

Leprosy

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Leprosy is a chronic disease caused by the bacillus *Mycobacterium leprae*, and is closely related to tuberculosis. Leprosy remains a leading infectious cause of disability in the world today; untreated it can lead to progressive and permanent damage in the nerves, skin, limbs and eyes. It is believed to be transmitted primarily from person-to-person via nasal droplets. Although prevalence has reduced dramatically following the introduction of multidrug therapy, there are still approximately 250 000 new cases of leprosy every year, most of which occur in 17 countries including India (accounting for about half of all new cases), Brazil, Ethiopia, China and Indonesia.¹

Leprosy is a relatively uncommon disease in Australia and New South Wales (NSW), with just 24 notifications in the state over the last 10 years. Most notifications are in migrants from countries where leprosy is endemic, with some locally acquired cases in Aboriginal communities.²

Leprosy is categorised according to the Ridley Jopling classification. On one end of the spectrum are patients with tuberculoid disease who have good cell-mediated immunity with few skin lesions and low bacterial load. At the other extreme are patients with lepromatous leprosy who have poor immunity and multiple lesions with high bacterial load. Between these two classifications are the borderline leprosy types, in which the immune response is thought to be unstable.

The World Health Organization (WHO) has introduced a simple classification that uses the number of skin lesions to classify disease as paucibacillary (up to five skin lesions) or multibacillary (more than five skin lesions). Multibacillary leprosy roughly correlates with the lepromatous side of the Ridley Jopling spectrum, and is both more infectious and takes longer to treat than other forms of the disease.³

Diagnosis

Diagnosis is based on clinical suspicion of leprosy as a cause of skin lesions, or peripheral nerve thickening or impairment in a person from a leprosy-endemic region. Confirmation is by demonstration of acid-fast bacilli in slit skin smears or biopsies. Other symptoms include numbness or tingling in the hands and feet and swelling

of the face or earlobes. Nerve damage can take place before, during or after treatment and may cause long-term disability and disfigurement. Patients can suffer immune-mediated reversal reactions during or after treatment, including spontaneous increase in inflammation in skin and nerve lesions, and erythema nodosum leprosum. Reversal reactions occur in approximately 30% of multibacillary patients and require treatment with corticosteroids for at least 5 months.

Most leprosy cases can be effectively treated with a multi-drug regimen for 6 (paucibacillary cases) or 12 (multibacillary cases) months. Patients are considered to be no longer infectious once they start multidrug therapy, and relapse rates are as low as 1% once treatment is complete.³

Prevention and control

The Bacille Calmette-Guérin vaccine given to prevent tuberculosis has a protective efficacy against leprosy of between 28 and 60%, and vaccine administration to neonates has contributed to a decrease in leprosy prevalence worldwide.

Leprosy is not highly infectious and the absolute risk of transmission has been estimated at 1% in endemic settings. Risk factors include proximity to a case for a prolonged period, for example through being in the same household. A single dose of rifampicin given to contacts of multibacillary patients has been found to reduce the incidence of leprosy in a 2-year period.⁴

Management of leprosy cases is sometimes complicated by the existing lack of clarity around the use of chemoprophylaxis in non-household contacts, as no definition exists of the amount of time or closeness of contact which places someone at risk. Further studies on the feasibility and cost-effectiveness of administering chemoprophylaxis to non-household contacts are needed.

References

1. World Health Organization. Leprosy Update 2011. *Wkly Epidemiol Rec* 2011; 86: 389–99.
2. Slaon-Gardner T, Stirzaker S, Knuckey D, Pennington K, Knope K, Fitzsimmons G et al. Australia's notifiable disease status 2009: Annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2011; 35: 61–131.
3. Rodrigues LC, Lockwood DNJ. Leprosy now: epidemiology, progress, challenges, and research gaps. *Lancet Infect Dis* 2011; 11: 464–70. doi:10.1016/S1473-3099(11)70006-8
4. Moet FJ, Pahan D, Oskam L, Richardus J. Effectiveness of single dose rifampicin in preventing leprosy in close contacts of patients with newly diagnosed leprosy: cluster randomised controlled trial. *BMJ* 2008; 336: 761–4. doi:10.1136/bmj.39500.885752.BE