Leprosy

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**Etiologic agent** | Leprosy is a chronic disease caused by a rod shaped bacillus, *Mycobacterium leprae*

**Taxonomy** | Domain = Bacteria | Phylum = Actinobacteria | Class = Actinobacteridae | Order = Actinomycetales | Suborder = Corynebacterineae | Family = Mycobacteriaceae | Genus = Mycobacterium | Species = M. leprae | (3).

**Transmission** | It is not known exactly how Hansen’s disease spreads between people. Scientists currently think it may happen when a person with Hansen’s disease coughs or sneezes, and a healthy person breathes in the droplets containing the bacteria. Prolonged, close contact with someone with untreated leprosy over many months is needed to catch the disease (6). Scientists also know that *M. leprae* has been found among wild armadillos in the Southern United States (Texas, Louisiana, Mississippi, Arkansas, Georgia and Florida) suggesting that human contact with infected armadillos might lead to infection (1). Hansen’s disease is also not passed on from a mother to her unborn baby during pregnancy and it is also not spread through sexual contact. Due to the slow-growing nature of the bacteria and the long time it takes to develop signs of the disease, it is often very difficult to find the source of infection (4).

**Reservoirs** | Armadillos can harbor the bacteria, but are not seen as a threat to human contraction of the disease. In addition, insects could be possible carriers of *Mycobacterium leprae* but this is unclear In humans, the bacteria is thought to be passed through skin and nasal mucosa.

**General Characteristics** | Leprosy (Hansen's Disease) is a chronic infectious disease that primarily affects the peripheral nerves, skin, upper respiratory tract, eyes, and nasal mucosa (lining of the nose).

**Key Tests for Identification** | The most complete test for the diagnosis of leprosy is a full-thickness skin biopsy sample obtained from the advancing margin of an active lesion, fixed in neutral buffered formalin, embedded in paraffin, and examined by an experienced pathologist. The primary characteristics to be observed are histological patterns of the host response, the involvement of cutaneous nerves, and the identification of acid-fast bacilli. In tuberculoid lesions, where bacilli may be rare and difficult to find, the differential diagnosis of the granulomatous response commonly includes cutaneous tuberculosis, sarcoidosis, and granuloma annulare. No serologic tests are available for the routine laboratory diagnosis of Hansen's disease, and no laboratories in the United States perform such assays routinely. Diagnosis is typically made upon recognition of acid-fast bacilli in a skin biopsy of a lesion (3). As some patients have few lesions, scientists are looking for immunodiagnostic tests to explain neuropathy and other symptoms that may be unaccompanied by lesions (3). For instance, tuberculoid leprosy (TT) often produces few lesions, so the disease can misdiagnosed. In
lepromatous (LL) cases, biopsy should be made from a nerve cell to rule out alternative diagnoses which might show similar symptoms and bacilli in tissue (3)

**Signs and Symptoms** | The main symptoms of leprosy include muscle weakness, numbness in the hands, arms, feet, and legs. But the most common sign of leprosy are skin lesions (6). The skin lesions have decreased sensation to touch, temperature, or pain. They don’t heal after several weeks and are lighter than your normal skin tone (2).

**Historical information** | Leprosy is an extremely old disease, described in the literature of many ancient civilizations. People afflicted with leprosy have often been ostracized by their communities and families throughout history. Leprosy was treated multiple ways in the past but the first true breakthrough occurred in the 1940s with the development of the drug dapsone. The duration of the treatment was many years, often a lifetime, making it difficult for patients to adhere to it. In the 1960s, *M. leprae* started to develop resistance to dapsone, the world’s only known anti-leprosy drug at that time. In the early 1960s, rifampicin and clofazimine were discovered and subsequently added to the treatment regimen, which was later labelled as multidrug therapy (MDT). In 1981, a WHO Study Group recommended MDT. MDT consists of 2 or 3 drugs: dapsone and rifampicin for all patients, with clofazimine added for multibacillary disease. This drug combination kills the pathogen and cures the patient.

**Virulence Factors** | *Mycobacterium leprae* can conserve some iron utilization genes to help the pathogen acquire nutrients for growth. NRAMP proteins, which are coded by one particular conserved gene, allows transportation of iron into the macrophage for survival (3). Bacteria of the *Mycobacterium* Genus are defined by their waxy exterior coat. In *Mycobacterium leprae*, the exterior coat allows for intake into the macrophage and into some dendritic cells, in which it can survive. *Mycobacterium leprae* survives and replicates in macrophages. *Mycobacterium leprae* also has the ability to survive emission of reactive oxygen species. However the major goal of *Mycobacterium leprae* is the Schwann cell. The bacteria seeks the temperature that corresponds to the peripheral nerves (3). To access the target cells, *Mycobacterium leprae* gets into the lymphatic system and the blood vessels. Once in the area, *Mycobacterium leprae* binds to the Schwann cell via laminin-binding protein. The infection then remains localized to the peripheral nervous system by rolling and binding to exposed Schwann cells (3).

**Control/Treatment** | Leprosy is curable with multidrug therapy (MDT). Hansen’s disease is treated with a combination of antibiotics. Typically, 2 or 3 antibiotics are used at the same time. These are dapsone with rifampicin, and clofazimine is added for some types of the disease. This is called multidrug therapy. This strategy helps prevent the development of antibiotic resistance by the bacteria, which may otherwise occur due to length of the treatment (6). Treatment usually lasts between one to two years. The illness can be cured if treatment is completed as prescribed. If you are treated for Hansen’s disease, it's important to take the correct antibiotics when prescribed and alert a doctor to any physical change to the lesions or nerves. If left untreated, the nerve damage can occur leading to paralysis and crippling of hands and feet. There is also a chance of the body reabsorbing the affected digits over time, resulting in the apparent loss of toes and fingers (6). Corneal ulcers or blindness can also occur if facial nerves are affected, due to loss of sensation of the cornea (outside) of the eye. Other signs of advanced leprosy may include loss of eyebrows and saddle-nose deformity resulting from damage to the nasal septum (6). Antibiotics used during the treatment are designed to kill the bacteria that cause leprosy. But
while the treatment can cure the disease and prevent it from getting worse, it does not reverse any nerve damage or physical disfigurement that may have already occurred. Therefore early diagnosis is very important to prevent permanent nerve (6).

**Prevention/Vaccine info/New Trials** | A highly effective vaccine has yet to be developed, and extensive laboratory efforts have not yet produced any practical tools for early diagnosis of the disease (1). WHO has provide free treatment to all leprosy patients in the world. Free MTD was initially funded by The Nippon Foundation, and since 2000 it is donated through an agreement with Novartis who recently committed to extend the donation to at least 2020 (5). In 2016 WHO has launched the "Global Leprosy Strategy 2016–2020: Accelerating towards a leprosy-free world" – which aims to reinvigorate efforts for leprosy control and to avoid disabilities, especially among children affected by the disease in endemic countries. The mechanism by which *M. leprae* is able to elicit the entire range of human cellular immune responses has still not been explained. Most clinical immunological inquiries have focused on the “immunologic defect” of lepromatous patients (1). Priorities for research in leprosy today include genetic probes for molecular epidemiology, and new immunologic tests for early detection of leprosy before nerve damage occurs. The goals are to provide evidence on routes of transmission and incubation periods and to develop new tools to prevent and, ultimately, eradicate leprosy (1).

**Local Cases/Outbreaks** | No large outbreaks in the US. It is hard to contract leprosy because approximately 95% of the population is immune to the disease (6).

**Global Cases/Outbreaks** | According to official reports received from 138 countries from all WHO regions, the global registered prevalence of leprosy at the end of 2015 was 176,176 cases (0.18 cases per 10,000 people). The number of new cases reported globally in 2015 was 211,973 (0.21 new cases per 10,000 people). The number of new cases indicates the degree of continued transmission of infection. Global statistics show that 203,600 (96%) of new leprosy cases were reported from 22 priority countries (5). Overall, the risk of getting Hansen’s disease for any adult around the world is very low. That’s because more than 95% of all people have natural immunity to the disease.

**References**


