WHY
ARE
YOU
LATE!

Brucella (Brucellosis, Bang's Disease)

Occurrence and classification.
The genus *Brucella* includes three medically relevant *species—B. abortus*, *B. melitensis*, and *B. suis* – besides a number of others. These three species are the causative organisms of classic zoonoses in livestock and wild animals, specifically in cattle (*B. abortus*), goats (*B. melitensis*), and pigs (*B. suis*). These bacteria can also be transmitted from diseased animals to humans, causing a uniform clinical picture, so-called undulant fever or Bang's disease.

Morphology and culture

Brucellae are slight, coccoid, Gram-negative rods with no flagella. They only reproduce aerobically. In the initial isolation the atmosphere must contain 5-10% CO_2. Enriched mediums such as blood agar are required to grow them in cultures.

Pathogenesis and clinical picture

Human brucellosis infections result from direct contact with diseased animals or indirectly by way of contaminated foods, in particular unpasteurized milk and dairy products. The bacteria invade the body either through the mucosa of the upper intestinal and respiratory tracts or through lesions in the skin, then enter the subserosa or subcutis. From there they are transported by microphages or macrophages, in which they can survive, to the lymph nodes, where a lymphadenitis develops. The pathogens then disseminate from the affected lymph nodes, at first lymphogenously and then hematogenously, finally reaching the liver, spleen, bone marrow, and other RES tissues, in the cells of which they can survive and even multiply.

Diagnosis

This is best achieved by isolating the pathogen from blood or biopsies in cultures, which must be incubated for up to four weeks. The laboratory must therefore be informed of the tentative diagnosis. Brucellae are identified based on various metabolic properties and the presence of surface antigens, which are detected using a polyvalent *Brucella*-antiserum in a slide agglutination reaction. Special laboratories are also equipped to differentiate the three *Brucella* species. Antibody detection is done using the agglutination reaction according to Gruber-Widal in a standardized method. In doubtful cases, the complement binding reaction and direct Coombs test can be applied to obtain a serological diagnosis.

Epidemiology and prevention

Brucellosis is a zoonosis that affects animals all over the world. Infections with *B. melitensis* occur most frequently in Mediterranean countries, in Latin America, and in Asia. The melitensis brucelloses seen in Europe are either caused by milk products imported from these countries or occur in travelers. B. abortus infections used to be frequent in central Europe, but the disease has now practically disappeared there thanks to the elimination of *Brucella*-infested cattle herds. Although control of brucellosis infections focuses on prevention of exposure to the pathogen, it is not necessary to isolate infected persons since the infection is not communicable between humans. There is no vaccine.

Bordetella (Whooping Cough, Pertussis)

The genus *Bordetella*, among others, includes the species *B. pertussis*, *B. parapertussis*, and *B. bronchiseptica*. Of the three, the pathogen responsible for whooping cough, *B. pertussis*, is of greatest concern for humans. The other two species are occasionally observed as human pathogens in lower respiratory tract infections.

Morphology and culture. B. pertussis bacteria are small, coccoid, non-motile, Gram-negative rods that can be grown aerobically on special culture mediums at 37°C for three to four days.

Pathogenesis. Pertussis bacteria are transmitted by aerosol droplets. They are able to attach themselves to the cells of the ciliated epithelium in the bronchi. They rarely invade the epithelium. The infection results in (sub-) epithelial inflammations and necroses.

Pathogenicity Factors of *Bordetella pertussis*

Adhesion factors. The two most important factors are filamentous hemagglutin (FHA) and pertussis toxin (Ptx). The latter can function both as an exotoxin and as an adhesin. The pathogenic cells attach themselves to the epithelial cilia.

Exotoxins. Pertussis toxin: AB toxin (see p. 16); the A component is an ADP-ribosyl transferase; mechanism of action via G_s proteins (as with cholera toxin A1); increased amount of cAMP in target cells, with a variety of effects depending on the type of cell affected by the toxin.

Invasive adenylate cyclase: AB toxin; A enters cells, acts in addition to pertussis toxin to increase levels of cAMP.

Endotoxins. Tracheal cytotoxin: murein fragment; kills ciliated epithelial cells. Lipopolysaccharide: stimulates cytokine production; activates complement by the alternative pathway.

Diagnosis

The pathogen can only be isolated and identified during the catarrhal and early paroxysmal phases. Specimen material is taken from the nasopharynx through the nose using a special swabbing technique. A special medium is then carefully inoculated or the specimen is transported to the laboratory using a suitable transport medium. *B. pertussis* can also be identified in nasopharyngeal secretion using the direct immunofluorescence technique. Cultures must be aerobically incubated for three to four days. Antibodies cannot be detected by EIA until two weeks after onset at the earliest. Only a seroconversion is conclusive.

Therapy. Antibiotic treatment can only be expected to be effective during the catarrhal and early paroxysmal phases before the virulence factors are bound to the corresponding cell receptors. Macrolides are the agents of choice.

Epidemiology and prevention. Pertussis occurs worldwide. Humans are the only hosts. Sources of infection are infected persons during the catarrhal phase, who cough out the pathogens in droplets. There are no healthy carriers. The most important preventive measure is the active vaccination (see vaccination schedule). Although a whole-cell vaccine is available, various acellular vaccines are now preferred.

Treponema (Syphilis, Yaws, Pinta)

Morphology and culture. These organisms are slender bacteria, 0.2 μm wide and 5–15 μm long; they feature 10–20 primary windings and move by rotating around their lengthwise axis. Their small width makes it difficult to render them visible by staining. They can be observed in vivo using dark field microscopy. In-vitro culturing has not yet been achieved.

Pathogenesis and clinical picture. Syphilis affects only humans. The disease is normally transmitted by sexual intercourse. Infection comes about because of direct contact with lesions containing the pathogens, which then invade the host through microtraumata in the skin or mucosa. The incubation period is two to four weeks.

Left untreated, the disease manifests in several stages:

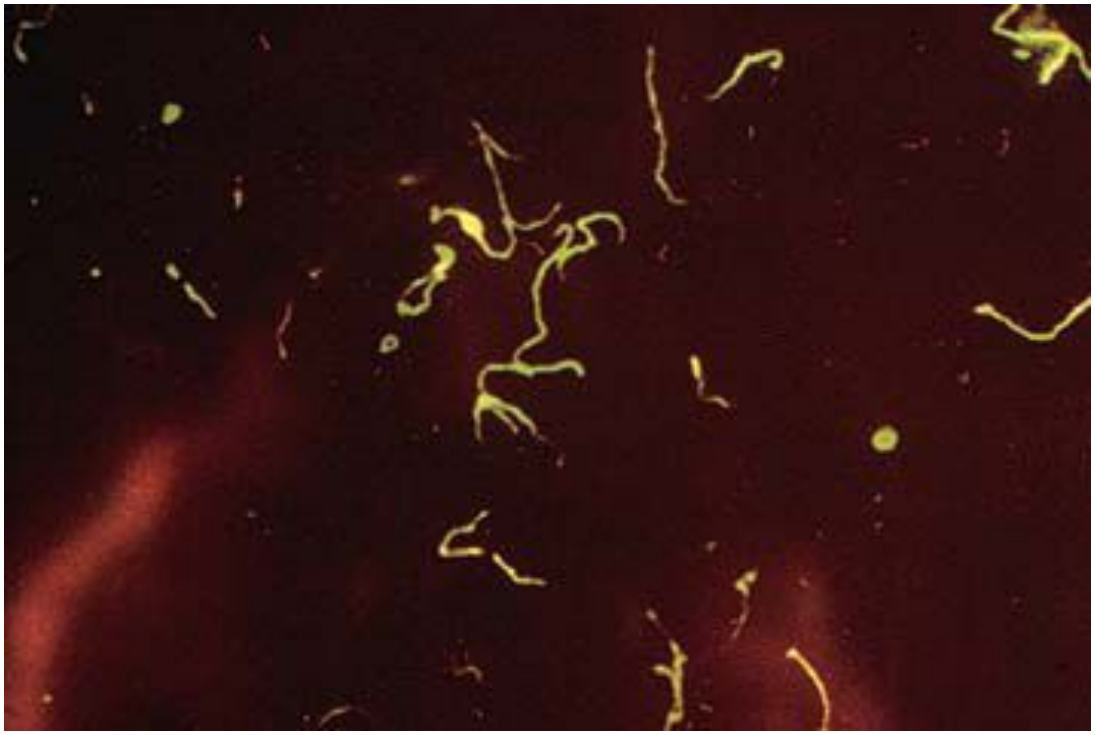
Stage I (primary syphilis). Hard, indolent (painless) lesion, later infiltration and ulcerous disintegration, called hard chancre. Accompanied by regional lymphadenitis, also painless. Treponemes can be detected in the ulcer.

Stage II (secondary syphilis). Generalization of the disease occurs four to eight weeks after primary syphilis. Frequent clinical symptoms include micropolylymphadenopathy and macular or papulosquamous exanthem, broad condylomas, and enanthem. Numerous organisms can be detected in seeping surface efflorescences.

Latent syphilis. Stage of the disease in which no clinical symptoms are manifested, but the pathogens are present in the body and serum antibody tests are positive. Divided into early latency (less than four years) and late latency (more than four years).

Stage III (tertiary or late syphilis). Late gummatous syphilis: manifestations in skin, mucosa, and various organs. Tissue disintegration is frequent. Lesions are hardly infectious or not at all. Cardiovascular syphilis: endarteritis obliterans, syphilitic aortitis. Neurosyphilis: two major clinical categories are observed: meningovascular syphilis, i.e., endarteritis obliterans of small blood vessels of the meninges, brain, and spinal cord; parenchymatous syphilis, i.e., destruction of nerve cells in the cerebral cortex (paresis) and spinal cord (tabes dorsalis). A great deal of overlap occurs.

Syphilis connata. Transmission of the pathogen from mother to fetus after the fourth month of pregnancy. Leads to miscarriage or birth of severely diseased infant with numerous treponemes in its organs.



Serous transudate from moist mucocutaneous primary chancre. Direct immunofluorescence.

Therapy.
Penicillin G is the antibiotic agent of choice. Dosage and duration of therapy depend on the stage of the disease and the galenic formulation of the penicillin used.

Epidemiology and prevention.
Syphilis is known all over the world. Annual prevalence levels in Europe and the US are 10–30 cases per 100 000 inhabitants. The primary preventive measure is to avoid any contact with syphilitic efflorescences. When diagnosing a case, the physician must try to determine the first-degree contact person, who must then be examined immediately and provided with penicillin therapy as required. National laws governing venereal disease management in individual countries regulate the measures taken to diagnose, prevent, and heal this disease. There is no vaccine.

Borrelia (Relapsing Fever, Lyme Disease)

Borrelia burgdorferi (Lyme Disease)

Classification.

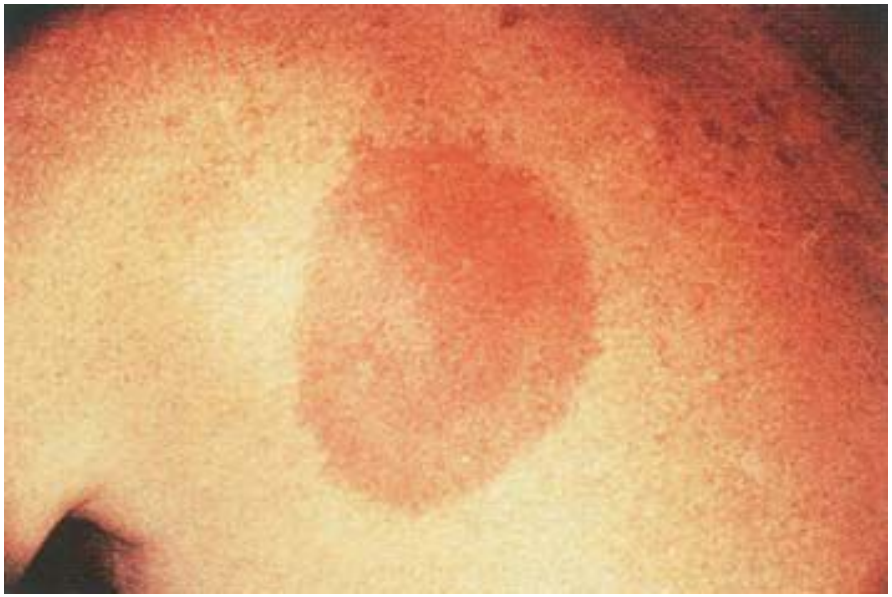
The etiology of an increase in the incidence of acute cases of arthritis among youths in the Lyme area of Connecticut in 1977 was at first unclear. The illness was termed Lyme arthritis. It was not until 1981 that hitherto unknown borreliae were found to be responsible for the disease. They were classified as *B. burgdorferi* in 1984 after their discoverer. Analysis of the genome of various isolates has recently resulted in a proposal to subclassify *B. burgdorferi* sensu lato in three species: *B. burgdorferi* sensu stricto, *B. garinii*, *B. afzelii*.

Morphology and culture

These are thin, flexible, helically wound, highly motile spirochetes. They can be rendered visible with Giemsa staining or by means of dark field or phase contrast microscopy methods. These borreliae can be grown in special culture mediums at 35°C for five to 10 days, although culturing these organisms is difficult and often unsuccessful.

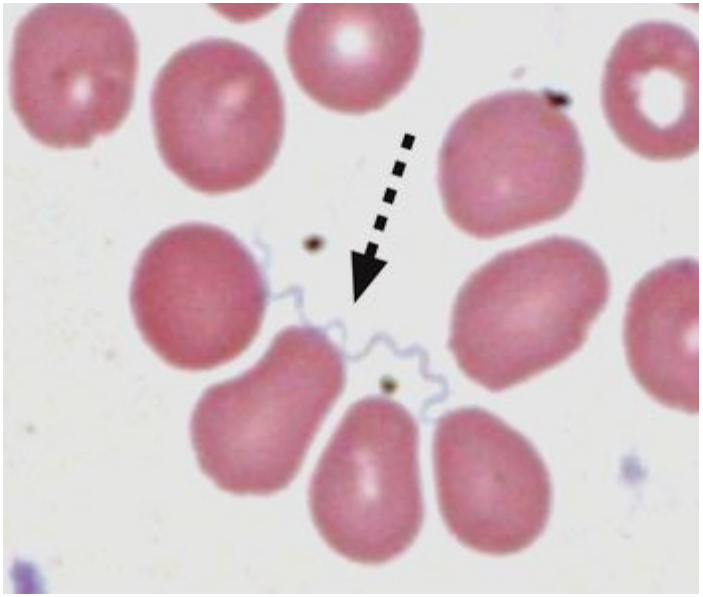
Pathogenesis and clinical picture

The pathogens are transmitted by the bite of various tick species. The incubation period varies from three to 30 days. Left untreated, the disease goes through three stages, though individual courses often deviate from the classic pattern. The presenting symptom in stage I is the erythema chronicum migrans.



Erythema chronicum migrans

Male tick (size: 2 mm)



Diagnosis

Direct detection and identification of the pathogen by means of microscopy and culturing techniques is possible, but laden with uncertainties. In a recent development, the polymerase chain reaction (PCR) is used for direct detection of pathogen-specific DNA. However, the method of choice is still the antibody test (EIA or indirect immunofluorescence,Western blotting if the result is positive).

Therapy

Stages I and II: amoxicillin, cefuroxime, doxycycline, or a macrolide. Stage III: ceftriaxone.

Epidemiology and prevention

Lyme disease occurs throughout the northern hemisphere. There are some endemic foci where the infection is more frequent. The disease is transmitted by various species of ticks, in Europe mostly by Ixodes ricinus (sheep tick). In endemic areas of Germany, approximately 3–7% of the larvae and 10–34% of nymphs and adult ticks are infected with B. burgdorferi sensu lato. The annual incidence of acute Lyme disease (stage I) in central Europe is 20–50 cases per 100 000 inhabitants. Wild animals from rodents on up to deer are the natural reservoir of the Lyme disease *Borrelia*, although these species seldom come down with the disease. The ticks obtain their blood meals from these animals.

Leptospira (Leptospirosis, Weil Disease)

Classification. Leptospirae belong to the family Leptospiraceae. The genus Leptospira comprises two species. L. biflexa includes all apathogenic leptospirae and L. interrogans represents the pathogenic species. Based on its specific surface antigen variety, L. interrogans is subclassified in over 100 serovars in 19 serogroups. Some of the most important serogroups are: icterohemorrhagiae, canicola, pomona, australis, grippotyphosa, hyos, and sejroe.

Morphology and culture. Leptospirae are fine spirochetes, 10–20 μm long, and 0.1–0.2 μm thick. They possess no flagella, but rather derive their motility from rotating motions of the cell corpus. Visualization of leptospirae is best done using dark field or phase contrast microscopy. Leptospirae can be grown in special culture mediums under aerobic conditions at temperatures between 27–30°C

Serogroup icterohemorrhagiae. Culture preparation. Dark field microscopy.

Pathogenesis and clinical picture. Leptospirae invade the human organism through microinjuries in the skin or the intact conjunctival mucosa. There are no signs of inflammation in evidence at the portal of entry. The organisms spread to all parts of the body, including the central nervous system, hematogenously. Leptospirosis is actually a generalized vasculitis. The pathogens damage mainly the endothelial cells of the capillaries, leading to greater permeability and hemorrhage and interrupting the O_2 supply to the tissues. Jaundice is caused by a nonnecrotic hepatocellular dysfunction. Disturbances of renal function result from hypoxic tubular damage. A clinical distinction is drawn between anicteric leptospirosis, which has a milder course, and the severe clinical picture of icteric leptospirosis (Weil disease). In principle, any of the serovars could potentially cause either of these two clinical courses. In practice, however, the serogroup icterohemorrhagiae is isolated more frequently in Weil disease.

Diagnosis

Detection and identification of leptospirae are accomplished by growing the organisms in cultures. Blood, cerebrospinal fluid, urine, or organ biopsies, which must not be contaminated with other bacteria, are incubated in special mediums at 27–30°C for three to four weeks. A microscope check (dark field) is carried out every week to see if any leptospirae are proliferating. The Leptospirae are typed serologically in a lysis-agglutination reaction with specific test sera.

The method of choice for a laboratory diagnosis is an antibody assay. The antibodies produced after the first week of the infection are detected in patient serum using a quantitative lysis-agglutination test. Viable culture strains of the regionally endemic serovars provide the test antigens. The reaction is read off under the microscope.

Therapy. The agent of choice is penicillin G.

Epidemiology and prevention. Leptospiroses are typical zoonotic infections. They are reported from every continent in both humans and animals. The most important sources of infection are rodents and domestic animals, mainly pigs. The animals excrete the pathogen with urine. Leptospirae show little resistance to drying out so that infections only occur because of contact with a moist milieu contaminated with urine. The persons most at risk are farmers, butchers, sewage treatment workers, and zoo staff.

Prevention of these infections involves mainly avoiding contact with material containing the pathogens, control of *Muridae* rodents and successful treatment of domestic livestock. It is not necessary to isolate infected persons or their contacts. There is no commercially available vaccine.

Rickettsia, Coxiella, Orientia, and *Ehrlichia* (Typhus, Spotted Fever, Q Fever, Ehrlichioses)

The genera of the *Rickettsiaceae* and *Coxelliaceae* contain short, coccoid, small rods that can only reproduce in host cells. With the exception of Coxiella (aerogenic transmission), they are transmitted to humans via the vectors lice, ticks, fleas, or mites. *R. prowazekii* and *R. typhi* cause typhus, a disease characterized by high fever and a spotty exanthem. Several rickettsiae species cause spotted fever, a milder typhuslike disease. Orientia tsutsugamushi is transmitted by mite larvae to cause tsutsugamushi fever. This disease occurs only in Asia. Coxiella burnetii is responsible for Q fever, an infection characterized by a pneumonia with an atypical clinical course.

Several species of *Ehrlichiaceae* cause ehrlichiosis in animals and humans. The method of choice for laboratory diagnosis of the various rickettsioses and ehrlichioses is antibody assay by any of several methods, in most cases indirect immunofluorescence. Tetracyclines represent the antibiotic of choice for all of these infections. Typhus and spotted fever no longer occur in Europe. Q fever infections are reported from all over the world. Sources of infection include diseased sheep, goats, and cattle. The prognosis for the rare chronic form of Q fever (syn. Q fever endocarditis) is poor. Ehrlichiosis infects mainly animals, but in rare cases humans as well.

The bacteria of this group belong to the families Rickettsiaceae (*Rickettsia* and *Orientia*), Coxelliaceae (*Coxiella*), and Ehrlichiaceae (*Ehrlichia, Anaplasma, Neorickettsia*). Some of these organisms can cause mild, selflimiting infections in humans, others severe disease. Arthropods are the transmitting vectors in many cases.

Morphology and culture. These obligate cell parasites are coccoid, short rods measuring 0.3–0.5 lm that take gram staining weakly, but Giemsa staining well. They reproduce by intracellular, transverse fission only. They can be cultured in hen embryo yolk sacs, in suitable experimental animals (mouse, rat, guinea pig) or in cell cultures.

Pathogenesis and clinical pictures.

With the exception of *C. burnetii*, the organisms are transmitted by arthropods. In most cases, the arthropods excrete them with their feces and ticks transmit them with their saliva while sucking blood. The organisms invade the host organism through skin injuries. *C. burnetii* is transmitted exclusively by inhalation of dust containing the pathogens. Once inside the body, rickettsiae reproduce mainly in the vascular endothelial cells. These cells then die, releasing increasing numbers of organisms into the bloodstream. Numerous inflammatory lesions are caused locally around the destroyed endothelia. Ehrlichiae reproduce in the monocytes or granulocytes of membrane-enclosed cytoplasmic vacuoles. The characteristic morulae clusters comprise several such vacuoles stuck together.

Diagnosis

Direct detection and identification of these organisms in cell cultures, embryonated hen eggs, or experimental animals is unreliable and is also not to be recommended due to the risk of laboratory infections. Special laboratories use the polymerase chain reaction to identify pathogen-specific DNA sequences. However, the method of choice is currently still the antibody assay, whereby the immunofluorescence test is considered the gold standard among the various methods. The Weil-Felix agglutination test is no longer used today due to low sensitivity and specificity.

Therapy

Tetracyclines lower the fever within one to two days and are the antibiotics of choice.

Epidemiology and prevention

The epidemic form of typhus, and earlier scourge of eastern Europe and Russia in particular, has now disappeared from Europe and occurs only occasionally in other parts of theworld. Murine typhus, on the other hand, is still a widespread disease in the tropics and subtropics. Spotted fevers (e.g., Rocky Mountain spotted fever) occur with increased frequency in certain geographic regions, especially in the spring. Tsutsugamushi fever occurs only in Japan and Southeast Asia.

Bartonella

Classification. The genus *Bartonella* includes, among others, the species *B. bacilliformis*, *B. quintana*, *B. henselae*, and *B. clarridgeia*.

Morphology and culture. Bartonella bacteria are small (0.6–1 μm), Gram-negative, frequently pleomorphic rods. Bartonellae can be grown on culture mediums enriched with blood or serum.

Pathogen	Transmission/ host	Disease	Clinical picture
Bartonella bacilliformis	Sand fly/ humans	Oroya fever (Carrion's disease)	Incubation: 15–40 days; high fever; lymphadenitis; splenohepatomegaly; hemolytic anemia due to lysis of erythrocytes invaded by *B. bacilliformis*
		Verruga peruana phase of Oroya fever	Multiple, wartlike skin lesions on extremities, face, mucosa; onset either months after abating of Oroya fever or without an acute preceding infection
B. quintana	Lice/humans	Five-day fever (Wolhynian fever, trench fever)	Periodic relapses of fever (3–8) every 5 days, sepsis; bacillary angiomatosis (see below); also endocarditis
B. henselae	Cats to humans/cats	Cat scratch disease	Lymphadenopathy; fever; cutaneous lesion (not always present)
		Sepsis, bacillary angiomatosis	In patients with immune deficiencies (HIV); vascular proliferation in skin and mucosa (similar to verruga peruana)
		Bacterial peliosis hepatis/splenica	Cystic, blood-filled lesions in liver and spleen
B. clarridgeia		Cat scratch disease	See above

Diagnosis. Special staining techniques are used to render bartonellae visible under the microscope in tissue specimens. Growth in cultures more than seven days. Amplification of specific DNA in tissue samples or blood, followed by sequencing. Antibody assay with IF or EIA.

Therapy. Tetracyclines, macrolides. Epidemiology and prevention. Oroya fever (also known as Carrion disease) is observed only in humans and is restricted to mountain valleys with elevations above 800 m in the western and central Cordilleras in South America because an essential vector, the sand fly, lives only there. Cat scratch disease, on the other hand, is known all over the world. It is transmitted directly from cats to humans or indirectly by cat fleas. The cats involved are usually not sick.

Chlamydia

Chlamydiae are obligate cell parasites. They go through two stages in their reproductive cycle: the elementary bodies (EB) are optimized to survive outside of host cells. In the form of the initial bodies (IB), the chlamydiae reproduce inside the host cells.

The three human pathogen species of chlamydiae are ***C. psittaci***, ***C. trachomatis***, and ***C. pneumoniae***. Tetracyclines and macrolides are suitable for treatment of all chlamydial infections.

C. psittaci is the cause of **psittacosis or ornithosis**. This zoonosis is a systemic disease of birds. The pathogens enter human lungs when dust containing chlamydiae is inhaled. After an incubation period of one to three weeks, pneumonia develops that often shows an atypical clinical course.

The bacteria in the taxonomic family Chlamydiaceae
are small (0.3–1 μm) obligate cell parasites with a Gram-negative cell wall. The reproductive cycle of the chlamydiae comprises two developmental stages: The elementary bodies are optimally adapted to survival outside of host cells. The initial bodies, also known as reticulate bodies, are the form in which the chlamydiae reproduce inside the host cells by means of transverse fission.

Two morphologically and functionally distinct forms are known

Elementary bodies. The round to oval, optically dense elementary bodies have a diameter of approximately 300 nm. They represent the infectious form of the pathogen and are specialized for the demands of existence outside the host cells. Once the elementary bodies have attached themselves to specific host cell receptors, they invade the cells by means of endocytosis. Inside the cell, they are enclosed in an endocytotic membrane vesicle or inclusion, in which they transform themselves into the other form - initial bodies - within a matter of hours.

Initial bodies. Chlamydiae in this spherical to oval form are also known as reticular bodies. They have a diameter of approximately 1000 nm. The initial bodies reproduce by means of transverse fission and are not infectious while in this stage. At the end of the cycle, the initial bodies are transformed back into elementary bodies. The cell breaks open and releases the elementary bodies to continue the cycle by attaching themselves to new host cells.

Chlamydia psittaci (Ornithosis, Psittacosis)

Pathogenesis and clinical picture

The natural hosts of *C. psittaci* are birds. This species causes infections of the respiratory organs, the intestinal tract, the genital tract, and the conjunctiva of parrots and other birds. Humans are infected by inhalation of dust (from bird excrements) containing the pathogens, more rarely by inhalation of infectious aerosols. After an incubation period of one to three weeks, ornithosis presents with fever, headache, and a pneumonia that often takes an atypical clinical course. The infection may, however, also show no more than the symptoms of a common cold, or even remain clinically silent. Infected persons are not usually sources of infection.

Diagnosis. The pathogen can be grown from sputum in special cell cultures. Direct detection in the culture is difficult and only possible in specially equipped laboratories. The complement binding reaction can be used to identify antibodies to a generic antigen common to all chlamydiae, so that this test would also have a positive result in the presence of other chlamydial infections. The antibody test of choice is indirect microimmunofluorescence.

Therapy. Tetracyclines (doxycycline) and macrolides.

Epidemiology and prevention. Ornithosis affects birds worldwide. It is also observed in poultry. Diagnosis of an ornithosis in a human patient necessitates a search for and elimination of the source, especially if the birds in question are household pets.

Chlamydia trachomatis (Trachoma, Lymphogranuloma venereum)

C. trachomatis is a pathogen that infects only humans. Trachoma is a follicular keratoconjunctivitis. The disease occurs in all climatic zones, although it is more frequent in warmer, less-developed countries. It is estimated that 400 million people carry this chronic infection and that it has caused blindness in six million. The pathogen is transmitted by direct contact and indirectly via objects in daily use. Left untreated, the initially acute inflammation can develop a chronic course lasting months or years and leading to formation of a corneal scar, which can then cause blindness. The laboratory diagnostics procedure involves detection of *C. trachomatis* in conjunctival smears using direct immunofluorescence microscopy. The fluorochrome-marked monoclonal antibodies are directed against the MOMP (major outer membrane protein) of *C. trachomatis*.

Lymphogranuloma venereum. This venereal disease (syn. Lymphogranuloma inguinale, lymphopathia venerea (Favre-Durand-Nicolas disease) not to be confused with granuloma inguinale) is frequently observed in the inhabitants of warm climatic zones. A herpetiform primary lesion develops at the site of invasion in the genital area, which then becomes an ulcus with accompanying lymphadenitis. Laboratory diagnosis is based on isolating the proliferating pathogen in cell cultures from purulent material obtained from the ulcus or from matted lymph nodes. The antibodies can be identified using the complement binding reaction or the icroimmunofluorescence test. Tetracyclines and macrolides are the potentially useful antibiotic types.

Chlamydia pneumoniae

This new chlamydial species (formerly TWAR chlamydiae) causes infections of the respiratory organs in humans that usually run a mild course: influenzalike infections, sinusitis, pharyngitis, bronchitis, pneumonias (atypical). Clinically silent infections are frequent. *C. pneumoniae* is pathogenic in humans only. The pathogen is transmitted by aerosol droplets. These infections are probably among the most frequent human chlamydial infections. Serological studies have demonstrated antibodies to C. pneumoniae in 60% of adults. Specific laboratory diagnosis is difficult. Special laboratories can grow and identify the pathogen in cultures and detect it under the microscope using marked antibodies to the LPS (although this test is positive for all chlamydial infections). *C. pneumoniae*-specific antibodies can be identified with the microimmunofluorescence method. In a primary infection, a measurable titer does not develop for some weeks and is also quite low. The antibiotics of choice are tetracyclines or macrolides. There is a growing body of evidence supporting a causal contribution by *C. pneumoniae* to atherosclerotic plaque in the coronary arteries, and thus to the pathogenesis of coronary heart
disease.

Mycoplasma

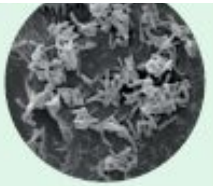
Mycoplasmas are bacteria that do not possess rigid cell walls for lack of a murein layer. These bacteria take on many different forms. They can only be rendered visible in their native state with phase contrast or dark field microscopy. Mycoplasmas can be grown on culture mediums with high osmotic pressure levels. *M. pneumoniae* frequently causes pneumonias that run atypical courses, especially in youths. Ten to twenty percent of pneumonias contracted outside of hospitals are caused by this pathogen. *M. hominis* and *Ureaplasma urealyticum* contribute to nonspecific infections of the urogenital tract. Infections caused by Mycoplasmataceae can be diagnosed by culture growth or antibody assays. The antibiotics of choice are tetracyclines and macrolides (macrolides not for *M. hominis*). Mycoplasmas show high levels of natural resistance to all betalactam antibiotics.

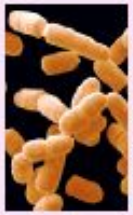
A map of diversity in the human microbiome


Streptococcus dominates the oral cavity with *S. mitis* > 75% in the cheek

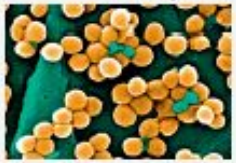
lives on the skin and nose of most people

species characterize different body sites:
C. matruchoti the plaque
C. accolens the nose
C. croppenstedtii the skin






Lactobacillus species (*L. gasseri, L. jensenii, L. crispatus, L. iners*) are predominant but mutually exclusive in the vagina

Staphylococcus epidermidis colonizes external body sites

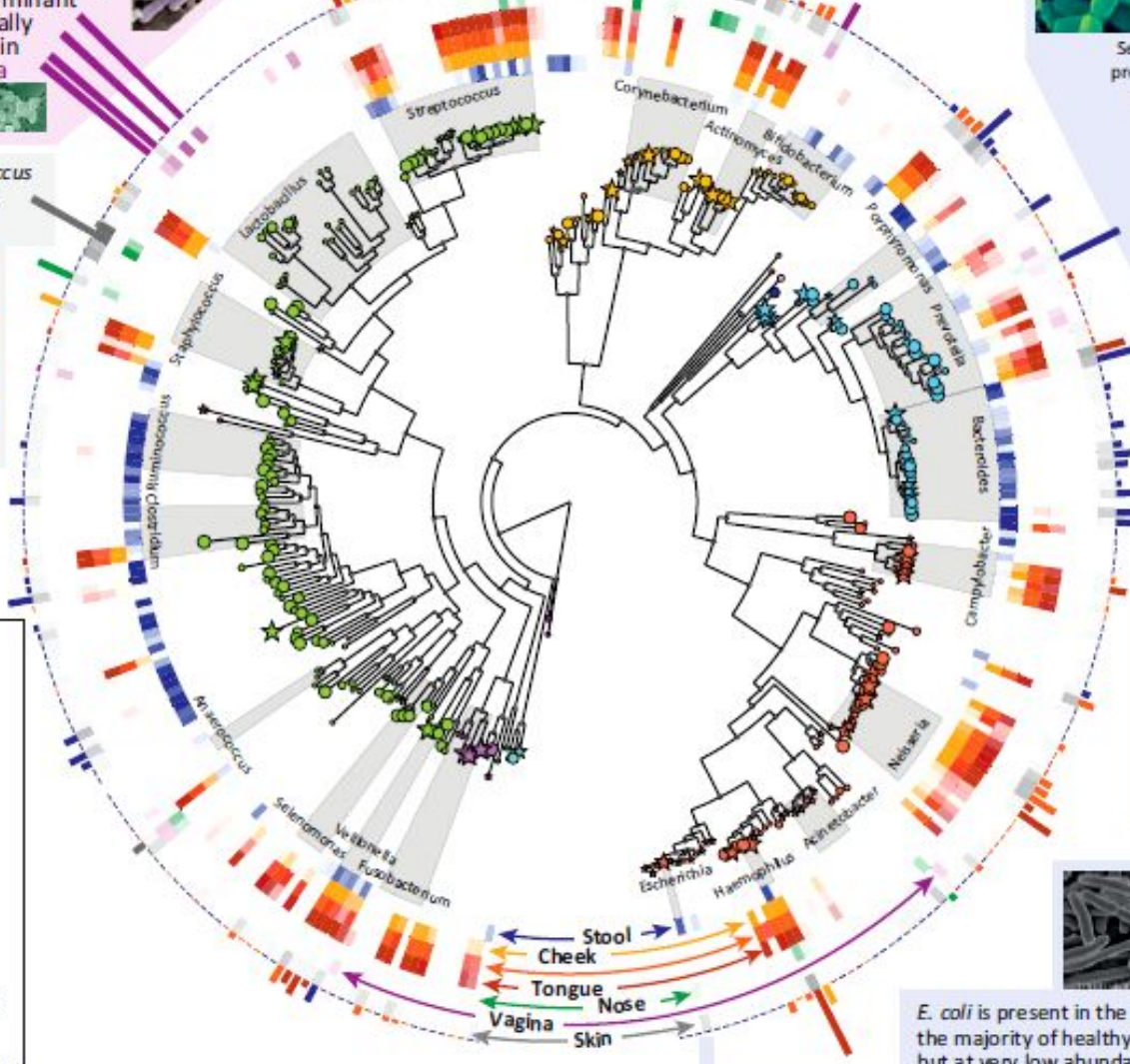
Several *Prevotella* species are present in the gastrointestinal tract. *P. copri* is present in 19% of the subjects and dominates the intestinal flora when present

Microscope from: http://bacmap.wishartlab.com

Bacteroides is the most abundant genus in the gut of almost all healthy subjects

Campylobacter includes opportunistic pathogens, but members live in the oral cavities of most healthy people in the cohort


E. coli is present in the gut of the majority of healthy subjects but at very low abundance



Key:

- Commensal microbes
- Potential pathogens

The four most abundant phyla

- Actinobacteria
- Bacteroidetes
- Firmicutes
- Proteobacteria

Low abundance phyla

- Chloroflexi
- Cyanobacteria
- Euryarchaeota
- Fusobacteria
- Lentisphaerae
- Spirochaetes
- Synergistetes
- Tenericutes
- Thermi
- Verrucomicrobia


You are not alone.

In the average adult are 100 trillion human cells and 1,500 trillion microbes.

At best you are little more than 10%[1] you.

We're all just petri dishes with shoes.