

The Quest for the Global Elimination of Leprosy

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In 1991, the World Health Organization (WHO) and its member states committed themselves to the challenge of eliminating leprosy as a public health problem by the year 2000 (1). Elimination was defined as a prevalence of less than one case per 10 000 persons. The introduction of multidrug therapy for leprosy recommended by WHO had a major impact on the global picture of leprosy in the early 1990s (2) and, at the end of the year 2000, the overall prevalence rate at the global level was below one case per 10 000 (3). Of the 122 countries where the disease was considered endemic in 1985, 107 countries have reached the elimination goal. In early 2002, 90% of cases detected worldwide were in the top six countries where the disease is most prevalent and endemic. These comprise India, Brazil, Nepal, Mozambique, Angola and Myanmar. Of these, 70% of the world's leprosy patients were in India (4). Although no Caribbean country has been named among those countries of concern, the challenge must be elimination in those countries with highest prevalence in order to attain global leprosy control.

Leprosy has been endemic in India and the Far East since ancient times. Although not all cases documented in the Bible may have been leprosy, it was certainly a medical and spiritual issue from the time of Moses. It spread to Europe in the 4th century BC. From there, it spread to Canada with the French settlers and with African slaves to America (2). The causative organism, *Mycobacterium leprae* (*M leprae*), was discovered in 1873 by the Norwegian physician, Armauer Hansen. The stigma and the fear of acquiring the gross deformities, which accompany this disease, remain a major obstacle to leprosy control despite advances in diagnosis and therapy.

M leprae is slow growing *in vivo* and difficult to grow *in vitro*. However, it is a resilient organism which remains viable in the environment for up to ten days (2). The immune response to *M leprae* determines not only whether disease will develop but also which type of leprosy. In paucibacillary leprosy, there is a strong cell-mediated response whereas, in multibacillary leprosy, patients are unable to mount a cell-mediated response or the response is weak. However, the clinical sub-types form a spectrum,

which ranges from tuberculoid leprosy (paucibacillary) to lepromatous leprosy (multibacillary) with borderline forms at the centre of the spectrum.

Serological tests show that infection usually occurs in childhood. The low incidence of leprosy acquired from a marriage partner suggests that adults are relatively non-susceptible, as only about 5% of those at risk acquire the disease. However, when one parent has multibacillary disease and remains untreated, up to 60% of the offspring may develop leprosy as children or young adults. Studies of twins also suggest the possibility of genetic susceptibility. The average incubation period is two to five years for tuberculoid disease and 8 to 12 years for lepromatous cases. American servicemen who developed leprosy after serving in the tropics presented up to 20 years after their presumed exposure. Nasal discharges from highly infectious individuals with lepromatous leprosy are the main source of infection in the community. There have been occasional reports of direct inoculation and leprosy has been transmitted to nude mice through pricks from cactus thorns, so transmission may be reduced by increased wearing of shoes. There is no evidence that biting arthropods transmit leprosy and there are few documented cases of leprosy occurring in medical or non-medical attendants of leprosy patients (2).

The major goals of the leprosy control programme are early detection, prompt and appropriate treatment, prevention of disabilities and rehabilitation when necessary. The antimicrobial agents, dapson, rifampicin, clofazimine, ofloxacin and minocycline, are the mainstay of multidrug therapy for leprosy as recommended by WHO (5).

The long period required for therapy, 6 months for paucibacillary and 12 months for multibacillary, poses a challenge as it may lead to poor compliance. This has led many countries to introduce monitoring and surveillance teams. Failure of therapy may also take years to emerge due to the slow nature of the disease. In addition, the emergence of resistance of *M leprae* to the major anti-leprosy drugs has also posed a problem in the past. The most commonly reported resistance is to dapson, followed by rifampicin and, more recently, ofloxacin. However, the use of multidrug regimes has greatly reduced the incidence of relapse which has been 0.77% over 9 years (6). No resistance of the bacillus to multidrug therapy has been detected (4). The elimination programme has also emphasized the need for patients to know the earliest signs and symptoms of relapse. These should be reported immediately to the relevant health centres. However, due to the improvements in the control

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programme, it is no longer necessary to continue active surveillance after multidrug therapy.

HIV does not seem to have affected leprosy control as was first feared. Existing data have shown that the response of leprosy in HIV patients to multidrug therapy has been similar to other patients and therefore the management of leprosy in these patients remains the same as in non-HIV patients (5). Vaccination against the leprosy bacillus may also be considered. BCG vaccination is partially effective against leprosy although such a programme worldwide is not cost effective. New and more cost effective vaccines may emerge with advances in molecular engineering.

With this present medical armament and continued surveillance, it is the hope that the elimination of leprosy will be achieved in all countries. It is certainly achievable from a medical point of view. However, social and political

obstacles remain. In the Caribbean, vigilance must be maintained so that the considerable gains made in leprosy control are not lost.

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