A Comprehensive Review of Various Mycobacterium Species

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Abstract

Mycobacterium species are pathogenic to animals and humans. These Mycobacterium species are gram-positive, non-motile, non-spores forming rod-shaped bacteria. The Mycobacterium genus comprises over 190 species, such as Mycobacterium leprae (M. leprae), tuberculosis (TB), Mycobacterium bovis (M.bovis) , and many more. These different species of mycobacterium affect the other organs in the body parts. M. leprae mainly invades and affects the skin and Schwan cells.M. leprae is gram-positive and multiplies slowly for 12 to 13 days. Different drug regimens are given to treat leprosy drugs, such as dapsone, rifampicin, and clofazimine. Mycobacterium bovis causes TB in animals and humans. In humans, M.bovis lesions are primarily extrapulmonary and transmitted to humans by ingestion of infected milk and milk products. The treatment of M.bovis involves using antibiotics together, including rifampicin, isoniazid, and ethambutol. Mycobacterium tuberculosis (M. tuberculosis) causes TB, affects the lungs, and attacks other body parts such as kidneys, brain, spine, etc. It is spread through tiny droplets of infected persons. TB can be fatal if not treated; different drug regimens are provided for 3 to 6 months. The most common medication includes isoniazid, rifampicin, pyrazinamide and ethambutol. This review shows other mycobacterium species, their etiology, pathogenesis, and treatment.

Keywords: Mycobacterium tuberculosis, TB, Mycobacterium bovis, Mycobacterium leprae, pathogenesis, etiology, treatment.

INTRODUCTION

The genus Mycobacterium comprises species, some of which are harmful to humans and other animals. This is evidenced by a rabbit model of tuberculosis [1]. Different strains of M. tuberculosis can cause varying spectrums of disease. The genus is divided according to the pigment production and growth rate of each species, and while the majority of species are non- pathogenic, some are harmful and have a characteristic complex cell wall that helps them evade host defenses [2]. Clinical strains of M. tuberculosis exhibit differential lipid metabolism- associated transcriptome changes in in vitro models of cholesterol and infection [3]. Mycobacteria are Gram-positive, catalase-positive, non-motile, non-spore-forming rod-shaped bacteria [2], shown in Fig no.1.Clinically significant tasks include differentiating between species of Mycobacterium and determining antibiotic resistance. High-density DNA probe arrays have been employed to test for rifampin resistance and identify species [4]. Different Mycobacterium TB strains can result in a range of illness manifestations. In the rabbit model of tuberculosis, a study comparing M. tuberculosis strains CDC1551, H37Rv, and Erdman discovered several noteworthy variations in each strain's capacity to generate lung disease, cause mortality, and be virulent [1]. According to a different study, clinical strains of M. tuberculosis display unique behavior, which could affect how well they metabolize lipids, particularly cholesterol, and jeopardize their virulence [3].

Fig no.1 Mycobacterium

Furthermore, the local ecology of mycobacterial genotypes or strains adds to geographic variance in tuberculosis disease, according to a systematic review and meta-analysis of global variation in bacterial strains that cause the disease [5]. Additionally, it was discovered that strains of the Beijing family of M. tuberculosis strains from the present sublineages are more likely than strains from the ancient Sublineages to exhibit enhanced virulence [6]. Mycobacterium is a phylum Actinomycetota genus with over 190 species with its own family,

Mycobacteriaceae. Pathogens in this genus have been linked to severe diseases in mammals, including tuberculosis (M. tuberculosis) and leprosy (M. leprae) in humans [2]. Mycobacteria are slender, non-spore-forming, rod-shaped bacteria that live in soil and water [4]. They are Gram-positive, catalase-positive rod-shaped bacteria that are non-motile and do not form spores [2]. Because of the increasing number of mycobacterial infections, it is critical to identify mycobacteria at the species level [4] quickly. Mycobacterium, the only genus in the Mycobacteriaceae family, currently has over 170 recognized species [7].

DIFFERENT SPECIES OF MYCOBACTERIUM

MYCOBACTERIUM LEPRAE

Skin macrophages and Schwann cells are the primary targets of infection and invasion by the pathogenic bacterium Mycobacterium leprae, which is pleomorphic, acid-fast, non-sporing, and non-motile [8]. This chronic infectious disease primarily affects the skin and peripheral nerves and is the causative agent of leprosy [9]. The taxonomy classification of M. leprae, an obligatory intracellular organism, includes the class Schizomycetes, order Actinomycetales, Genus Mycobacterium, and family [10]. A three-layer lipoprotein membrane surrounds the bacteria and is slightly curved, measuring 1 to 8µm in length and 0.3 to 0.5µm in diameter, shown in Fig. no.2, M., metabolites, ribosomes, soluble proteins, DNA, and soluble RNA makeup Mycobacterium leprae. Gram staining confirms that Leprae is an aerobic bacillus with round ends and gram-positive parallel sides. Nonetheless, carbon fuchsin was traditionally used to stain it using the Kiehl-Nielsen stain [11].

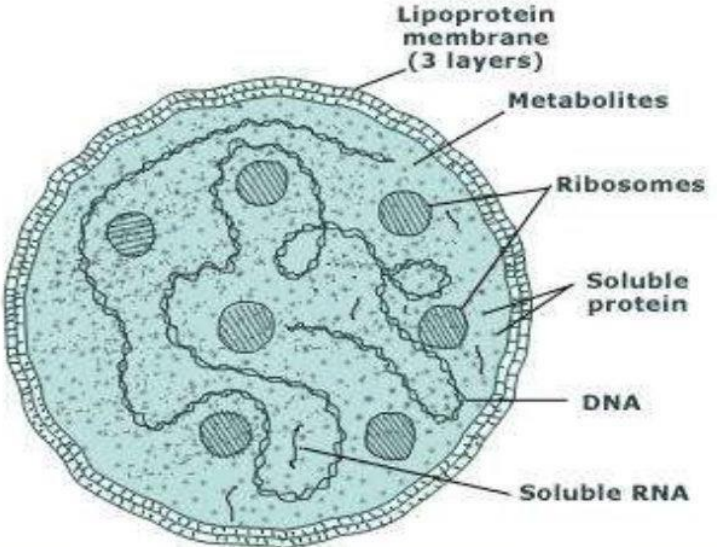

Fig no. 2 Mycobacterium leprae

ETIOLOGY

Leprosy, sometimes referred to as Hansen's disease, is a persistent infectious illness brought on by the rod-shaped, acid-fast Mycobacterium Leprae [9]. This bacterium belongs to the Mycobacterium leprae complex, which also comprises Mycobacterium lepromatosis. These two mycobacteria are obligatory intracellular organisms that share many characteristics and cause the same clinical condition. M. Leprae is a slow-growing, gram-positive, acid-fast bacillus that takes 12 to 13 days to multiply. It has less than half of the TB function genes and cannot be grown in artificial media. Mycobacterium leprae has many pseudogenes in its genetic makeup and lacks several genes for necessary metabolic pathway enzymes [12]. Despite its small genome, the bacterium's generation time is unusually long [13]. It may impact the nose lining, skin, eyes, and nerves [14]. Leprosy is spread by respiratory droplets from the mouth and nose, and months of close contact with an untreated leprosy patient are required for infection. When patients start treatment, they can no longer spread the illness through casual contact [9].

PATHOGENESIS

Leprosy is a chronic infectious disease that primarily affects humans' skin and peripheral nerves. It is caused by Mycobacterium leprae. The capacity of M. leprae to infect and invade skin macrophages and Schwann cells in peripheral nerves contributes to the pathogenesis of the infection, which can become chronic. To live and avoid immune surveillance systems for long-term parasitization, M. leprae is an obligatory intracellular organism that parasitizes host cells. It does this by carrying out various metabolic processes, including lipid metabolism, which is crucial for the synthesis of cell walls. Leprosy is a chronic infectious disease that primarily affects humans' skin and peripheral nerves. It is caused by Mycobacterium leprae. The capacity of M. leprae to infect and invade skin macrophages and Schwann cells in peripheral nerves contributes to the pathogenesis of the infection, which can become chronic. To live and avoid immune surveillance systems for long-term parasitization, M. leprae is an obligatory intracellular organism that parasitizes host cells. It does this by carrying out various metabolic processes, including lipid metabolism, which is crucial for the synthesis of cell walls. The illness is characterized by progressive debilitation, disfiguring skin sores, and nerve damage. Peripheral nerve system Schwann cells are infected by M. leprae, which causes leprosy in different forms (type 1, 2, and 3) and a range of leprosy reactions (indeterminate, tuberculoid, borderline tuberculoid, mid-borderline, borderline lepromatous, and lepromatous) [15]. Mycobacterium leprae's capacity to infiltrate and persist within host cells is a pathogenic

mechanism that results in persistent infection and the telltale signs and symptoms of leprosy [8].

TREATMENT

Combinations of antibiotics are used to treat leprosy, also referred to as Hansen's disease. Known as multidrug therapy (MDT), the currently advised course of treatment consists of three medications: clofazimine, rifampicin, and dapsone [9]. Two or three antibiotics are usually used simultaneously to stop the bacteria from becoming resistant to antibiotics [14]. The course of treatment lasts six months for paucibacillary leprosy patients, while for multibacillary leprosy cases, it lasts twelve months. Early diagnosis and prompt treatment can help prevent disabilities, and multidrug therapy cures the patient and kills the pathogen [9]. Finishing the entire antibiotic course is critical because stopping early could allow the bacteria to grow back. Even though the disease can be cured and prevented from worsening, the treatment cannot undone nerve damage or physical deformity that may have already happened before the diagnosis. Depending on the type of leprosy, multidrug therapy using antibiotics, usually dapsone, rifampicin, and clofazimine, is used to treat Mycobacterium leprae infections. This treatment can last anywhere from six to twelve months. Finishing the entire course of antibiotics is essential to ensure a successful outcome and stop the bacteria from developing antibiotic resistance [14]. Chaulmoogra oil was widely used to treat leprosy worldwide until the 1940s [23]. Aspirin, digitalis, paracetamol, quinine, and vinblastine are some other herbal medications and compounds utilized in leprosy research and treatment; these substances are derived from the natural compounds of medicinal plants [23].

MYCOBACTERIUM BOVIS

Mycobacterium bovis is responsible for tuberculosis (TB) in humans and animals, such as bison, deer, and cattle. It can cause tuberculosis (TB), which affects the lungs, lymph nodes, and other body parts in humans. It is most frequently found in cattle and other animals like bison, elk, and deer [16]. Human *M. bovis* lesions have historically been extrapulmonary or outside the lungs and are contracted by consuming infected milk [17]. Indirect wound contact, such as that which might happen during hunting or slaughter, or breathing in the bacteria from animals exhaling *M. bovis* infected air can also result in infection. Though direct airborne transmission from animals to humans is thought to be uncommon, *M. bovis,* shown in Fig no. 3, can be directly transferred from person to person through coughing or sneezing infected individuals. Eating unpasteurized dairy products is the most frequently mentioned cause of *M. bovis* infection in humans [16].

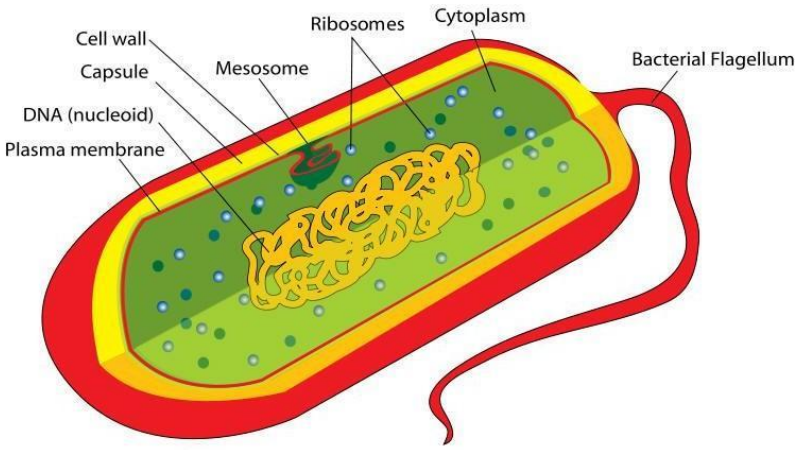

Fig no.3. Cell contents of *Mycobacterium bovis*

ETIOLOGY

Both humans and animals can contract tuberculosis (TB) from the bacteria *Mycobacterium bovis*. Cattle and other animals like deer, elk, and bison are the most common hosts of this condition. *M. bovis* can cause tuberculosis (TB) in humans, which can impact the lymph nodes, lungs, and other body parts. However, not everyone infected with *M. bovis* develops symptoms; some may have latent TB infection (LTBI). Humans can contract the bacteria by breathing in the air exhaled by infected animals, consuming contaminated, raw dairy products, or coming into direct contact with a wound while hunting or slaughtering animals. Though direct airborne transmission from animals to humans is thought to be uncommon, *M. bovis* can be directly transmitted from person to person through coughing or sneezing infected individuals [16]. Additionally, *Mycobacterium bovis* can cause latent TB infection (LTBI), also referred to as asymptomatic TB, which may not exhibit any symptoms. Infection with Mycobacterium bovis can cause symptoms like fever, sweating at night, weight loss, coughing, swelling of the abdominal and lymph nodes, and exhaustion. The bacteria is a zoonotic illness that can spread from humans to animals and infect both. Medical professionals often cannot figure out which strain of Mycobacterium is causing the infection, so antibiotics like rifampicin, isoniazid, and ethambutol are often used to treat M. bovis infections [18]. Although consuming contaminated milk was once the primary way that *M. bovis* infection spread to humans, tuberculosis caused by *M. bovis* infection is now relatively uncommon. For those particularly vulnerable, like those who work in abattoirs, it is still a cause for concern [17].

PATHOGENESIS

Both humans and animals can contract tuberculosis (TB) from the bacteria *Mycobacterium bovis*. Infections in animals can cause lesions, necrosis, and caseation, which are occasionally linked to mineralization [19]. Humans can contract the disease by direct contact with infected animals, consuming contaminated milk, or breathing in bacteria inhaled by infected animals' exhaled air. The condition can affect many body parts, including the lungs and lymph nodes. Some people may have a latent TB infection without symptoms, and not al*l* infections develop into TB disease [16]. Numerous elements, including the bacterial strain, the infection route, and the infectious dose, are involved in the pathogenesis of *M. bovis* infection [19]. Agricultural workers may contract *M. bovis* by breathing in cough spray from infected cattle, as the disease primarily affects the respiratory system in cattle [20]. Controlling the disease's spread in humans and animals requires understanding the pathogenesis of *M. bovis* infection [19].

TREATMENT

Mycobacterium bovis (M. bovis) is responsible for human tuberculosis (TB). Treatment for *M. bovis* and *M. tuberculosis* is comparable because medical professionals may only sometimes be able to distinguish between the two. Pyrazinamide, an antibiotic that is commonly used to treat tuberculosis, usually causes resistance in *M. bovis*. However, since TB disease is treated with a combination of several antibiotics, resistance to pyrazinamide typically does not result in treatment problems. For *M. bovis*, the most widely advised treatment plan consists of rifampicin, isoniazid, and ethambutol; excluding pyrazinamide typically extends the treatment period to nine months [21]. Up To Date recommends using an appropriate dosage of an adequate four-drug regimen of isoniazid, rifampicin, ethambutol, and ofloxacin [16]. This is consistent with the course of therapy listed in the search results. It's crucial to remember that consuming tainted, raw dairy products is a daily way for *M. bovis* to spread, so avoiding them is advised to prevent infection [22]. The potential use of several medicinal plants in treating *Mycobacterium bovis*, the bacterium that causes tuberculosis, has been investigated. A few of these plants have hepatoprotective qualities that may lessen the harm that anti-TB medications can do to the liver [24]. Some plants, like Eucalyptus camaldulensis, Artemisia abyssinica leaves, Croton macrostachyus, and Ocimum basilicum seeds, have roots that can kill strains of M. tuberculosis and M. bovis [25]. Researchers have also examined how phytochemicals like coumarin, allicin, andrographolide, glabridin, and vaccine acetate can kill mycobacteria [24].

MYCOBACTERIUM TUBERCULOSIS

The causative agent of tuberculosis (TB), *Mycobacterium tuberculosis,* also called Koch's bacillus, is a pathogenic bacterium in the Mycobacteriaceae family [26]. Two million people die from tuberculosis (TB) every year, making it one of the oldest known human diseases and a significant cause of death [27]. The bacteria typically attack the lungs but can also damage the kidneys, spine, and brain [26]. M. tb. is renowned for its capacity for adaptation and host-specific survival. While the disease is in its latency phase, the bacteria use different effector proteins to hide from the host's immune system and live in granulomas, which are complex, well-organized, resistant cell structures the host makes in response to a persistent infection. When the host's immune system is weakened, or the bacteria are exposed to ideal circumstances, they can lie dormant for years before reactivating and spreading illness [29]. TB disease can progress in several ways, mainly depending on the host's immune system and the particular strain of *M. tuberculosis* that is causing the illness. The pathogen may contribute to the development of the disease because certain strains of *M. tb* are more virulent than others. New vaccinations and medications are required to combat the ongoing global TB epidemic [27]. However, as illustrated in Fig. 4, it was conventionally stained with carbofurfur (Fushsin) in the Ziehl-Neelsen stain [11].

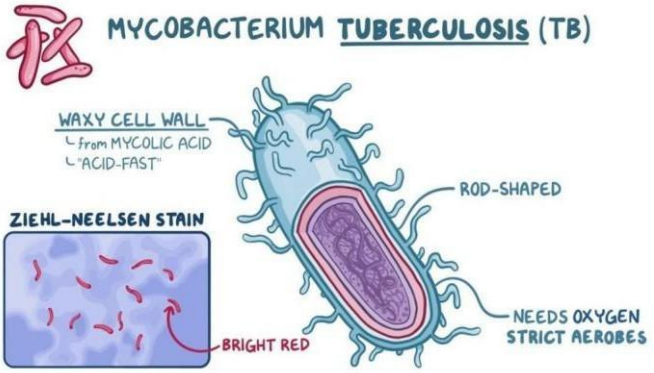

Fig no.4. Ziehl- Neelsen Stian of *M.TB*

ETIOLOGY

Mycobacterium tuberculosis is the bacterium that causes tuberculosis (TB). Although TB bacteria typically affect the lungs, they can also affect the kidney, spine, and brain [30]. When a person with tuberculosis coughs, sneezes or sings, microscopic droplets containing the germs are released into the air and can spread the illness. The germs can then enter the lungs of another

person who inhales the droplets [31]. Mycobacterium tuberculosis primarily affects the lungs but can also impact the respiratory, gastrointestinal, lymphoreticular, skin, central nervous, musculoskeletal, reproductive, and liver systems [27].

PATHOGENESIS

The bacterium *Mycobacterium tuberculosis* is the source of tuberculosis (TB), a chronic infectious disease that mainly affects the lungs but can also spread to the bone and central nervous system [27]. A dynamic interaction between the pathogen and the host characterizes tuberculosis (TB) pathogenesis, with multiple factors impacting the disease's progression [28]. Inhaling infectious droplet nuclei carrying live bacilli is how tuberculosis is spread [30]. Numerous symptoms, such as fever, weight loss, and cough in the event of pulmonary reactivation, can indicate the illness [26].

TREATMENT

Tuberculosis is an infection that usually affects the lungs and can be treated with antibiotics, but it can be severe if not treated. The primary treatment for tuberculosis is to take antibiotics for at least six months. If you have TB but do not have symptoms (latent TB), you usually need to take antibiotics for 3 to 6 months. If TB has spread to your brain, spinal cord, or the area around your heart, you may also need to take steroid medicine for a few weeks [27]. Most latent TB infections are treated for three or four months, while active TB disease may be treated for four, six, or nine months. Common medications used to treat tuberculosis include isoniazid, rifampin, rifabutin, rifapentine, pyrazinamide, and ethambutol. You might be prescribed different medicines if your illness is causing complications or if you have drug-resistant tuberculosis. Even if you feel better, continuing your antibiotics as defined is crucial until the entire course is finished. TB may recur if you discontinue treatment too soon [29]. Traditional medicine has employed several medicinal plants to treat tuberculosis (TB). For instance, wrapping them in newspapers and smoking them twice or three times a day has been used to treat tuberculosis (TB) in *Artemisia afra*, Citrus lemon, and Mentha sp. [32]. Furthermore, it has been discovered that a few medicinal plants possess hepatoprotective qualities, meaning they can be utilized as a complementary therapy to stop the liver damage brought on by anti- TB medications. Aswagandha, *Acalypha indica, Adhatoda vasica*, *Allium sativum, Andrographis isease te*, and *Cassia sophera* are a few examples of these plants [24]. It's crucial to remember that these herbal treatments should only be used as an adjuvant therapy to minimize side effects rather than as a replacement for prescription anti-TB medications [The

review of *Mycobacterium* reveals that the different species of *Mycobacterium* can cause various diseases in both animals and humans. There are 170 recognized species of *Mycobacterium*, the only genus in the family Mycobacteriaceae. Different species infect different parts of the organ and the body, such as *Mycobacterium bovis, which* leads to chronic and cavitary diseases; *Mycobacterium leprae, which causes* skin lesions; and *Mycobacterium tuberculosis, which* infects the lungs. Rifampicin, Isoniazid, Ethambutol, Pyrazinamide, and other antibiotics are just a few of the drug regimens available to treat diseases brought on by Mycobacterium. There are many herbal drugs also present to treat Mycobacterium infection, such as Chaulmoogra oil, Vinblastine, Allium sativum, Citrus lemon, Ashwagandha, seeds of Ocimum basilicum, Artemisia, Mentha, etc., and the use of these herbal drugs can be used as adjuvants to drug therapies.

CONCLUSION:

The review of Mycobacterium reveals that the different species of Mycobacterium can cause various disease spectrum in both animals and humans. Currently there are 170 recognised species of in Mycobacterium the only genus in the family Mycobacteriaceae. Different species infect the different parts of organ and body such as Mycobacterium bovis leads to chronic and cacitary isease, Mycobacterium leprae cause skin lesions and Mycobacterium tuberculosis infects the lungs .Different drug regimens are provided to treat diseases caused by Mycobacterium such as Rifampicin,Isoniazid, Ethambutol and Pyrozinamide along with other antibiotic.There are many herbal drugs also present to treat Mycobacterium infection such as Chaulmoogra oil,Vinblastine,Allium sativum,Citrus lemon,Ashwagandha,seeds of Ocimum basilicum, Artemisia ,Mentha etc.and use of these herbal drugs can be used adjuvant to drug therapies .

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