

Mycobacterium

Mycobacterium is a genus of Actinobacteria, given its own family, the Mycobacteriaceae. The genus includes pathogens known to cause serious diseases in mammals, including tuberculosis (*Mycobacterium tuberculosis*) and leprosy (*Mycobacterium leprae*).^[1] The Greek prefix "myco—" means *fungus*, alluding to the way mycobacteria have been observed to grow in a mould-like fashion on the surface of liquids when cultured. ^[2]

Microbiologic characteristics

Mycobacterial cell wall: 1-outer lipids, 2-mycolic acid, 3-polysaccharides (arabinogalactan), 4-peptidoglycan, 5-plasma membrane, 6-lipoarabinomannan (LAM), 7-phosphatidylinositol mannoside, 8-cell wall skeleton

Mycobacteria are aerobic and nonmotile bacteria (except for the species *Mycobacterium marinum*, which has been shown to be motile within macrophages) that are characteristically acid-alcohol-fast.^[1] Mycobacteria do not contain endospores or capsules and are usually considered Gram-positive. A recent paper in PNAS showed sporulation in *Mycobacterium marinum* and perhaps in *M. bovis*.^[3] However, this has been strongly contested by other scientists.^[4] While mycobacteria do not seem to fit the Gram-positive category from an empirical standpoint (i.e., in general, they do not retain the crystal violet stain well), they are classified as an acid-fast Gram-positive bacterium due to their lack of an outer cell membrane. All *Mycobacterium* species share a characteristic cell wall, thicker than in many other bacteria, which is hydrophobic, waxy, and rich in mycolic acids/mycolates. The cell wall consists of the hydrophobic mycolate layer and a peptidoglycan layer held together by a polysaccharide, arabinogalactan. The cell wall makes a substantial contribution to the hardness of this genus. The biosynthetic pathways of cell wall components are potential targets for new drugs for tuberculosis.^[5]

Many *Mycobacterium* species adapt readily to growth on very simple substrates, using ammonia or amino acids as nitrogen sources and glycerol as a carbon source in the presence of mineral salts. Optimum growth temperatures vary widely according to the species and range from 25 °C to over 50 °C.

Some species can be very difficult to culture (i.e. they are fastidious), sometimes taking over two years to develop in culture.^[citation needed] Further, some species also have extremely long reproductive cycles — *M. leprae*, may take more than 20 days to proceed through one division cycle (for

comparison, some *E. coli* strains take only 20 minutes), making laboratory culture a slow process.^[1] In addition, the availability of genetic manipulation techniques still lags far behind that of other bacterial species.^[6]

A natural division occurs between slowly- and rapidly-growing species. Mycobacteria that form colonies clearly visible to the naked eye within seven days on subculture are termed rapid growers, while those requiring longer periods are termed slow growers. Mycobacteria cells are straight or slightly curved rods between 0.2 and 0.6 μm wide by 1.0 and 10 μm long.

Following are the different species of the genus mycobacterium:

- Mycobacterium avium
- Mycobacterium bovis
- Mycobacterium intracellulare
- Mycobacterium smegmatis
- Mycobacterium pneumoniae
- Mycobacterium tuberculosis
- Mycobacterium laprae ect

Pathogenicity

Mycobacteria can colonize their hosts without the hosts showing any adverse signs. For example, billions of people around the world have asymptomatic infections of *M. tuberculosis*.

Mycobacterial infections are notoriously difficult to treat. The organisms are hardy due to their cell wall, which is neither truly Gram negative nor positive. In addition, they are naturally resistant to a number of antibiotics that disrupt cell-wall biosynthesis, such as penicillin. Due to their unique cell wall, they can survive long exposure to acids, alkalis, detergents, oxidative bursts, lysis by complement, and many antibiotics. Most mycobacteria are susceptible to the antibiotics clarithromycin and rifamycin, but antibiotic-resistant strains have emerged.

As with other bacterial pathogens, surface and secreted proteins of *M. tuberculosis* contribute significantly to the virulence of this organism. There is an increasing list of extracytoplasmic proteins proven to have a function in the virulence of *M. tuberculosis*.^[8]

Medical classification

Mycobacteria can be classified into several major groups for purpose of diagnosis and treatment: *M. tuberculosis* complex, which can cause tuberculosis: *M. tuberculosis*, *M. bovis*, *M. africanum*, and *M. microti*; *M. leprae*, which causes Hansen's disease or leprosy; Nontuberculous mycobacteria (NTM) are all the other mycobacteria, which can cause pulmonary disease resembling tuberculosis, lymphadenitis, skin disease, or disseminated disease.

Mycobacterium tuberculosis complex

Mycobacterium tuberculosis are causative agents of human and animal tuberculosis. Species in this complex include:

Mycobacterium avium

Mycobacterium avium causes a disseminated infection but not lung infection, used to be a significant cause of death in AIDS patients.

Mycobacteriophage

Mycobacteria can be infected by Mycobacteriophage, bacterial viruses that may be used in the future to treat tuberculosis and related diseases by phage therapy.

Staphylococcus is group of bacteria that can cause a multitude of diseases.

Staph infections may cause disease due to direct infection or due to the production of toxins by the bacteria. Boils, impetigo, food poisoning, cellulitis, and toxic shock syndrome are all examples of diseases that can be caused by *Staphylococcus*.

Symptoms and signs of a localized staph infection include a collection of pus, such as a boil, furuncle, or abscess. The area is typically tender or painful and may be reddened and swollen.

Methicillin-resistant *Staphylococcus aureus*, known as MRSA, is a type of *Staphylococcus aureus* that is resistant to the antibiotic methicillin and other drugs in this class.

Staph infections are treated with topical, oral, or intravenous antibiotics, depending upon the

The name *Staphylococcus* comes from the Greek *staphyle*, meaning a bunch of grapes, and *kokkos*, meaning berry, and that is what staph bacteria look like under the microscope, like a bunch of grapes or little round berries. (In technical terms, these are gram-positive, facultative anaerobic, usually unencapsulated cocci.)

Over 30 different types of staphylococci can infect humans, but particularly *Staphylococcus aureus* and *Staphylococcus epidermidis*, are opportunistic pathogens and cause significant disease. Staphylococci can be found normally in the nose and on the skin (and less commonly in other locations) of around 25%/ 30% of healthy adults and in 25% of hospital workers. In the majority of cases, the bacteria do not cause disease. However, damage to the skin or other injury may allow the bacteria to overcome the natural protective mechanisms of the body, leading to infection.

people are at greater risk, including newborn infants, breastfeeding women, and people with chronic conditions such as diabetes, cancer, vascular disease, and lung disease. Injecting drug users, those with skin injuries or disorders, intravenous catheters, surgical incisions, and those with a weakened immune system due either to disease or a result of immune suppressing medications all have an increased risk of developing staph infections.

Staph infections are contagious until the infection has resolved. Direct contact with an infected sore or wound, or with personal-care items such as razors, bandages, etc., are common routes of transmission. Casual contact such as kissing or hugging does not pose a great risk for transmission if there is no direct contact with the infected area.

Staphylococcal disease of the skin usually results in a localized collection of pus, known as an abscess, boil, or furuncle, depending upon the exact type of lesion that is present. The affected area symptoms may be

red,
swollen,
tender or painful.

Drainage or pus is common. When staph is in the blood (bacteremia or sepsis), it can cause high fevers, chills, and low blood pressure.

Skin infections (see above) are the most common type of disease produced by *Staphylococcus*. Staph infections of the skin can progress to impetigo (a crusting of the skin) or cellulitis (inflammation of the deeper layers of skin and connective tissue under the skin, leading to swelling and redness of the area). In rare situations, a serious complication known as scalded skin syndrome (see below) can develop. In breastfeeding women, staph can result in mastitis (inflammation of the breast) or in abscess of the breast. Staphylococcal breast abscesses can release bacteria into the mother's milk.

When the bacteria enter the bloodstream and spread to other organs, a number of serious infections can occur. Spread of the organisms to the bloodstream is known as bacteremia or sepsis. Staphylococcal pneumonia predominantly affects people with underlying lung disease and can lead to abscess formation within the lungs. Infection of the heart valves (endocarditis) can lead to heart failure. Spread of staphylococci to the bones can result in severe inflammation of the bones known as osteomyelitis. Septic arthritis occurs when staphylococci infect a joint space. Thrombophlebitis occurs when the bacteria infect a vein. Thrombophlebitis from staphylococci most frequently occurs in hospitalized patients at the site of a venous catheter. When staph bacteria are present in the blood, a condition known as staphylococcal sepsis (widespread infection of the bloodstream) or staphylococcal bacteremia exists. Staphylococcal sepsis is a leading cause of shock and circulatory collapse, leading to death, in people with severe burns over large areas of the body. When untreated, *S. aureus* sepsis carries a mortality (death) rate of over 80%. Although not common, *S. aureus* has been reported as a cause of chorioamnionitis and neonatal sepsis in pregnancy, but group B streptococci are the most common bacterial cause of this life-threatening condition for the fetus.

Staphylococcal infections are contagious and can be transmitted from person to person. Since pus from infected wounds may contain the bacteria, proper hygiene and hand washing is required when caring for staph-infected wounds.

Staphylococcal food poisoning is an illness of the bowels that causes nausea, vomiting, diarrhea, and dehydration. It is caused by eating foods contaminated with toxins produced by *Staphylococcus aureus* rather than a true infection with the bacteria. Symptoms usually develop within one to six hours after eating contaminated food. The illness usually lasts for one to three days and resolves on its own. Patients with this illness are not contagious since toxins are not transmitted from one person to another.

Toxic shock syndrome is an illness caused by toxins secreted by *S. aureus* bacteria growing under conditions in which there is little or no oxygen. Toxic shock syndrome is characterized by the sudden onset of high fever, vomiting, diarrhea, and muscle aches, followed by low blood pressure (hypotension), which can lead to shock and death. There is often a rash resembling sunburn, with peeling of skin. Toxic shock syndrome was originally described and still occurs especially in menstruating women using tampons. Continue Reading

Pseudomonas aeruginosa is a common bacterium that can cause disease in animals, including humans. It is citrate, catalase, and oxidase positive. It is found in soil, water, skin flora, and most man-made environments throughout the world. It thrives not only in normal atmospheres, but also in hypoxic atmospheres, and has, thus, colonized many natural and artificial environments. It uses a wide range of organic material for food; in animals, its versatility enables the organism to infect damaged tissues or those with reduced immunity. The symptoms of such infections are generalized inflammation and sepsis. If such colonizations occur in critical body organs, such as the lungs, the urinary tract, and kidneys, the results can be fatal.[1] Because it thrives on moist surfaces, this bacterium is also found on and in medical equipment, including catheters, causing cross-infections in hospitals and clinics. It is implicated in hot-tub rash. It is also able to decompose hydrocarbons and has been used to break down tarballs and oil from oil spills. [2][3] On 29 April 2013, scientists in Rensselaer Polytechnic Institute, funded by NASA, reported that, during spaceflight inside the International Space Station, *P. aeruginosa* bacteria seem to adapt to the microgravity and the biofilms formed during spaceflight exhibited a column-and-canopy structure that has "not been observed on Earth

An opportunistic, nosocomial pathogen of immunocompromised individuals, *P. aeruginosa* typically infects the airway, urinary tract, burns, wounds, and also causes other blood infections. [18]

Infections	Details and common associations	High-risk groups
Pneumonia	Diffuse bronchopneumonia	Cystic fibrosis patients
Septic shock	Associated with a purple-black skin	<u>Neutropenic</u> patients

lesion ecthyma gangrenosum

Urinary tract infection	Urinary tract catheterization	
Gastrointestinal infection	<u>Necrotising enterocolitis</u> (NEC)	Premature infants and neutropenic cancer patients
Skin and soft tissue infections	Hemorrhage and necrosis	Burns victims and patients with wound infections

It is the most common cause of infections of burn injuries and of the outer ear (otitis externa), and is the most frequent colonizer of medical devices (e.g., catheters). *Pseudomonas* can, in rare circumstances, cause community-acquired pneumonias,[19] as well as ventilator-associated pneumonias, being one of the most common agents isolated in several studies.[20] Pyocyanin is a virulence factor of the bacteria and has been known to cause death in *C. elegans* by oxidative stress. However, research indicates salicylic acid can inhibit pyocyanin production.[21] One in ten hospital-acquired infections are from *Pseudomonas*. Cystic fibrosis patients are also predisposed to *P. aeruginosa* infection of the lungs. *P. aeruginosa* may also be a common cause of "hot-tub rash" (dermatitis), caused by lack of proper, periodic attention to water quality. The most common cause of burn infections is *P. aeruginosa*. *Pseudomonas* is also a common cause of postoperative infection in radial keratotomy surgery patients. The organism is also associated with the skin lesion ecthyma gangrenosum. *P. aeruginosa* is frequently associated with osteomyelitis involving puncture wounds of the foot, believed to result from direct inoculation with *P. aeruginosa* via the foam padding found in tennis shoes, with diabetic patients at a higher risk.

***Klebsiella* infection:**

Members of the *Klebsiella* genus typically express 2 types of antigens on their cell surface. The first is a lipopolysaccharide (O antigen); the other is a capsular polysaccharide (K antigen). Both of these antigens contribute to

pathogenicity. About 77 K antigens and 9 O antigens exist. The structural variability of these antigens forms the basis for classification into various serotypes. The virulence of all serotypes appears to be similar.

Three species in the genus *Klebsiella* are associated with illness in humans: *Klebsiella pneumoniae*, *Klebsiella oxytoca*, and *Klebsiella granulomatis*. Organisms previously known as *Klebsiella ozaenae* and *Klebsiella rhinoscleromatis* are considered nonfermenting subspecies of *K pneumoniae* that have characteristic clinical manifestations. With those exceptions, strains within this genus ferment lactose, most produce highly mucoid colonies on plates because of the production of a luxuriant polysaccharide capsule, and all are nonmotile.^[1]In recent years, klebsiellae have become important pathogens in nosocomial infections.^[2]See the image below.

Pathogenesis:

Host defense against bacterial invasion depends on phagocytosis by polymorphonuclear granulocytes and the bactericidal effect of serum, mediated in large part by complement proteins. Both classic-pathway and alternate-pathway complement activation have been described, but the latter, which does not require the presence of immunoglobulins directed against bacterial antigens, appears to be the more active pathway in *K pneumoniae* infections.

Recent data from preclinical studies suggest a role for neutrophil myeloperoxidase and lipopolysaccharide-binding protein in host defense against *K pneumoniae* infection. Neutrophil myeloperoxidase is thought to mediate oxidative inactivation of elastase, an enzyme implicated in the pathogenesis of various tissue-destroying diseases. Lipopolysaccharide-binding protein facilitates transfer of bacterial cell wall components to inflammatory cells. Investigators showed higher rates of infection in experimental mice deficient in the genes that control expression of these 2 agents.

The bacteria overcome innate host immunity through several means. They possess a polysaccharide capsule, which is the main determinant of their pathogenicity. The capsule is composed of complex acidic polysaccharides. Its massive layer protects the bacterium from phagocytosis by polymorphonuclear granulocytes. In addition, the capsule prevents bacterial death caused by bactericidal serum factors. This is accomplished mainly by inhibiting the activation or uptake of complement components, especially C3b. The bacteria also produce multiple adhesins. These may be fimbrial or

nonfimbrial, each with distinct receptor specificity. These help the microorganism to adhere to host cells, which is critical to the infectious process.

Lipopolysaccharides (LPS) are another bacterial pathogenicity factor. They are able to activate complement, which causes selective deposition of C3b onto LPS molecules at sites distant from the bacterial cell membrane. This inhibits the formation of the membrane attack complex (C5b-C9), which prevents membrane damage and bacterial cell death.

Availability of iron increases host susceptibility to *K pneumoniae* infection. Bacteria are able to compete effectively for iron bound to host proteins because of the secretion of high-affinity, low molecular weight iron chelators known as siderophores. This is necessary because most host iron is bound to intracellular and extracellular proteins. In order to deprive bacteria of iron, the host also secretes iron-binding proteins.

Clostridium is a genus of Gram-positive bacteria belonging to the Firmicutes. They are obligate anaerobes capable of producing endospores.^{[1][2]} Individual cells are rod-shaped, which gives them their name, from the Greek *kloster* (κλωστήρ) or spindle. These characteristics traditionally defined the genus; but many species originally classified as *Clostridium* have been reclassified in other genera.

Clostridia are relatively large, anaerobic, sporeforming, rod-shaped, gram-positive organisms. They are found either as living cells (vegetative forms) or as dormant spores. Their natural habitats are soils and intestinal tracts of animals, including people. Dormant spores of several clostridial species have been found in healthy muscular tissue of horses and cows. The endospores are oval, sometimes spherical, and are located centrally, subterminally, or terminally. The vegetative forms of clostridia in tissue fluids of infected animals occur singly, in pairs, or rarely in chains. Differentiation of the various pathogenic and related species is based on cultural characteristics, spore shape and position, biochemical reactions, and the antigenic specificity of toxins or surface antigens. The genomes of many clostridia have been sequenced and are available online. Pathogenic strains or their toxins may be acquired by susceptible animals by either wound contamination or ingestion. Diseases thus produced are a constant threat to successful livestock production in many parts of the world.

Clostridial diseases can be divided into two categories: 1) those in which the organisms actively invade or when locally dormant spores are activated and reproduce in the tissues of the host, with the production of toxins that enhance the spread of infection (the gas-gangrene group; the clostridial cellulitides group); and 2) those characterized by toxemia resulting from the absorption of toxins produced by organisms within the digestive system (the enterotoxemias), in devitalized tissue (tetanus), or in food or carrion outside the body (botulism). Clostridial diseases are not spread from animal to animal.

Salmonella: Pl refer Text Book of Microbiology by Michael J Pelczar, Jr. Pg No.682-685