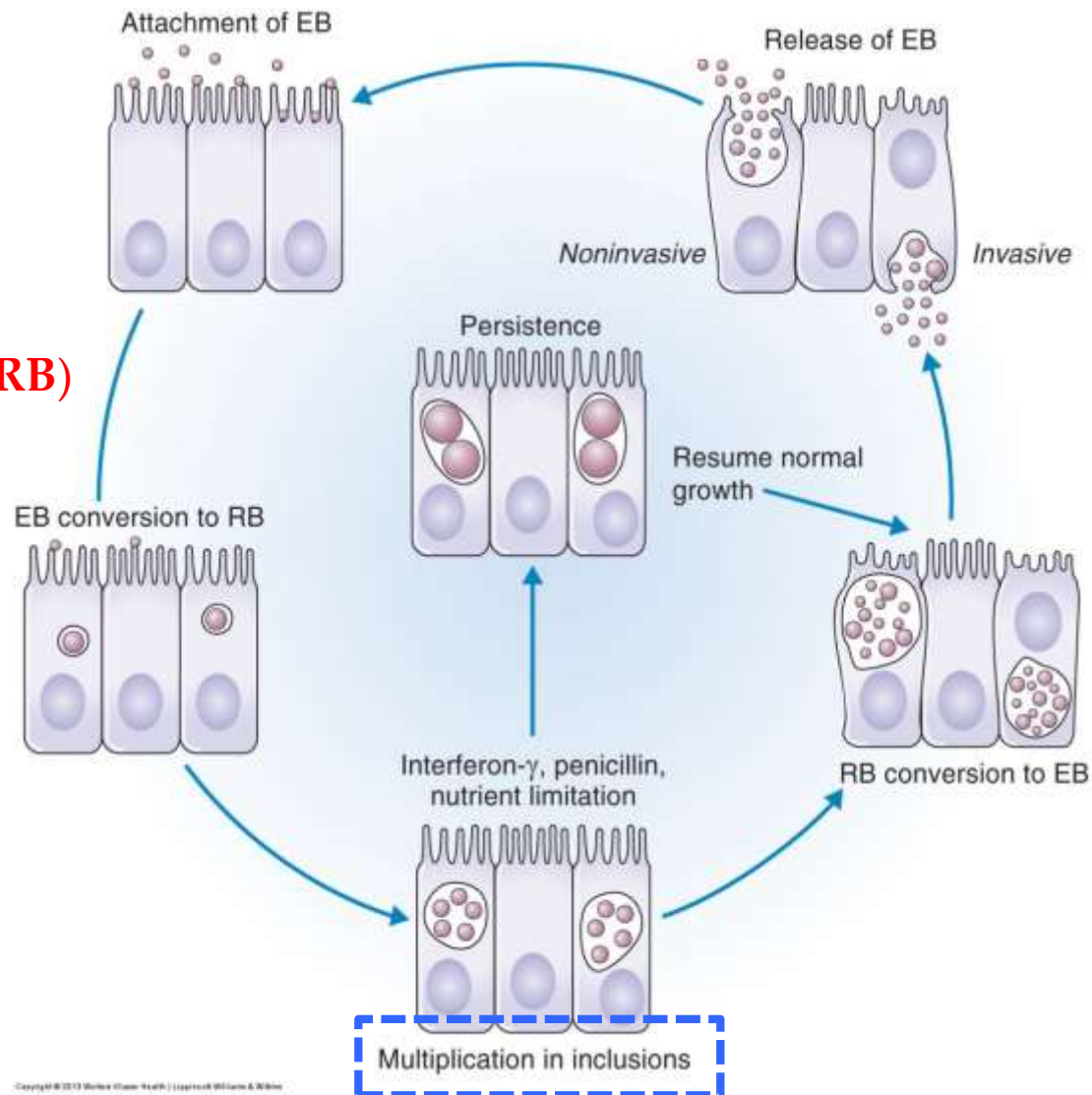


Expansion and multiplication of *Chlamydia*

Chlamydia growth in targeted epithelial cells is characterized by a unique developmental cycle.

✓ elementary body(EB)

✓ reticular body (RB)



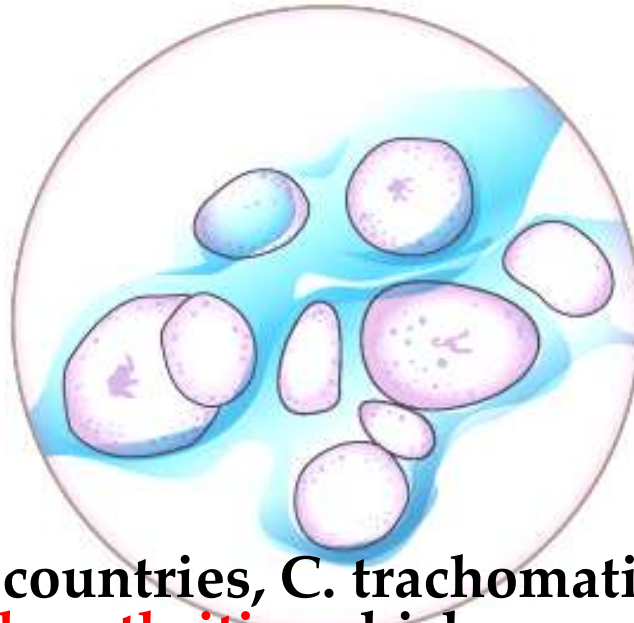
C. trachomatis, serovars D-K, initially infect and grow in the epithelial cells of the genital mucosa. **Chronic inflammation** causes epithelial cell necrosis, fibroblast proliferation, and eventual **scar tissue formation**.

Clinically, the early phase of genital infections caused by *C. trachomatis*, serovars D-K, is either very **discrete** or completely **asymptomatic**.

In women, the **endocervix** is sometimes **red with a mucoid exudate** that is less purulent than that of gonococcal infection. In men, a **scanty mucoid exudate from the urethra** may occur.



In developed countries, **urogenital serovars (D-K) of *C. trachomatis*** are one of the most common causes of nongonorrheic urethritis.



In developing countries, *C. trachomatis* often causes **postgonococcal urethritis**, which occurs when there is a coinfection of *N. gonorrhoeae* and *C. trachomatis*, in which the gonococcal infection is cured by the use of appropriate antibiotics but the chlamydial infection of the urethra is not.

Diseases caused by *C. pneumoniae*

C. pneumoniae is a common cause of **atypical pneumonia** accompanied by chronic inflammation. EBs are present in respiratory secretions.

In healthy adults, respiratory infections are usually mild or asymptomatic. Clinical manifestations include **pharyngitis, laryngitis, bronchitis, sinusitis, otitis media, or pneumonia.**



Diseases caused by *C. psittaci*

In humans, inhalation of *C. psittaci* causes an illness that initially resembles the flu, rapidly progressing to severe pneumonia. Rarely, infection can cause hepatitis, endocarditis, and/or encephalitis.





Mycoplasma species
Ureaplasma species

A diverse group of bacteria that lacks a cell wall and requires sterols for growth

Mycoplasmas

Mycoplasmas have a few characteristics...

...**they don't have rigid cellular wall** (they don't have murein) and because of that they have various shapes.

...their cellular membrane contains **sterols**.

Mycoplasmas

-epidemiological characteristics-

Micoplasma and ureaplasma cause infections of **respiratory** and **urogenital** system...

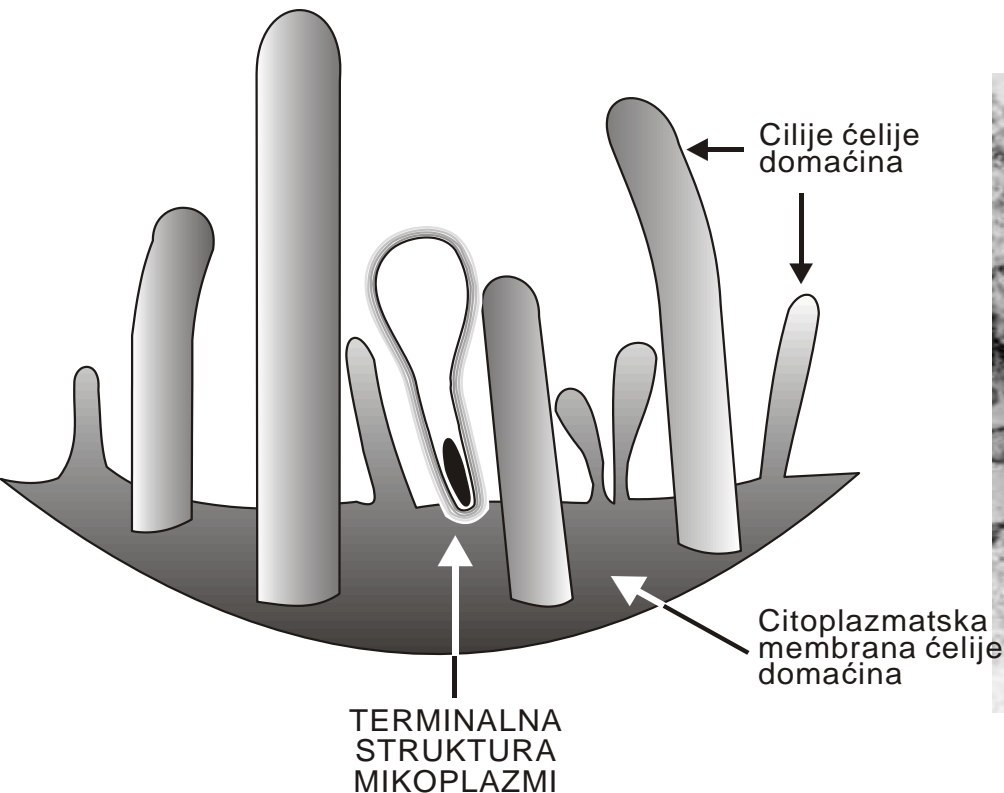
Mycoplasma pneumoniae -causative agent of respiratory infections

M.genitalium- causative agent of genitourinary infections

M. hominis -causes different infections of urogenital system, joints as well as meningitis in newborns

Mycoplasma pneumoniae

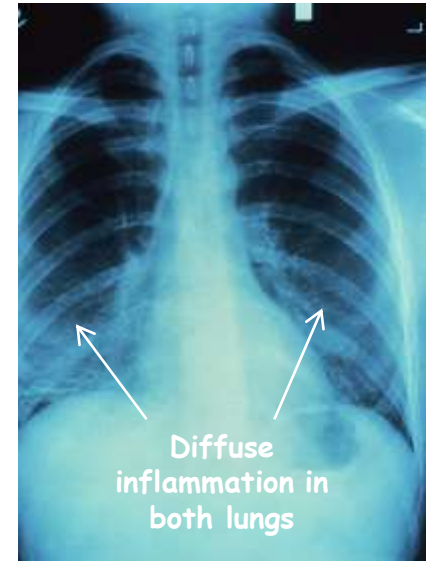
Infection begins with the **attachment** of the bacterium to the **respiratory epithelium**. *M. pneumoniae* possesses a **unique terminal attachment structure**. Specialized **adhesin** and **accessory adhesion proteins** are part of the attachment-mediating organelles, which are located at the **tip of the bacterium** and help the bacterium bind to **carbohydrate receptors** on the respiratory epithelium.



Mycoplasma pneumoniae

-diseases-

M. pneumoniae is a common cause of **pneumonia** that usually does not start suddenly and has a milder course than pneumococcal pneumonia. Previously, the imprecise term "**walking pneumonia**" was used. This disease is also referred to as primary **atypical pneumonia**, which is different from "typical" cases of lobar pneumonia (usually caused by pneumococcus). Patients with typical pneumonia respond well to penicillin, while patients with atypical pneumonia do not.



Genital mycoplasmas

M. genitalium is an important cause of non-chlamydial, non-gonococcal urethritis in men, and in women it is also associated with cervicitis, endometritis, and pelvic inflammatory disease.

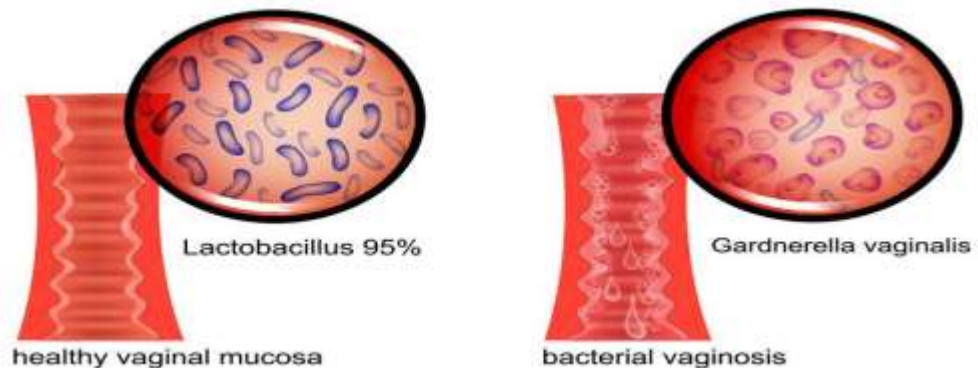
U. urealiticum, *U. parvum*, and *M. hominis* are part of the flora of the urogenital tract and infections are generally asymptomatic. It is **transmitted by sexual contact** between adults and is transmitted **vertically from mother to newborn** during pregnancy and childbirth.

U. urealiticum causes **non-gonococcal urethritis in men**.

M. hominis and *Ureaplasma species* can cause chorioamnionitis (inflammation of the fetal membranes) and postpartum endometritis.

Gardnerella vaginalis

- Gram negative anaerob
- small, round, convex, gray colonies on chocolate agar
- causative agent of **bacterial vaginosis** due to disruption of the normal vaginal microflora.
- in genital cultures, in blood, urine and throat samples.
- Although it predominates in bacterial vaginosis, it can also be isolated in women without any signs or symptoms of infection.
- produces a toxin that creates pores, **vaginolysin**
- metronidazole, clindamycin



Haemophilus ducreyi

- Gram negative coccobacillus
- causes a sexually transmitted disease
- Soft chancre - **Mole ulcer**
- Soft, painful shallow ulceration
- Inflammation - lymphocytes, macrophages and granulocytes.
- regional lymphadenitis
- therapy with macrolides, third-generation cephalosporins, or fluoroquinolones



Laboratory diagnostics of infectious diseases

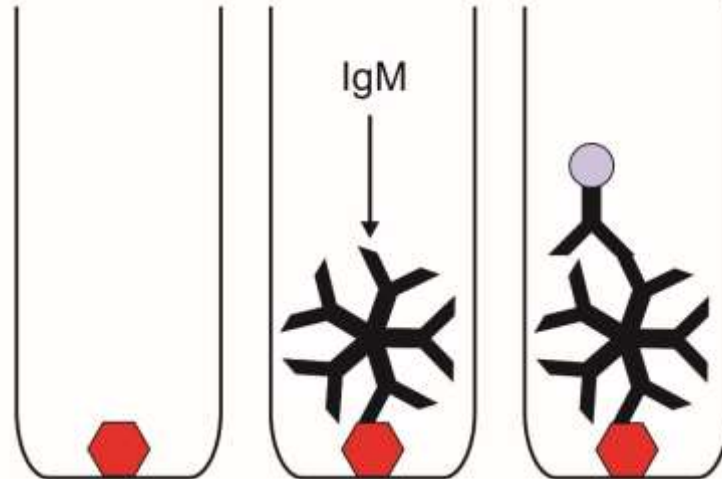
Immunological (serological) approach

- antigen or antibody detection

ELISA



**Ag-Ab reaction with Ab
enzyme-labeled**



 Viral
antigen



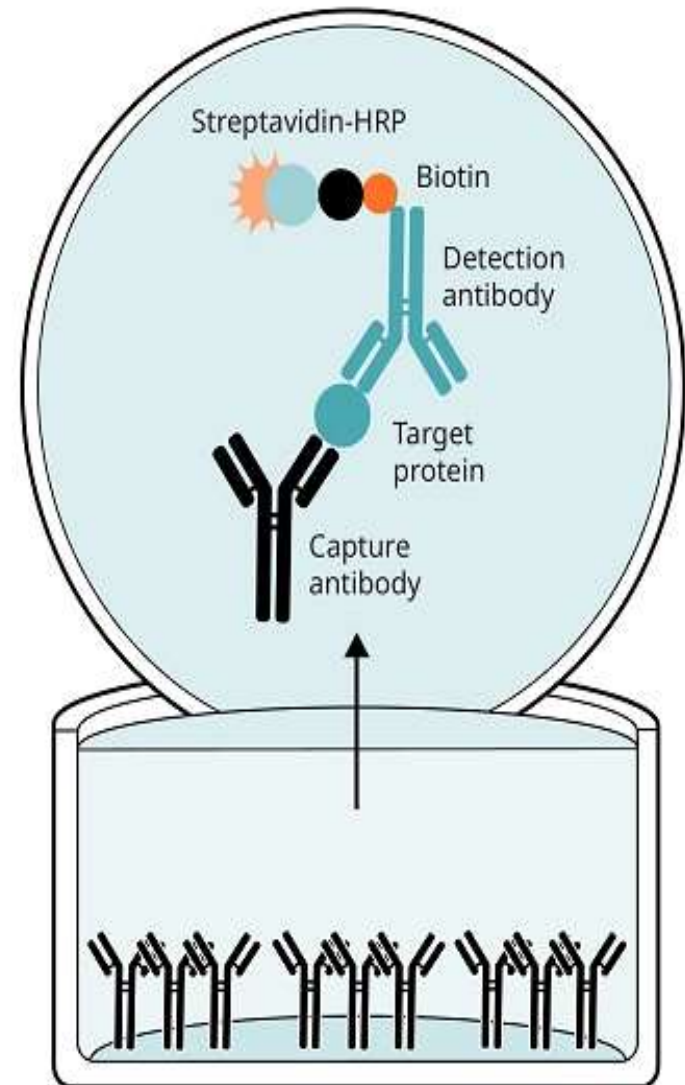
enzyme-labeled
anti-IgM specific
antibody

Basic principles of the ELISA test

In an ELISA test, the antigen is immobilized on a solid surface.

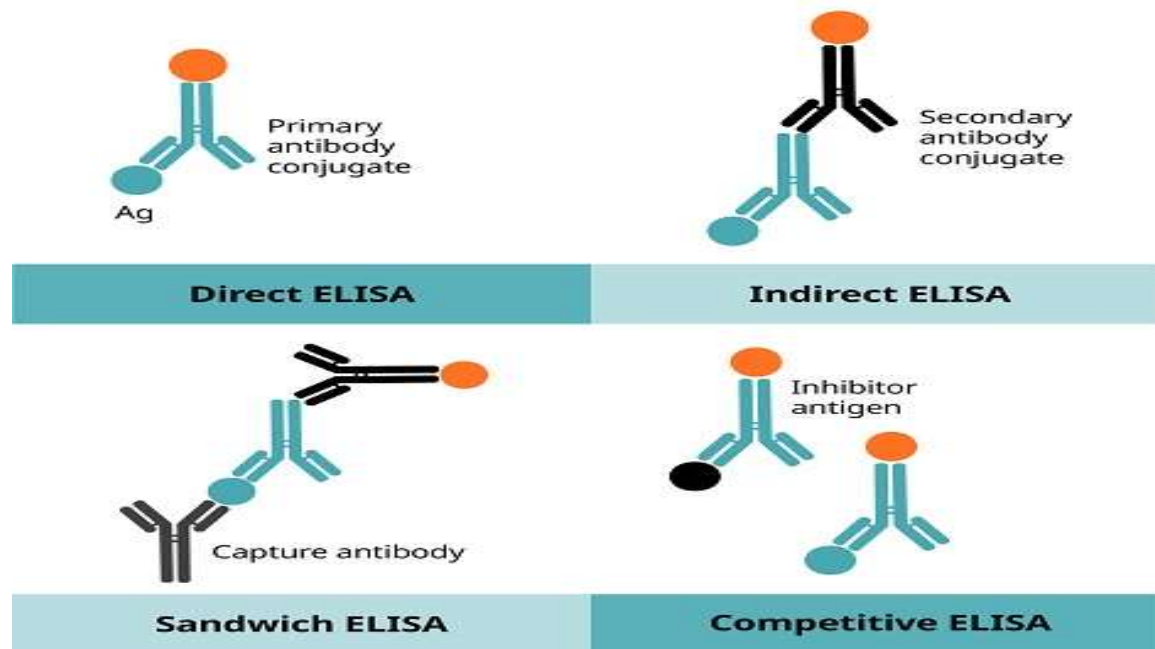
This is done directly or by using the antibody itself to capture an immobilized antigen on the surface.

The antigen then binds to a detection antibody conjugated to a molecule suitable for detection, such as an enzyme or fluorophore.



ELISA

- **Direct:** Antigen is fixed, and enzyme-labeled antibody is added
- **Indirect:** Uses primary and secondary antibody for greater sensitivity
- **Sandwich:** two antibodies specific for the same antigen
- **Competitive:** Based on competitive binding between free and labeled antigen



Western blot

- Western blot a method for **detection and identification of specific proteins** in the sample
- It is based on a combination of:
 - **electrophoresis (SDS-PAGE)**– protein size separation
 - **Transfer** membrane proteins
 - **immunochemical detections** using antibodies
- The primary antibody binds to the target protein
- Secondary antibody allows visualization (enzyme/fluorescence)
- Result: **tapes** which represent the presence of proteins

