

# Determinants of Leprosy with Special Focus on Children: A Socio-Epidemiologic Perspective

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**Abstract** It is well known that clustering and close contact increases risk for leprosy infection which is 5–10 times higher among exposed however dynamics that facilitate such infection is poorly understood. This paper attempts to elucidate domains of poverty that may enhance the risk of the transmission of *M. leprae* and/or facilitate the progress from infection to disease especially among children. There is also attempt to review some of the epidemiological and biological explanations for some Socioeconomic, environmental, and behavioural risk factors for leprosy facilitate the transmission of leprosy.

**Keywords** Leprosy, Social-Epidemiology, Determinants, Risk Factors

## 1. Introduction

In 1991, the 44th World Health Assembly resolved to eliminate leprosy as a public health problem by the year 2000. Elimination of leprosy as public health problem was defined as reduction in the registered prevalence of leprosy patients receiving MDT to less than 1 per 10,000 population. The prevalence of leprosy varies greatly from country to country, but most cases occur in the developing world, with 16 nations (led by India and Brazil) accounting for 92% of all cases; Southeast Asia also contributes significantly to the global caseload. the majority of these (87.6%) occurring in Southeast Asia and India. On 30<sup>th</sup> January, 2006, the Ministry of Health, Government of India formally announced that India achieved the elimination target (leprosy prevalence as on 31<sup>st</sup> December was 0.95 per 10,000). As of March 2006, registered prevalence of leprosy in India was 0.84 per 10,000 population. While 22 states and five union territories reached prevalence levels below 1 per 10,000; six states (Bihar, Chhattisgarh, Jharkand, West Bengal, Orissa and Uttar Pradesh)[1]

By the end of 2000, 108 of the 122 countries originally listed as leprosy endemic by the WHO, Attained the elimination goal at the national level. By the end of 2005, 116 of the 122 leprosy endemic countries had attained the goal. Extra efforts were still needed to achieve the elimination goal at the national level in the remaining six countries. The elimination strategy is based on detecting and treating all leprosy cases with MDT and The global target of eliminating leprosy was reached in 2000 with world prevalence of less than 600,000 cases. According to official

reports received during 2010 from 141 endemic countries and territories, the global registered prevalence of leprosy at the beginning of 2010 stood at 211,903 cases, while the number of new cases detected during 2009 was 244,796[2].

## 2. Epidemiology of Childhood Leprosy

A staggering 70% of the world's leprosy burden is shouldered by India (WHO, 2002). Ironically, one-fourth of those affected are below 15 years of age, a stratum that accounts for 40% of the total population. The observation made in several studies that male cases outnumber females to the order of 2:1. It has been proposed that the female child is brought late for diagnosis and treatment and is a victim of prejudice. More so, they have higher rates of deformity[3].

Leprosy in children can be an indicator of disease prevalence in the general population and its detection helps determining the disease transmissibility. Due to its long incubation period, leprosy characteristically occurs in adults but children can also be susceptible. Hence, they are at higher risk of developing the disease when living in endemic areas and when exposed to family contacts. Ongoing high endemic levels of leprosy suggest children can be exposed to cases that go undetected in the health system. In settings of high disease transmissibility and early exposure to leprosy bacillus people are at higher risk of developing the disease and thus detection rate in this specific age group can be an indicator of more severe endemic disease. Majority of children affected are between 10 to 14 years of age, which could be explained by the long incubation period of leprosy. However, the number of children affected aged one to nine reflects their early exposure to bacilliferous cases. The literature describes higher prevalence of leprosy among male adults and the risk of exposure is a determinant for this gender difference. Among children, there is no gender difference[4].

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The frequency of occurrence in children is an important epidemiological index for determining the level of transmission of the disease in endemic area. Peak of incidence is in the age group of 10-14 years. The reported prevalence of childhood leprosy in high endemic areas of the world varies from 0.012 to 41.6 per thousand children[5].

Leprosy detected in child in a school can be sourced to their families hence follow of their contacts is an efficient method of cases detection of leprosy in general population. In the case of children the source of infection may be case of untreated multibacillary leprosy patient within the family or the community because of the presence of high level of infection in the community to which the children are exposed. As children are more susceptible to infection from affected family members i.e. upto 60% develop the disease. Now there are grounds for believing that transplacental transmission of *M. leprae* may occur as a rare event, thus accounting for a number of reports of clinical leprosy development in infants[6].

However, within the overall ambit of the concept of a School Health Program, teachers are usually the first point of contact of school children and as such, are supposed to be keeping a watchful eye on the health of their pupils. Besides, during routine school health examinations, the services of the teachers are co-opted for conducting various screening activities, rendering the teachers attuned to such activities and sensitive to further orientation[7].

### 3. Risk of Infection

Transmission of leprosy is accepted to be primarily person-to-person: the risk of developing leprosy is 5–10 times higher if one member of the family has developed the disease previously and higher if the primary case has lepromatous leprosy and lower if tuberculoid leprosy. Although a family contact increases the risk of leprosy, in a typical endemic area the majority of new cases cannot be linked to intra-domiciliary contact with a leprosy patient. This suggests the existence of unrecognized human-to-human contacts or more intriguing other modes of transmission. As *Mycobacterium leprae* can persist and possibly proliferate in the environment in association with certain plants and animals it is conceivable that infection may result through prolonged or repeated exposure to an environmental source containing viable bacilli. This is difficult to investigate experimentally because *M. leprae* cannot be cultivated in vitro and evidence can only be obtained indirectly through epidemiological studies[8].

That "contact" (defined in terms of physical proximity, if not actual touching) with a known patient is a risk factor for leprosy has long been recognized. Indeed, given that humans constitute the only known reservoir of *Mycobacterium leprae*, except in those areas with armadillos, it is generally assumed that all diseased individuals must have contracted their infection directly or indirectly from some other infected, if not also diseased. Although it is commonly believed that the

risk associated with household contact reflects the intimacy of contact within the home, it could as well reflect other risk factors shared by household members, such as genetic traits, behavior, diet, inter-current infections, or some physical feature of the house or its surroundings (including possible environmental sources of *M. leprae*). Very few studies have attempted to disentangle these factors. Most studies have not distinguished between families and household members, thus confusing genetic with household proximity. The only study that has attempted to separate any of these factors, one based on data from Uganda, concluded that the apparent clustering among closest relatives could be explained by their more intimate household contact[9].

Apart from undetected or hidden leprosy patients, other major sources of ongoing transmission are likely to be those who are infected subclinically with *M. leprae*; especially people incubating multibacillary disease. There is increasing evidence from nasal PCR studies that subclinical transmission may exist and that those infected may go through a transient period, not resulting in disease development, but allowing transmission of infection to other individuals by nasal excretion[10].

Only 5-10% of the population is susceptible to develop the disease. The attack rate among close contacts is around 5%. Lepromatous and borderline cases are infectious while indeterminate and tuberculoid are considered as non-infectious. The disease is not hereditary but a genetic susceptibility may be inherited as shown by the aggregation of cases in some families. Twin data seem to indicate that the concordance of the types of leprosy is higher among pairs of identical twins than among pairs of fraternal twins. HLA linked genes seem to control the type of disease that develops. Incubation The bacillus reproduces at a very slow rate and therefore the incubation period is an average of 3 to 5 years. It is difficult to find out precisely the incubation period because exposure time and degree of exposure are impossible to determine. Few cases were diagnosed in infants less than 1 year, for other cases the incubation may have lasted 20 years or more[11].

As a maximum of about 5% develop manifest clinical leprosy even in the most hyperendemic areas of the world, it is likely that the initial encounter with the leprosy bacillus will have led to immune protection in the other 95%. This being so, the mode of spread of the organism previously assumed to be skin to skin is likely to be from respiratory tract to respiratory tract, a situation analogous to tuberculosis (Shepard, 1962), explaining why less than 50% of leprosy patients know of any occupational or household contact with leprosy patients when they present for diagnosis[12].

### 4. Clinical Manifestation

In an exposed person who is susceptible to leprosy, a single skin lesion may develop after an incubation period averaging 2 to 4 years (range 3 months to 40 years). The clinical spectrum, in order of decreasing cell-mediated immune response to *M. leprae*, are tuberculoid leprosy (TT),

characterized by few skin lesions and low bacterial loads, borderline tuberculoid leprosy (BT), borderline leprosy (BB), borderline lepromatous leprosy (BL) and lepromatous leprosy (LL), characterized by diffuse skin lesions and high bacterial loads. Leprosy can also be classified according to the number of skin lesions present and the number of bacilli found on slit-skin smear examination. paucibacillary disease (indeterminate, TT and BT forms) is defined as fewer than 6 skin lesions with no bacilli on slit-skin smear testing. Multibacillary disease (BB, BL and LL forms) is characterized by 6 or more lesions with or without positive skin smear results.

Leprosy typically presents with anesthetic skin lesions associated with thickened peripheral nerves. The appearance of the skin lesions varies according to the spectrum of disease. The lesions of indeterminate leprosy tend to be hypopigmented and ill-defined, and they heal on their own in approximately 75% of cases; consequently, they are ignored by many patients. Tuberculoid form on the other hand typically present with just a few (less than 6) asymmetrically distributed, well-circumscribed skin lesions, with elevated margins and marked hypopigmentation. The lesions have a dry, scaly appearance, with impairment of sweating because of disruption of autonomic nerve function; they are typically hairless and anesthetic. Enlargement of a single nerve is common, and marked nerve damage can occur early in the course of tuberculoid disease, often resulting in wrist drop, clawing of the hand and foot drop. Patients at the lepromatous pole present with skin lesions that are widely and symmetrically disseminated, often demonstrating only slight hypopigmentation or erythema [13] (Fig. 2B). The lesions have a smooth, shiny surface, and impaired sweating and hair growth are late features, as is loss of sensation. Nerve damage tends to be slow but progressive. If untreated, lepromatous disease progresses, and the affected skin begins to thicken, predominantly in the forehead, earlobes, eyebrows and cheeks, which eventually leads to the classic leonine (lion-like) facies [13].

Clinical feature of leprosy in children may sometime be confusing. Sensory testing is difficult in them and slit skin smears are usually negative. Histopathological may be rewarding in early tuberculoid and indeterminate leprosy and there may be marked disparity between clinical and histopathological features. It has been suggested that clinical spectrum of leprosy is incomplete in 0-14 year age group [14]. Most children have single lesion and reveals difficulties in classifying children with single large patch. There is no system for followups of children with MB who migrate out of the area. This leaves possibility of their transmitting the disease to others. Girls drop out of the school hence are missed in school survey [15].

Many studies have shown tuberculoid form as the most common clinical presentation, followed by dimorphous, indeterminate and Lepromatous, although in children paucibacillary forms (indeterminate and tuberculoid) are expected to be the most frequent ones due to the incubation period. The indeterminate form is the early presentation of

leprosy and it may progress to either spontaneous cure or polarized forms. The predominance of non-contagious forms is most often reported among children, but multibacillary forms have also been detected in endemic areas. The literature shows that the diagnosis of leprosy is usually made after the age of three. [4]

Deformity affecting children with MB disease are significantly more frequent than in PB cases. Studies have found a significantly higher incidence of nerve function impairment in MB as compared to PB leprosy patients. High disability rates among MB patients is explained by wide spread nerve damage after several years of exposure in lepromatous cases and due to extensive large nerve involvement in borderline cases compared to localized nerve involvement in PB Cases. Children with MB leprosy are found to be higher risk of several reactions and require a regular follow up and prompt intervention for deformities. Higher bacterial load increases the risk for deformities. Reactional episode are reportedly less frequent in children in the range of 20-30%. Such patients are at increased risk of developing deformity (14 times). In a hospital study incidence of deformity among leprosy children found to be 15 % which could be less in community study. Children presenting with deformity indicates delayed diagnosis, reluctance to treatment, stigma, access issues [16]

## 5. Diagnosis

Infection with *M. leprae* is detected by lymphocyte transformation test, immunogel diffusion, radioimmunoassay and fluorescent antibody absorption test (FLA-ABS). An ELISA test using a highly specific phenolic glycolipid (PGL) antigen of *M. leprae* is more sensitive but slightly less specific than the FLA-ABS. Although the results of these tests are diverse, they show that infection with *M. leprae* is far more common than it is generally accepted: From 5% to 50% of contacts. A positive ELISA is present in 95% of cases 1 or 2 years before the development of clinical signs. There is good evidence that a high proportion of early clinical leprosy (up to 12%) will resolve spontaneously. Infection. Minority susceptible to leprosy disease (5%) Majority will not develop disease. Although physical examination, skin smear and biopsy are all used for the diagnostic, subjective diagnoses are sometimes made depending on clinical and histological impressions. The following criteria are required:

1. Negative skin smears for one year
2. Absence of clinical activity and/or reactions
3. Negative skin biopsy after one year.

Inactivity does NOT mean that all *M. leprae* have been killed in the body. It means that they can not be found by the usual methods; however, special studies may show the existence of some viable bacilli [11].

It is widely believed that poor housing conditions, inadequate nourishment, overcrowding in homes particularly night times and poor sanitation facilitate the transmission of

leprosy. Disability rates in children are lower compared to adults because of shorter duration and milder form and infrequent occurrence of reaction in children. Occurrence of deformity increases with age with is exacerbated by onset of puberty[17].

## 6. Poverty as Determinant for Leprosy Transmission

Although it is well established that leprosy is associated with poverty, it is important to elucidate aspects of poverty that may enhance the risk of the transmission of *M. leprae* and/or facilitate the progress from infection to disease. Certain socioeconomic, environmental, and behavioural risk factors for person-to-person transmission—such as crowding or sharing the bed or hammock with other household members also Low education level, the experience of food shortage, frequent contact with natural water bodies previously have been shown as significant risk factors favouring the occurrence of leprosy in an endemic area. Low Education is generally associated with lower economic stratum of society that share other health hazards, including lack of health education and access to health care. Hence low education as a distant determinant of leprosy. Food shortage determined by history of suffering from hunger are likely to have experienced nutritional deficiencies in previous periods of their life. It is conceivable that inadequate nutrition weakens the immune competence against infection and, thereby, the infection with *M. leprae*. The low frequency of changing bed linen is related to water shortage, poverty, and hygiene. It is mainly the consequence of water shortage that is much more frequent in the poorest areas or irregular change of bed-linen may be a behavioural characteristic linked to inappropriate hygiene perception. *M. leprae* can survive out of the human body for several months even under unfavourable conditions. It is possible that this behaviour could maintain the *M. leprae* in the bed or hammock and facilitate longer contact and transmission to the user. Water shortage increase risk for leprosy as people tend to concentrate around some scanty source of water. Another variable with a strong association with leprosy was frequent contact with water bodies such as creeks, rivers, ponds, or lakes during non rainy season transform into swamps and become a habitat for a variety of plants and small animals. It is well demonstrated that in water plants such as *Sphagnum* species suggested as a reservoir for *M. leprae*. People walk barefooted and have to cross rivers and swamps to reach their fields or neighbouring villages. walking barefooted facilitate the infection with *M. leprae*. There are evidence to show that the prevalence of leprosy among individuals who used water sources containing *M. leprae* for bathing and washing clothes or dishes was significantly higher than that among individuals who used water free of *M. leprae*[8]

## 7. Role of BCG Vaccination in Leprosy

Meta-analysis of several experimental studies and observational studies demonstrated that BCG reduces clinical leprosy among vaccinees. The average protective effect among the experimental studies was 26% and among the observational studies was 61%. BCG vaccination offered greater protection against multibacillary leprosy. However, given the protective effects and its easy availability, additional BCG vaccination may be warranted, at least among the household contacts of multibacillary patients who are at greater risk of acquiring the infection[18].

Thus, it has been concluded that the protective effect of BCG has generally been modest. By itself, BCG vaccination is no more considered to be a modality for immunoprophylaxis of leprosy. Although *M. leprae* is yet to be cultivated, it grows profusely in armadillos. It showed that the immunogenicity of *M. leprae* (which is a weak immunogen) is enhanced by the addition of BCG and subsequently established the concept of a mixed vaccine containing a mixture of heat-killed armadillo-derived *M. leprae* (*M. leprae*-A') + BCG[19].

## 8. Conclusions

Leprosy programme management which is currently focused on implementation of multidrug therapy need to develop strategies to address Socioeconomic, environmental, and behavioral risk factors for leprosy to achieve the goal of leprosy elimination.

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