Leprosy

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Leprosy, also known as Hansen's disease, is a curable granulomatous condition caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis*, disproportionately affecting impoverished communities across the globe. The bacteria's tropism for dermal histiocytes, endothelial cells, and Schwann cells causes neuronal and dermal damage that often results in disability, permanent disfigurement, stigma, and social exclusion. Despite important achievements in the understanding of the disease, its elimination has been hampered by the scarcity of sensitive diagnostic tools, sub-optimal integration and implementation of coordinated and financially stable preventive interventions, persistent stigmatisation, and failure to efficiently and sustainably address the socioeconomic and demographic factors associated with an increased risk of leprosy. In this Seminar, we provide an update on key public health and clinical aspects of the disease.

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Introduction

Leprosy, or Hansen's disease, is a curable chronic granulomatous condition caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis*.[1] *M leprae*, first described by Gerhard Armauer Hansen in 1873, remains a key global health issue, disproportionately affecting impoverished communities.[2] *M lepromatosis*, first identified in 2008, appears to be geographically restricted to the American continent (particularly in the western Pacific coastal states of Mexico) and to the Asia–Pacific region.[3]

The bacteria's tropism for dermal histiocytes, endothelial cells, and Schwann cells in cooler body regions causes clinically evident pathology in the skin, peripheral nerves, eyes, testes, and the upper respiratory tract, often leading to disability, permanent disfigurement, stigma, and social exclusion.[4] Efforts to control and eliminate leprosy face multifaceted challenges, including the disease's inherent complexities, shortcomings in current diagnostics and treatment regimens, deeply ingrained social attitudes, deficiencies in health-care infrastructure and funding, insufficient political will, and various demographic and socio-economic factors.[5] To exemplify the tangible consequences of these unmet needs, we begin with a patient's narrative (panel 1) before examining the disease's public health and clinical aspects.

Search strategy and selection criteria

We used medical subject heading (MeSH) terms to conduct a literature search in PubMed, Google Scholar, and Scopus, for English-language articles published from the database's inception to July 31, 2025. MeSH terms included "leprosy", "leprosy, lepromatous", "leprosy, multibacillary", and "*Mycobacterium leprae*". Examples of search strings include "Mycobacterium leprae", "Mycobacterium lepromatosis", "leprosy", and "Hansen's". Additional sources included clinical practice guidelines and reference lists from key articles, with prioritisation of recent publications focused on epidemiology, diagnosis, management, and prevention.

Biology

M leprae is an obligate intracellular, non-motile, non-spore-forming, microaerophilic, acid-fast bacillus measuring 1–8 μm by 0·3–0·5 μm.[6] *M leprae* does not produce toxins and has evolved to depend on an intracellular niche within macrophages, Schwann cells, and endothelial cells.[7] Since *M leprae* cannot be grown in standard laboratory culture, early advances relied on two specialised animal models. The mouse footpad technique was the first reliable and reproducible animal model of infection, and showed that the bacillus has a slow doubling time of approximately 13 days.[8,9] Later, the nine-banded armadillo, whose naturally low body temperature of 32–35°C supports more extensive bacterial growth, enabled researchers to generate sufficient material for whole-genome sequencing in 2001.[10,11]

Panel 1: Patient's perspective

"My experience with getting diagnosed with leprosy has been a rough journey. I saw many doctors before a diagnosis was made; I was initially told I had lupus. At the beginning, there were a lot of medications, and I was in and out of the hospital because of chronic pain. I still have flare-ups, and it was very hard. I am a lot better now; I just have to take my medications to avoid more flare-ups. I have a lot of scars and my skin has changed colour. I stayed home for 2 years because of the way I looked; only my family knew what was going on. People would often stare at me and that's why I stayed inside for so long—because yes, I was treated differently by people who didn't know me. I have nerve damage in my right leg that I will probably always have, but I'm thankful that I'm better now, even though I still have some difficulties. It hasn't been easy; I was in deep depression for 2 years, but I am mostly recovered now that I have accepted the change of my body. I am feeling much better for the most part. There are days I can't do much because of the pain, but I just get through it."
This patient is female, age 43 years, and resides in Oklahoma, USA, without history of residence or travel abroad or to any other state within the USA.

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Genome sequencing of *M leprae* and *M lepromatosis* revealed extreme reductive evolution, with nearly half of the genome comprising pseudogenes.[11] The potential functions of these non-coding genomic regions (colloquially called junk DNA) are the focus of ongoing research, particularly regarding their possible role in regulating gene expression, immune evasion, and adaptation.[12] Despite the extensive genome reduction, most anabolic pathways have been preserved. However, compared with *Mycobacterium tuberculosis, M leprae* retained a third of its functional genes, restricting metabolic pathways such as lipid metabolism and the use of diverse carbon sources.[13] Notably, *M leprae* relies on glucose for oxaloacetate synthesis—a key amino acid precursor—via phosphoenolpyruvate carboxylase, an enzyme absent in humans and thus a potential therapeutic target.[14]

Genome sequencing of *M leprae* isolates from different regions has revealed a 99·995% identical sequence and synteny supporting the classification of *M leprae* into four single-nucleotide polymorphism types subdivided into 16 subtypes (1A to 4P).[15–18] This classification has been important in elucidating the bacteria's evolutionary relationships, phylogeography, and antimicrobial resistance profiles.[17,18] Furthermore, whole-genome sequencing has become a pivotal tool for the study of the bacteria's genetic variation, allowing for the distinction between relapse and reinfection by mapping transmission chains.[17,19] To date, approximately 324 genomes from 50 countries, distributed into 19 genotypes and nine branches, are publicly available (figure 1).[20] However, an over-representation of lepromatous-case samples limits knowledge of strain diversity and the correlation of yet unidentified strains with disease phenotype.[16] Ongoing genomic studies are uncovering novel drug-resistance mechanisms and hypermutated genes, deepening insight into the molecular basis of *M leprae* pathogenicity and disease progression.[21]

M leprae and *M lepromatosis* diverged from a common ancestor roughly 13·9 million years ago, sharing 71·4% of their gene and pseudogene complement.[22,23] Key differences include the presence of the *hemN* and *sirA* genes exclusively in *M lepromatosis* and variations in the ESX-1 secreted protein genes, which are important determinants of virulence. *M lepromatosis* has a marginally smaller genome size, fewer coding regions, but slightly more pseudogenes than *M leprae*, and 84 unique genomic regions with no counterparts in *M leprae*.[22] Despite these genomic variations, the two pathogens are clinically indistinguishable, requiring molecular techniques for accurate species identification.[23]

Epidemiology

The initial case definition of leprosy was based on the identification of hypoaesthetic or anaesthetic skin lesions associated with enlarged peripheral nerves and the presence of acid-fast bacilli in a slit-skin smear examination.[24] The case definition evolved to include any person with any of these clinical findings who had not completed a full course of antimicrobial therapy.[25]

The introduction of WHO-recommended multidrug therapy in 1981 marked a turning point in leprosy care, replacing lifelong monotherapy with fixed-duration regimens of 6 months to 2 years, depending on disease classification.[26] In 1998, the recommended duration of therapy for multibacillary disease was further shortened from 2 years to 1 year.[25] These changes led to a substantial decline in the global prevalence of leprosy, enabling many countries to achieve the goal set in the 44th World Health Assembly in 1991 of eliminating leprosy as a public health problem by 2000 (defined as a national prevalence of less than one case per 10 000 individuals).[26] In 2000, WHO announced that global elimination had been achieved—that is, the worldwide average prevalence had fallen below the threshold. However, six countries (Brazil, the Democratic Republic of the Congo, Madagascar, Mozambique, Nepal, and Tanzania) still had prevalence rates above this level, and intensified efforts were subsequently directed towards these countries over the following 5 years.[27]

Building on the success of early case detection and multidrug therapy, programmes were expanded to curtail transmission through early case finding and the provision of preventive chemotherapy to close contacts. Concurrently, initiatives were introduced to address the long-term biological and psychosocial disabilities of affected individuals and to foster patient and community empowerment as means of stigma reduction. Accordingly, the WHO target was revised to achieve a minimum 35% reduction in the rate of new patients presenting with grade 2 disability by the end of 2015.[28] The Global Leprosy Strategy 2016–20 also targeted the elimination of newly detected cases of infection in children (under 15 years) with grade 2 disability, a rate of grade 2 disability in newly detected patients with leprosy of less than one per million population, and annulment of discriminatory legislation.[29]

The new 2021–30 Global Strategy targets the elimination of autochthonous cases in 120 countries, a 70% reduction in the annual number of new cases globally, and a 90% reduction in the rate per million population (including children) of new cases with grade 2 disability by 2030 from a 2020 projected baseline.[30] This strategy, in contrast to its predecessor, outlines concrete and time-bound targets within a global health integration framework, and emphasises programme monitoring and accountability.[30]

The true incidence and prevalence of leprosy are unknown. In 2023, 182 815 new cases of infection were reported, with 39·8% being in women and 5·6% in children. Nearly 10 000 new leprosy diagnoses were associated with grade 2 disability, 2·7% of which were in children.[31] Although leprosy affects all age groups, incidence peaks during the most productive years of life, with the burden of disability concentrated in individuals aged 20–49 years.[32] These estimates,

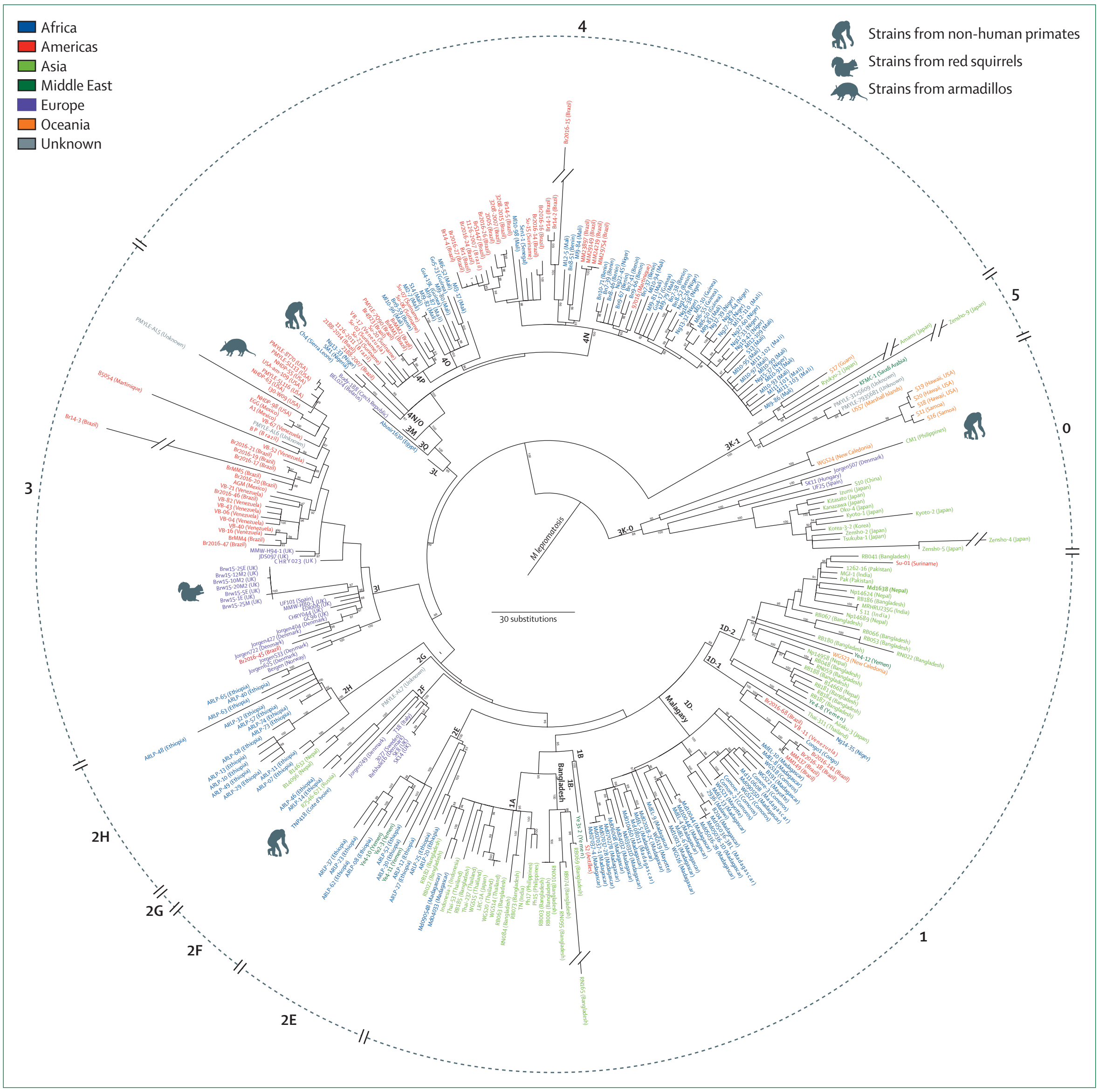

Figure 1: **Phylogenetic tree of 324 *Mycobacterium leprae* strains**

The tree was built using the Maximum Parsimony method in MEGA-X using an alignment of 4812 genome-wide homozygous single-nucleotide polymorphisms, allowing for 20% missing data. *Mycobacterium lepromatosis* was used as an outgroup. Bootstrap support was estimated from 500 replicates; branches with greater than 80% bootstrap support are denoted. Branch lengths of certain hypermutator strains (depicted by ||) are shortened for aesthetic purposes. Strains are colour-coded according to continent of origin using UN macro-geographical regions. The nine major *Mycobacterium leprae* branches are denoted in grey and the genotypes in black.

however, fail to capture the substantial and persistent hidden burden of leprosy.

Although many countries have met elimination targets, subnational hotspots with prevalence rates above the WHO's elimination threshold persist. These clusters are commonly found in periurban fringes, where rapid, unplanned expansion outpaces delivery of basic services, and in remote rural hamlets hindered by under-resourced clinics and long travel distances. Such settings share several characteristics: low average income that limits access to adequate nutrition, housing, and health insurance; widely dispersed populations that render active case-finding and contact tracing logistically burdensome; deficient infrastructure that compounds delays in reporting and case management; and pervasive social marginalisation and stigma, which often deters individuals from seeking care until disability or deformity has already developed.[33–35] Understanding how these interlinked factors drive spatial variation in leprosy is therefore essential to planning targeted surveillance, expanding access to care in underserved areas, and tailoring health education to vulnerable populations.

Transmission

The transmission dynamics of the bacillus are not yet fully understood, given the inability to culture the bacteria in vitro, its prolonged incubation period, the absence of dependable diagnostic tools for detecting asymptomatic infection, and the fact that over 95% of individuals exposed to the bacteria exhibit protective immunity.[5,36] However, respiratory spread via nasal droplets from patients with multibacillary disease is considered to be the main route of infection.[37,38] Host susceptibility, environmental factors, and socioeconomic conditions—including the multidimensional and interconnected effects of intergenerational poverty such as overcrowded housing, manual labour, food insecurity, poor education, inadequate sanitation, and stigma—modulate transmission and perpetuate a cycle of heightened disease risk and disability at diagnosis.[39,40] Epidemiological data indicate a four-fold increase in risk among household contacts compared with those residing more than 100 metres from an index case.[38] Nonetheless, asymptomatic infected carriers might serve as reservoirs and contribute to transmission.[41]

Importantly, viable *M leprae* bacilli have been isolated from soil, water, and free-living amoeba, suggesting an environmental reservoir that might sustain pathogen transmission in the absence of human contact.[38,42] Furthermore, triatomines (kissing bugs) and ticks have been proposed as possible vectors, although there is insufficient robust evidence to support their role.[43,44] Percutaneous transmission is supported by case reports of leprosy occurring after tattooing, injections, or minor trauma.[45,46]

Although humans constitute the primary reservoir, zoonotic transmission is playing an emerging role in southern regions of the USA, where two different strains of *M leprae* have been found in nine-banded armadillos and humans.[47,48] In the city of Belterra, state of Pará, in the Brazilian Amazon region, higher rates of armadillo consumption by residents were associated with a nearly 2% higher risk of developing leprosy.[49] In the UK, red squirrels are reservoirs of both *M leprae* and *M lepromatosis*, with strains closely related to medieval fur trade lineages.[50,51] *M leprae* has also been isolated from captive and wild non-human primate populations in western Africa and Asia, but the origin of the infection remains to be elucidated.[52,53] These diverse human, animal, and environmental reservoirs threaten current eradication initiatives, underscoring the imperative for a One Health strategy that integrates multisectoral interventions to curtail leprosy's global burden.

Pathogenesis

Pathogen-directed and immune-mediated peripheral nerve damage is the key feature of the disease. The bacilli's tropism to Schwann cells is mediated by binding of its surface ligands—phenolic glycolipid I (PGL-I) and *M leprae* laminin-binding protein 2—to the G domain of the α2 subunit of laminin-2 in the basal lamina. This extracellular matrix protein acts as a bridge between the bacteria's surface molecules and the α-dystroglycan receptor present in the cell membrane of Schwann cells.[54] The interaction activates the phosphoinositol-3-kinase signalling pathway, promoting bacterial internalisation.[55]

In parallel, the bacilli can directly bind to receptor tyrosine-protein kinase ErbB-2 on the Schwann cell membrane, triggering the mitogen-activated protein kinase cascade. Subsequent activation of Erk1/2 mitogen-activated protein kinase induces cyclin D1 overexpression, which leads to cell dedifferentiation and demyelination in the absence of inflammation.[54] Cyclin D1 might also facilitate myelin degradation through alternative routes such as the Notch signalling pathway.[55] Moreover, ErbB-2 receptor phosphorylation impedes its heterodimerisation with ErbB3, thereby reducing the expression of the myelin-maintaining transcription factors Krox-20, Oct-6, and Sox-10, perpetuating cyclin D1-mediated demyelination.[55] Notably, these signalling events and the resultant demyelination occur even in the absence of viable bacilli.[56]

In addition, cyclin D1-driven Schwann cell dedifferentiation yields a progenitor, stem-like cell phenotype that not only downregulates myelin-related genes but also facilitates bacterial dissemination to other tissues, such as muscle and blood vessels.[54] These progenitor, stem-like phenotype cells can, for example, recruit macrophages, which in turn act as carriers to amplify systemic spread.[54] Additional mechanisms of nerve damage include neurofilament hypophosphorylation, reduced expression of neurotrophins, and polymorphisms in the gene coding for the adhesion molecule Ninjurin-1, all of which impair nerve regeneration.[55]

The immune response to *M leprae* plays a crucial role in the pathogenesis of nerve damage, particularly in paucibacillary forms and in leprosy reactions.[57,58] The host's genetic background regulates the nature of the immune response, thereby affecting both susceptibility to infection and the degree and type of the ensuing specific cellular immune response. Polymorphisms in how *M leprae* is recognised through pathogen-associated molecular patterns, in intracellular microbicidal functions, and in antigen processing and presentation, are believed to have a central role in the divergence of macrophage differentiation, which dictates the polarity of the cellular immune response.[57,58] For instance, genome-wide association studies have shown that single-nucleotide polymorphisms in the nucleotide-binding oligomerisation domain 2-like receptors (an intracellular pattern recognition receptor gene) are associated with an increased susceptibility to leprosy.[59] Moreover, the low antigen presentation associated with HLA *DRB1*15:01* allele is hypothesised to contribute to an impaired cellular immune response and increased predisposition to lepromatous leprosy.[60]

The tuberculoid pole of leprosy is characterised by a robust T-helper-1 (Th1) cell-driven immune response and

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a relative paucity of regulatory T cells in both peripheral blood and skin. Effector T-cell subsets such as Th1, Th9, Th17, and Th22 secrete signature cytokines that induce macrophages to adopt an epithelioid phenotype and to fuse into multinucleated giant cells. This process yields well organised, paucibacillary granulomas with pronounced perineural and intraneural infiltration. The resulting sausage-type dermal infiltrates compress and infiltrate nerve fascicles. Dermal infiltration manifests clinically as erythematous plaques, nodules, and ulcers, ultimately leading to fibrosis and loss of adnexal structures.[57,58]

By contrast, polar lepromatous leprosy is defined by a Th2-skewed immune response, with elevated amounts of IL4, IL5, and IL10, expansion of programmed cell death protein 1-expressing regulatory T cells, and increased programmed cell death ligand 1 expression on antigen-presenting cells; all of which suppress effector T-cell activity. The failure to form organised granulomas allows for unrestrained mycobacterial proliferation and accumulation of foamy, lipid-laden macrophages. This immunosuppressive milieu fosters high bacillary loads, diffuse skin infiltrates, and systemic dissemination.[57,58]

Clinical presentation

The cardinal signs of leprosy are hypopigmented or erythematous skin lesions accompanied by sensory loss resulting from peripheral nerve involvement. Leprosy is considered one of the great imitators, underscoring the need to increase awareness of its diverse clinical presentations.[61]

The primary systems for classification of leprosy are the Ridley and Jopling scheme, developed in 1966, and the WHO classification.[62,63] The Ridley–Jopling system correlates clinical, immunological, and pathological features with the bacteriologic index, defined as the number of acid-fast bacilli in the dermis expressed in a logarithmic scale from zero to six.[62] Patients are stratified along a spectrum that spans polar lepromatous, borderline lepromatous, borderline-borderline, borderline tuberculoid, and polar tuberculoid leprosy. This continuum is fluid, with frequent upgrading as cell-mediated immunity improves (moving towards polar tuberculoid leprosy) and downgrading as cell-mediated immunity worsens (moving towards lepromatous leprosy) within the immunologically unstable borderline forms. The differential diagnosis of leprosy encompasses a broad spectrum of conditions that vary by the clinical classification. Impaired pinprick or light touch sensation can aid in its distinction from other disorders. Clinicopathological correlations and important differential diagnoses are summarised in the table. Figures 2A to F and the appendix (pp 3–6) provide representative illustrations of the various clinical forms.

WHO's classification was developed to simplify therapeutic decisions by quantifying cutaneous involvement: up to five skin lesions define paucibacillary disease, whereas six or more skin lesions or any detectable nerve involvement, irrespective of skin lesion count, is considered multibacillary leprosy. When slit-skin smear microscopy is available, the presence of acid-fast bacilli in any biopsy material mandates multibacillary classification.[63] This distinction is crucial in regions with a high prevalence of multibacillary disease, given that reliance on lesion count alone can misclassify multibacillary cases as paucibacillary.[66]

Diffuse lepromatous leprosy, also known as diffuse leprosy of Lucio and Latapí, is a distinct variant of the lepromatous spectrum. In the early stages, diffuse dermal infiltration gives rise to a smooth, shiny, unwrinkled skin, hence the term *lepra bonita* (meaning "beautiful leprosy" in Spanish).[1,67] As the disease progresses, the skin becomes thin and atrophied, leading to a prematurely aged appearance.[67] Notably, approximately half of affected patients experience Lucio's phenomenon, a necrotising leprosy reaction.[3]

Atypical clinical presentations of leprosy include verrucous lesions predominantly affecting the knees and the anterior aspect of the ankles and feet.[68] Histoid leprosy is characterised by painless cutaneous and subcutaneous plaques and nodules on a background of normal appearing skin. Skin lesions usually occur on the face, the posterior and lateral aspects of the extremities, lower back, and bony prominences (appendix pp 4–5). Lesions can also appear on mucosal surfaces, the genitalia, and along the course of peripheral nerves.[69] Pure neuritic leprosy is exceedingly difficult to diagnose due to the restricted involvement of peripheral nerves in the absence of skin lesions. Mononeuritis is the most common manifestation, usually involving the ulnar, radial, common peroneal, and posterior tibial nerves.[70]

Although leprosy principally affects the skin and peripheral nerves, systemic involvement substantially increases morbidity.[65,71] The nasal mucosa and paranasal sinuses are the most affected extracutaneous sites, manifesting as chronic rhinitis, epistaxis, septal perforation, and atrophic changes that correlate with bacillary index. Ocular complications arising from neural impairment and direct bacillary infiltration include lagophthalmos, corneal hypoesthesia with exposure keratitis, anterior uveitis, and, if left untreated, irreversible vision loss. In multibacillary disease, visceral spread to the liver, spleen, bowel, adrenal glands, and testicles, potentially leading to hypogonadism and infertility, has been documented.[65,71] Moreover, involvement of the spinal cord, brainstem, and cerebral hemispheres gives rise to distinct clinical syndromes.[72,73]

When classifying disability in leprosy, grade 1 disability is characterised by sensory impairment without overt deformity, placing patients at heightened risk for burns, cuts, and other unnoticed injuries that might progress to severe tissue damage. The presence of visual deformity and damage (grade 2 disability) often follows predictable

See Online for appendix

	Clinical characteristics	Pathology	Bacillary index[64]	Differential diagnosis[65]
Tuberculoid leprosy	One to three hypoesthetic or anaesthetic lesions (lesions in face can have normal sensation); hairless, and sometimes scaly erythematous plaque with raised well defined outer borders; thickened and irregular peripheral nerves can be palpable close to the lesion; cold abscesses can develop along the path of a peripheral nerve	Mature granuloma with well developed epithelioid cells with variable presence of Langhans cells surrounded by a dense lymphocyte infiltrate extending to the epidermis without an over-riding clear zone; when discernible, nerves are densely infiltrated within the granuloma	Usually 0, rarely 1+	Sarcoidosis; lupus vulgaris; granuloma annulare; pityriasis rosea; vitiligo; chromoblastomycosis; mycosis fungoides
Tuberculoid borderline leprosy	Usually, five to 25 asymmetrically distributed macules or plaques that are larger in size than tuberculoid leprosy lesions; satellite or finger-like lesions; loss of sensation is less conspicuous than tuberculoid leprosy; nerves are asymmetrically and irregularly thickened	Similar to tuberculoid leprosy except for the presence of a subepidermal clear zone; when noticeable, the nerves are densely infiltrated leading to reduced innervation	1–2+	Discoid lupus; tinea corporis; pityriasis versicolor; psoriasis; eczematous reaction; secondary syphilis; drug eruptions; anergic cutaneous leishmaniasis; post-kala-azar dermal leishmaniasis; lobomycosis; systemic lupus erythematosus; neurofibromatosis; Kaposi's sarcoma; differential diagnosis of nerve involvement: Déjérine–Sottas's disease, diabetic neuropathy, compression neuropathy, medication-induced neuropathy, and syringomyelia
Borderline-borderline leprosy	Typically, more than 25 asymmetrically distributed annular appearing macules or plaques with normal appearing skin in the centre surrounded by a well defined inner border and poorly defined outer border giving the appearance of punched out lesions; asymmetrical enlarged peripheral nerves	Epithelioid cells are scattered in the granuloma and are not contained by a lymphocyte zone; Langhans giant cells are absent; nerve bundles are easily identified with a variable degree of lymphocyte infiltration	2–3+	As for tuberculoid borderline leprosy
Borderline lepromatous leprosy	Innumerable symmetrically distributed macules and plaques with associated infiltrative nodular lesions; lack of infiltration of ear lobes, madarosis, keratitis, iritis, and normal-appearing skin between nodules which tend to dimple; enlarged peripheral nerves have a more symmetrical distribution than borderline-borderline leprosy	Histiocytes have the propensity to evolve into epithelioid cells; no foamy changes, or histiocytes with foamy changes but without clumps of acid-fact bacilli (*globi*); lymphocytes are present in the perineural cuffs or in an entire section of the granuloma; the structure of the nerve is severely affected but without increased cellularity	3–4+	As for tuberculoid borderline leprosy
Lepromatous leprosy	Widespread symmetrically distributed macules, plaques, and nodular lesions; thickened facial skin with loss of eyebrows; nasal mucosa, eyes, and larynx can be affected; symmetrical involvement or nerves can lead to so-called glove-and-stocking anaesthesia	Histiocytes are inactive and filled with host lipids (Virchow cells), associated with absence of lymphocytes; nerves show structural damage but without infiltration	4–6+	As for tuberculoid borderline leprosy

The bacterial index is the number of bacilli per oil immersion field(s). 1+: 1–10 bacilli per 100 fields; 2+: 1–10 bacilli per 10 fields; 3+: 1–10 bacilli per field; 4+: 10–100 bacilli per field; 5+: 100–1000 bacilli per field; and 6+: More than 1000 bacilli per field.[64]

***Table:* Clinicopathological correlation of disease staging according to the Ridley–Jopling classification**[62,63]

patterns of nerve damage and repetitive trauma.[30,65] Ulnar and median neuropathies produce a lesion known as claw hand (a flexion contracture of the metacarpophalangeal joints with hyperextension at the interphalangeal joints; figure 2D), and peroneal nerve palsy causes foot drop, resulting in a steppage gait and increased risk of plantar ulceration (figure 2E). Trophic ulcers typically develop on weight-bearing areas of the foot, often complicated by secondary infection and osteomyelitis.[65] Beyond physical impairments, leprosy exerts profound psychosocial effects. The visible deformities and enduring misconceptions foster isolation, diminished self-esteem, and mental health disorders. This multifaceted psychosocial burden underscores the necessity of a holistic management that integrates psychosocial assessment and support with dermatological, neurological, and rehabilitative care.[30]

The influence of immunosuppression on infection susceptibility, disease progression, and treatment response is uncertain and likely depends on the underlying aetiology, mechanism, duration, and severity of immunosuppression. For example, although the incidence of leprosy is not higher in people living with HIV than in the general population and shows a similar clinicopathologic spectrum in those with HIV, initiation of antiretroviral therapy can precipitate reactivation of latent leprosy and increase the frequency of immune-mediated reactions.[74]

Coinfections are common in certain regions, such as Honduras and Nicaragua, where approximately a third of patients with atypical cutaneous leishmaniasis, caused by *Leishmania poncei,* an entity that was previously attributed to *L infantum*, are coinfected with *M leprae*.[75,76] A 2023 systematic review found that coinfections with tuberculosis, leishmaniasis, chromoblastomycosis, and helminths were frequent. Notably, coinfection with tuberculosis and leishmaniasis appeared to confer a protective effect against the development of leprosy immune-mediated reactions, although further studies are needed to better define the correlation.[77]

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Diagnosis

The diagnosis of leprosy depends on the recognition of one of the three cardinal signs: loss of sensation in pale (hypopigmented) or erythematous skin lesions, thickened or enlarged peripheral nerve with associated loss of sensation, muscle weakness, or both and microscopic detection of the bacilli in slit-skin smears.[63] However, considerable heterogeneity in the morphology and characteristics of skin lesions, inconsistent sensory loss across the disease spectrum (eg, infiltrated skin lesions in lepromatous leprosy), and the absence of, or inability to detect, peripheral nerve enlargement complicate case recognition. Furthermore, the dwindling global pool of expertise in leprosy diagnosis and management, together with delays in recognition by patients and front-line health-care workers, are important barriers to interrupting transmission, reducing morbidity, and alleviating stigma.[34,35,78]

Disease classification into paucibacillary or multibacillary forms should ideally be based on histopathological and microbiological criteria rather than the less dependable measure of lesion count, given the important therapeutic and prognostic implications.[66] Conventional methods, such as slit-skin smears and skin biopsies, are limited by an overall sensitivity of 10–50%, with lower diagnostic accuracy in paucibacillary disease.[79,80] The incorporation of quantitative polymerase chain reaction for bacterial DNA detection improves the sensitivity of paucibacillary disease diagnoses (57%) compared with histopathological analysis (35%).[79,81] Moreover, combining multi-target nested PCR with enzyme-linked immunospot improves the sensitivity of detection of minimal bacterial loads and specific T-cell responses, overcoming traditional diagnostic gaps and reducing the misdiagnosis of paucibacillary leprosy.[82]

Immunodiagnostic approaches have been based on the detection of antibodies to key cell wall components, most notably anti-PGL-I antibodies and the anti-natural octyl disaccharide-Leprosy Infectious Disease Research Institute diagnostic assay (NDO-LID).[83] The sensitivity of these assays varies between 82–87% for multibacillary disease and 65–78% for paucibacillary disease.[81] Compared with slit-skin smears, detection of anti-PGL-I antibodies could help reduce misclassification of multibacillary disease as paucibacillary.[84,85] Furthermore, PGL-I and NDO-LID-based serological testing could be useful in identifying latent infection among at-risk individuals, estimating community transmission intensity, and monitoring the effect of both prophylactic and therapeutic interventions.[86–88] When combined with skin-slit smears and molecular diagnostics, these complementary assays can substantially increase the diagnostic yield of multibacillary leprosy to 90·5% and paucibacillary leprosy to 70·6%.[89]

Innovative techniques, such as the identification of leprosy-specific mRNA biomarkers in skin lesions, enable discrimination from other dermatological conditions and might improve diagnostic accuracy when integrated with emerging technologies such as CRISPR-based diagnostics.[90] The sustainable implementation of advanced diagnostic technologies is, however, hampered by their cost, the scarcity of laboratory infrastructure, and a shortage of expertise in the most affected communities. The development of low-cost, point-of-care serological and molecular diagnostic tests could help to overcome these obstacles. The immunochromatography test ML Flow, for example, has been incorporated by the Brazilian Unified Health System as a complementary tool for contact tracing.[83,91] Likewise, a multi-biomarker point-of-care test that detects host cellular and serological biomarkers to confirm infection and distinguish disease phenotype is promising.[92,93]

In this context, assays capable of detecting *M leprae* DNA from easily obtained samples such as saliva or urine could help to curtail operational barriers.[94,95] Additionally, high-definition ultrasound of all principal peripheral nerves that can be affected by leprosy has emerged as a non-invasive technique for the early detection of neuropathy in otherwise asymptomatic individuals.[96] A diagnostic and therapeutic algorithm integrating these approaches is presented in figure 3.

Figure 2: **Clinical spectrum of leprosy**
Diverse cutaneous and neurological manifestations of leprosy. (A) A patient with polar tuberculoid leprosy with a large solitary hypopigmented macule with ill-defined borders. (B) A patient with borderline tuberculoid leprosy with finger-like satellite lesions extending beyond the edges. (C) A patient with borderline lepromatous leprosy with large asymmetrically distributed plaques with raised erythematous borders suggestive of type 1 reversal reaction. (D) Involvement of the ulnar and median nerve with the clinical aspect of claw hand with bone involvement and loss of fingers. (E) Damage of the common peroneal nerve with ulceration on the lateral aspect of the feet with loss of digits. (F) Corresponds to a patient with polar lepromatous leprosy experiencing a type 2 reaction with painful hand oedema, dactylitis, and skin ulceration.

Clinical suspicion

Any of the following:
- Skin lesions: hypopigmented or erythematous macules or papules
- Sensory loss: diminished or absent sensation within the skin lesions; loss of thermal sensitivity usually precedes loss of tactile sensitivity
- Nerve involvement: thickened peripheral nerves, especially the ulnar, radial, median, posterior tibial, and common peroneal nerves

Multibacillary disease: more than five skin lesions, nerve involvement, or positive skin smear

Ancillary diagnostics

Slit-skin smear
- Sample typical lesions and at least six sites (both earlobes, elbows, and knees)

Skin biopsy
- Histopathological analysis aids with disease classification especially for the borderline forms

Nerve biopsy
- Better sensitivity than skin biopsy
- Invasive and can lead to irreversible nerve damage

PCR
- Most sensitive and specific assay
- Lack of availability and standardisation precludes widespread use

Serological tests
- A negative test does not exclude leprosy (especially paucibacillary disease)

High resolution ultrasound of nerves
- Can detect nerve enlargement before clinically evident
- Operator dependent

Emerging mobile technologies
- Useful in resource-constrained endemic regions

Treatment

Screening
- When feasible, consider screening for G6PD deficiency and *HLA-B*13:01* before prescribing dapsone
- Screen for HIV, latent tuberculosis, strongyloidiasis, and hepatitis B and C
- Screen for *Trypanosoma cruzi* in endemic areas of Latin America (continental America, excluding the Caribbean)

Antimicrobial treatment
- Monthly rifampicin 600 mg and clofazimine 300 mg plus daily dapsone 100 mg and clofazimine 50 mg, respectively
  - 12 months for multibacillary disease
  - 6 months for paucibacillary disease
    - Two drugs can be used: rifampicin 600 mg monthly plus dapsone 100 mg daily
  - Alternative treatment regimen should be ideally provided under research protocols

Monitor for development of reactions
- Educate patients that their development does not imply treatment failure and to seek immediate care to prevent disability

Assess nerve function and physical and psychological disability
- Provide self-care information
- Referral to rehabilitation services and surgical specialties if clinically indicated
- Promote interventions aimed at reducing stigmatisation

Contact tracing and prophylaxis
- Provide single-dose rifampicin to close contacts after exclusion of tuberculosis and active leprosy

Figure 3: **Diagnostic and therapeutic algorithm of leprosy**

Treatment

The discovery of sulfonamides in the 1940s laid the foundations of modern therapy: curability, need of multidrug therapy, and finite treatment duration (figure 3).[26] The treatment of leprosy is nevertheless unsupported by stringent and high-quality data, as a result of insufficient well designed clinical research.[97]

Traditional multidrug therapy remains hampered by long treatment courses, complexity of the treatment regimen, operational challenges, and notable drug-related toxicities. The availability of alternative bactericidal agents with better safety profiles compared with traditional bacteriostatic drugs, such as dapsone and clofazimine, is a compelling argument for the re-evaluation of current therapeutic strategies. For example, dapsone is known to induce haemolysis in individuals with glucose-6-phosphate deficiency. Furthermore, carriers of the *HLA-B*13:01* allele are at increased risk of developing a potentially life-threatening dapsone hypersensitivity syndrome.[98]

Considering these concerns, preliminary evidence supports the efficacy and safety of a monthly regimen comprising rifampicin 600 mg, moxifloxacin 400 mg, and minocycline 100 mg (appendix pp 4–5).[99–101] This regimen has been adopted by the Hospital for Tropical Diseases (London, UK) and, more recently, by the US National Hansen's Disease Program, with treatment duration of 12 months for paucibacillary disease and 24 months for multibacillary disease. However, the routine implementation of this regimen in endemic regions—particularly where fluoroquinolone rates are low—requires validation in prospective studies assessing long-term safety and efficacy.

Further innovation is exemplified by a proof-of-concept study showing that an 8-week course of bedaquiline monotherapy was both safe and effective in achieving microbiological clearance of *M leprae* in two cohorts of previously untreated patients with multibacillary disease.[102] Furthermore, a randomised controlled trial supports the simplification of multidrug therapy to a uniform 6-month duration irrespective of the bacillary load.[103,104] Streamlining treatment into shorter, safer regimens effective across all bacillary loads could be fast-tracked by incorporating emerging technologies such as molecular viability assays, removing the need for decades-long efficacy studies.[105–107]

Antimicrobial resistance poses a key threat to leprosy control. A prospective surveillance initiative in 19 countries identified resistance to either rifampicin, dapsone, or ofloxacin in 8% of 1932 analysed samples. Resistance to rifampicin was 2% in newly diagnosed cases and approximately 5% in relapsed cases.[108] In India, resistance to ofloxacin, an important second-line treatment agent, has been detected in 11–12% of treatment-naive or recurrent cases.[109,110] Surveillance of drug resistance patterns and mechanisms in areas with high transmission rates is therefore a vital component of the global elimination strategy.[111] Rifampicin-resistant leprosy should be treated with at least two second-line agents (clarithromycin 500 mg daily, minocycline 100 mg daily, or a daily quinolone [ofloxacin 400 mg/levofloxacin 500 mg/moxifloxacin 400 mg]), in combination with clofazimine 50 mg daily for 6 months, followed by 18 months of daily clofazimine with a second-line agent, typically minocycline or, if susceptibility permits, a fluoroquinolone.[63]

Despite bacteriological cure, nerve damage and immune-mediated reactions might persist due to the slow clearance of nonviable bacilli. The bacteriologic index declines by roughly 1 log-unit every 14 to 24 months, and patients who have an initially high bacillary load take longer to become smear-negative. Erythema nodosum leprosum, for example, can still occur in patients even after their smears become negative.[112]

Surgical interventions are pivotal in the management of acute and chronic leprosy-related complications. Acute neuritis might require urgent nerve decompression, and grade 2 disability management relies on a comprehensive, multifaceted approach, including tendon transfers for claw hand and other reconstructive procedures to restore form and function. Trophic ulcers require meticulous wound care, custom-made protective footwear, and orthotic offloading. Regular ophthalmologic assessments, combined with surgical correction of lagophthalmos or cataracts, address ocular sequelae.[113] Additionally, providing self-care patient education is essential, as it underpins effective disability prevention and rehabilitation.

Comprehensive psychosocial interventions, such as therapeutic workshops, cognitive-behavioural therapy, and individual and family counselling, have shown efficacy in alleviating depression and anxiety and in enhancing quality of life. Integrating these services into routine clinical practice optimises patients' outcomes and ensures sustained psychosocial support.[114]

Equally important is the mitigation of stigma through a rights-based framework, employing validated tools to assess individual and community attitudes. Interventions should be tailored to local contexts, prioritising high-impact settings such as schools and workplaces. Core strategies include targeted information and education, community-led stigma-reduction clubs, socioeconomic rehabilitation programmes, training health workers in the delivery of respectful care, and measures to address visible deformities.[115]

Leprosy reactions

Immune reactions and their treatment toxicities are major drivers of leprosy-related morbidity and disability. Leprosy reactions might herald disease onset or arise during treatment or years after successful antimicrobial therapy.

Type 1 (reversal) reactions predominate in tuberculoid borderline and borderline-borderline leprosy, but can also occur in borderline lepromatous and lepromatous leprosy.[116] These reactions reflect a heightened type 4 hypersensitivity cellular immune response, in which macrophages with the classic M1 pro-inflammatory phenotype recruit Th1 and Th17 lymphocytes.[117] Clinically, reversal reactions manifest as an acute or subacute exacerbation of inflammation in pre-existing cutaneous lesions and nerves, rendering affected areas erythematous, oedematous, and painful (figure 2C). New skin lesions might appear, often with associated facial and hand oedema. Rapid nerve function loss can lead to paralysis and anaesthesia if treatment is delayed. Reversal reactions affect 20–40% of patients, with a higher incidence in males, individuals with borderline-borderline leprosy with positive skin-slit smears, individuals who are seropositive for anti-PGL-I antibodies, those older than 15 years, and during the puerperium.[118–121] Diagnosis is clinical, as no specific serological markers or pathognomonic histopathologic features have been identified.[120]

Type 2 reaction, or erythema nodosum leprosum, occurs in approximately 9% of patients with borderline lepromatous leprosy and 49% of patients with lepromatous leprosy.[122] The reaction might present as a single episode, recurrent episodes, or more frequently, as a chronic form.[122] Immune complex tissue deposition, an increase in Th17 cells, and a decrease in regulatory T cells are the major immunopathogenic mechanisms.[117] A high initial bacteriologic index of four or greater and classification as lepromatous leprosy and borderline lepromatous leprosy forms are the primary risk factors, and hormonal shifts during pregnancy, lactation, and puberty can further predispose individuals to erythema nodosum leprosum episodes.[123] Erythema nodosum leprosum is a multisystemic disorder often mistaken for systemic lupus erythematosus, rheumatoid arthritis, vasculitis, sarcoidosis, or lymphoma.[124] Clinically, patients present with an abrupt onset of fever, arthritis, dactylitis, accompanied by crops of painful erythematous nodules—frequently ulcerate and become pustular—especially on the face and extensor surfaces of the extremities. Other manifestations include orchitis, lymphadenopathy, glomerulonephritis, osteitis, and iridocyclitis (figure 2F and appendix p 6).[125]

Skin biopsies of type 1 reactions show an exuberant lymphocytic infiltrate with multinucleated giant cells and well formed granulomas associated with papillary dermis oedema.[126] Epidermotropism, a feature classically associated with mycosis fungoides, has likewise been observed in reversal reactions.[127] Neutrophilic infiltration of granulomas in the epidermis and dermis in lesions of less than 24–72 h is the hallmark of erythema nodosum leprosum.[127] Additionally, neutrophilic infiltration and fibrin deposition in the vessel walls of the dermis and subcutaneous, often with intraluminal thrombus, are also common but not a universal finding.[127]

Lucio's phenomenon is a life-threatening type 3 hypersensitivity reaction predominantly seen in patients from South America and central America with persistently high bacillary loads of *M leprae* or *M lepromatosis*.[128] Immune complexes deposit in small and medium-sized blood vessels, triggering complement activation, phagocyte influx, and platelet aggregation.[117] Histologically, Lucio's phenomenon is characterised by endothelial proliferation, thrombosis, leukocytoclastic vasculitis, ischaemic necrosis, and abundant bacilli within endothelial cells. Clinically, skin necrosis follows a net-like pattern, primarily affecting previously cyanotic areas on the limbs, but also occurs on the trunk and face. Systemic signs of sepsis are generally absent, although superimposed infections are common.[128]

The disability burden attributable to leprosy reactions and their treatment is difficult to capture, as manifestations might arise decades after therapy. Management of leprosy reactions relies on immunosuppression, with corticosteroids being first-line therapy for both type 1 and type 2 reactions.[129] Azathioprine, methotrexate, and cyclosporine can be used as steroid-sparing agents for type 1 reactions.[130,131] Treatment of erythema nodosum leprosum is difficult and currently suboptimal, often requiring long-term, potentially toxic regimens. Because type 1 interferons are associated with erythema nodosum leprosum pathogenesis, thalidomide, an interferon modulating agent, is considered the treatment of choice. By contrast, agents such as methotrexate and clofazimine offer additional steroid-sparing options.[132,133] No standardised treatment exists for Lucio's phenomenon; current practice targets bacterial clearance with antimicrobials alongside anti-inflammatory and anticoagulant therapies, and supportive wound care.[128]

Immune-mediated reactions pose greater clinical complexity and a higher risk of toxicity than aetiological treatment, and represent a greatly neglected aspect of care. Although the concurrent administration of corticosteroids

Panel 2: Important advances and future directions in leprosy

Pathophysiology

- The identification of phenolic glycolipid 3 is being explored as a novel therapeutic target. The molecule is a potent immunostimulatory glycolipid that provides immunity against infection and might also play a role in leprosy reactions.[145]
- The role of regulatory T cells and T-helper-17 cells in the pathogenesis of leprosy and its reactions could help to elucidate potential therapeutic applications.[58]
- Advances in genetic sequencing technologies, together with the development of functional models through genome editing and cell reprogramming, have expanded the understanding of the role that genetic polymorphisms play in the susceptibility to disease and clinical phenotype.[146]

Diagnostics

- A wide array of complementary serological, molecular, and host-derived diagnostic methods including point-of-care tests could help reduce misclassification of multibacillary as paucibacillary disease, improve the recognition of latent infections in communities with high disease burden, and provide a rapid evaluation of the effect of preventive and therapeutic measures.[91]
- Emerging mobile technologies such as the Leprosy Alert and Response Network System and SkinApp are promising tools to aid frontline health-care workers in expediting diagnosis and treatment.[91,147]
- Development of a molecular viability assay based on the measurement of hsp18 and esxA transcripts could help fast-track development of shorter treatment courses and early identification of treatment failure or relapse.[105–107]

Treatment

- Preliminary data support the effectiveness of better tolerated regimens such as the monthly administration of moxifloxacin, rifampicin, and minocycline, the shortening of multidrug therapy to a uniform 6-month regimen irrespective of the bacillary index, and the repurposing of new drugs such as bedaquiline.[99–104] The implementation of a molecular viability assay in larger confirmatory studies could help advance the potential application of new treatment regimens.
- Telacebec (Q203), an inhibitor of the cytochrome bcc:aa3 terminal oxidase, the only terminal electron acceptor in *Mycobacterium leprae*, is a promising treatment candidate and has shown potent antibacterial activity in the mouse footpad model.[148]
- Immunosuppressive therapy remains the cornerstone of leprosy reactions treatment. The effect of potential new leprosy treatment regimens on the incidence and severity of these reactions should be considered during the design of clinical studies. The concomitant administration of vaccines developed against leprosy could reduce the incidence of reactions.[149]
- Immunomodulatory therapies for erythema nodosum leprosum such as the combination of methotrexate, prednisolone, and metformin are being evaluated in clinical trials.[150,151] The Medicines Development for Global Health has received US Food and Drug Administration clearance to conduct a global phase 2 clinical trial with dovralimast, a phosphodiesterase-4 inhibitor, for treatment of erythema nodosum leprosum.[152]

Prevention

- Rifapentine has been shown to be more effective than rifampicin for postexposure prophylaxis in low levels of endemicity.[140] Enhanced postexposure regimens are being explored, such as rifampicin (150–600 mg) and clarithromycin (150–500 mg), administered every 4 weeks for a total of three doses, and bedaquiline with rifampicin, with dose depending on age and proximity to an index case.[141,142]
- Beyond their potential role in preventing infection, several leprosy vaccine candidates are being evaluated as adjunct immunotherapy given alongside multidrug therapy with the aim of enhancing bacterial clearance and possibly shortening treatment duration. The efficacy of post-exposure prophylaxis could improve with concomitant administration of leprosy vaccines.[143,149]

Stigma and discrimination

- People living with leprosy continue to be segregated from society and their families. A list of current laws and regulations that discriminate against people living with leprosy and their families can be found in the International Federation of Anti-Leprosy Associations website.
- In response, the UN Human Rights Council (UNHRC) adopted a resolution on elimination of discrimination against persons affected with leprosy and their family members in 2008. A Special Rapporteur was appointed in 2017 by UNHRC on the elimination of discrimination.[80]

For more on the **International Federation of Anti-Leprosy Associations** see ilepfederations.org

with multidrug therapy reduces the incidence of reactions and associated nerve damage, this effect is not maintained long term.[134] The efficacy of other immunomodulatory agents administered pre-emptively, and the effect of novel antimicrobial regimens on the incidence of leprosy reactions, merits further assessment. Equally important is the development of scavenger-targeted therapies, which use scavenger molecules to bind, neutralise, or remove toxic substances from the body, to eradicate the persistent antigenic stimuli that drive leprosy reactions. Finally, routine screening for latent infections that might reactivate under long-term immunosuppression is essential.[135]

Prevention

Elimination of leprosy is centred on early diagnosis, timely and equitable access to multidrug therapy, and contact surveillance. The failure to decrease new case detection rates underscores the need to repurpose existing control tools. The BCG vaccine, for example, provides an overall protective effect of 26% against clinical leprosy, particularly multibacillary forms.[136] Booster BCG doses among household contacts further enhance the protective effect.[137]

Chemoprophylaxis has been explored as a strategy for the prevention of leprosy transmission since the 1960s, with dapsone being the first drug used for post-exposure prophylaxis.[138] Subsequent research showed that dapsone alone was less effective in preventing leprosy than other regimens. A single dose of rifampicin in close contacts, for example, yielded a 57% reduction in leprosy incidence at 2 years and of 30% at 5–6 years.[139] This study underpins the current WHO recommendation of single-dose rifampicin as the standard post-exposure prophylaxis regimen alongside active surveillance and early case detection.[63,139] Later studies have shown that rifampicin and rifapentine have variable degrees of protection among household and social contacts contingent on the level of endemicity, with lower efficacy in high transmission settings.[38,139,140] For example, in a low-endemicity setting, a single dose of rifapentine administered to close contacts was more effective than a single dose of rifampicin in preventing leprosy over a 4-year follow-up.[140]

Ongoing challenges in high-endemic regions have spurred research into enhanced post-exposure prophylaxis regimens.[141] For example, there is an ongoing trial examining the combined effect of bedaquiline with single-dose rifampicin in a high-endemicity area.[142] Implementation strategies should be tailored to regional contexts and supported by clear operational guidelines at local, regional, and national levels to maximise their effect.

Vaccines developed from closely related non-pathogenic mycobacterial species, such as the *Mycobacterium indicus pranii* vaccine, have been approved for use in India.[143] Recombinant protein subunit formulations such as LEP-F1 and GLA-SE (LepVax), designed to elicit *M leprae*-specific immune responses, are undergoing further evaluation.[143] Notably, previous BCG vaccination improves the efficacy of rifampicin post-exposure prophylaxis from 57% to 80%.[144]

Effective prevention extends beyond curtailing transmission. Addressing the long-term physical and psychological disabilities of the disease demands a genuine, sustainable, and accountable investment from key stakeholders and society. Such a commitment is essential to tackling not only the biomedical challenges but also the broader societal issues—poverty, inequity, insufficient health-care infrastructure, stigma, and discrimination—that contribute to and exacerbate the impact of the disease.

Finally, patient and community participation are indispensable partners in the global endeavour to eliminate leprosy. Individuals affected by leprosy should actively participate in every facet of leprosy initiatives to ensure scientific rigour and equity. Their active involvement in early detection, treatment adherence, stigma reduction, and programme governance amplifies the effectiveness and sustainability of public health interventions. Strengthening patient empowerment frameworks and integrating patient voices into all levels of leprosy control will accelerate progress towards the WHO's goal of zero leprosy by 2030. Important advances and future directions in leprosy are depicted in panel 2.

Conclusion

In an era of shifting global dynamics, effectively reducing the burden of leprosy requires adapting to an evolving landscape. This adaptation hinges on the integration of robust public health policies with community-based initiatives. Concurrent investment in biomedical research and operational innovation must be underpinned by sustained political will and reliable funding. Such coordinated action, supported by an unwavering commitment to equity, is essential to eliminate leprosy as a public health issue.

Contributors

NIAH conceptualised the article and wrote the initial draft. NIAH, CA, JSS did the literature review. NIAH, CA, AFH-M, TPH, NB, CF-P, RRY, MR, NM, and JSS supervised and revised the manuscript. All authors had final responsibility for the decision to submit for publication.

Declaration of interests

We declare no competing interests.

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