CHAPTER – III
LEPROSY: A REVIEW

3.0 Introduction

Through history, people have lived in cities, urban areas where traders gathered to sell their wares and soldiers returned from wars. Neighbors met on the street and in each other, some. The aristocracy could not avoid contact with peasants. Every day, people of all kinds mixed together.

Within this mix, disease spread more easily than it did in rural areas. The life expectancy of the leprosy did in rural areas. The life expectancy of the citizens of ancient Rome, for example, was much shorter than that of people living in the far reaches of Rome Empire. Only about one-third of city-dwelling Romans lived to be thirty years of age. In outlying areas, 70 percent of the population reached thirty. Some 15 percent lived to be eight years of age.

The close human contact created by living conditions in the city made the spread of infectious diseases-diseases that can be “caught”-nearly impossible to control. Houses were built right next to each other. Rats ran freely from home to home, carrying fleas, ticks, and other insects that could cause illness. People crowded into small rooms, where a cough or a sneeze sent bacteria (microorganisms that cause sickness) through the air from one person to another.

During the Middle Age (the fifth century to the fifteenth century) in Europe, disease terrorized people. No one knew what caused bubonic plague, tuberculosis, or leprosy. Some people believed that God sent the disease to punish people for their sins.

Their wondered it Satan was at work. Sometimes, groups of innocent people were killed by angry mobs who blamed them for causing disease.

The terror continued until the late seventeenth century, when the microscope was invented. Before that, no one knew that bacteria existed. These tiny organisms cannot be seen with the naked eye. Even when Norwegian biologist Gerhard Henrick Armaure Hansen first discovered in 1873 the bacteria that cause leprosy, he had result in such a horrible disease. However, his discovery led to further research and eventually to a cure. Today, leprosy is also called Hansen’s disease in his honor.
Through scientific advances, researchers have now discovered the bacteria or viruses that cause many infectious diseases. Drugs have been developed to treat and cure these diseases. For many today, vaccines can prevent infection.

But new diseases still appear, no less terrifying than the plagues of the Middle Ages. AIDS, which destroys the body’s immune system—its ability to fight off infection—has killed millions around the world. AIDS can now be better controlled by treatment with drugs, although there is no cure. But in poorer countries, especially those in Africa, the drugs are not widely available. People continue to die by the thousands.

Like AIDS, today leprosy can be controlled by drugs. But in poor countries, people still are not able to get the care they need. The same conditions of overcrowding, malnutrition, and poor sanitation that caused the spread of leprosy in cities in the Middle Ages still exist today in places like India and Brazil.

### 3.1 Definition

Leprosy chronic granulomatous communicable disease occurring in tropical and subtropical regions; characterized by inflamed nodules beneath the skin and wasting of body parts; caused by the bacillus Mycobacterium leprae (Webster's Revised Unabridged Dictionary)

Leprosy is a chronic, infectious disease caused by M. Leprae. It affects mainly the skin and the peripheral nerves.

A chronic, mildly contagious disease of tropical and subtropical regions, caused by the bacillus Mycobacterium Leprae, marked by lesions of the skin and mucous membranes and damage to peripheral nerves and other organs that, if untreated, can progress to disfigurement, lack of sensation, and blindness. Also called Hansen's Disease. A Chronic, slowly progressing, use. Mildly infectious disease caused by the bacillus Mycobacterium Leprae, marked by destruction of tissue and loss of sensation and characterized in persons with poor resistance by numerous inflamed skin nodules and in persons with better resistance by local areas of firm, dry patches. Also called Hansen's disease. (Random House Kernerman Webster's College Dictionary, 2010)

A disease caused by a bacterium that damages nerves, skin, and mucous membranes. Leprosy progresses slowly, but if untreated it can destroy the affected body tissues. (The American Heritage Dictionary of Student Science, 2014)
3.2 Illustrative Leprosy

It is an infectious disease that develops very slowly. It is caused by germs (bacilli) that affect mostly the skin and nerves. It can cause a variety of skin problems, loss of feeling, and paralysis of the hands and feet:

Fig No. 3.2: Illustrative Leprosy

Leprosy is a disease caused by the bacteria Mycobacterium leprae, which causes damage to the skin and the peripheral nervous system. The disease develops slowly (from six months to 40 years!) and results in skin lesions and deformities, most often affecting the cooler places on the body (for example, eyes, nose, earlobes, hands, feet, and testicles). (Medgyn, 2016) The skin lesions and deformities can be very disfiguring and are the reason that infected individuals historically were considered outcasts in many cultures. Although human-to-human transmission is the primary source of infection, three other species can carry and (rarely) transfer M. leprae to humans: chimpanzees, mangabey monkeys, and nine-banded armadillos. The disease is termed a chronic granulomatous disease, similar to tuberculosis, because it produces inflammatory nodules (granulomas) in the skin and nerves over time.

3.3 Terminological Development

3.3.1 Etymology

The word leprosy comes from ancient Greek Λέπρα [léprâ], "a disease that makes the skin scaly", in turn, a nominal derivation of the verb Λέπω (lérō), "to peel, scale off". Λέπος (Lepos) in ancient Greek means peel, or scale, so from Λέπος we have Λεπερός (Leperos) = ho has peels—scales) and then Λεπρός (=leprous). (Greek Dictionary, 1999) The word came into the English language via Latin and old French. The first attested English use is in the Ancrene Wisse, a 13th-century manual for nuns ("Moyseses hond..bisemde o þe spitel uuel & þuhte lepruse." The Middle English Dictionary, s.v., "leprous"). A roughly
contemporaneous use is attested in the Anglo-Norman Dialogues of Saint Gregory, "Esmondez i sont li lieprous" (Anglo-Norman Dictionary, s.v., "leprus").

Throughout history, individuals with leprosy have been known as lepers. In the 21st century, this term is falling into disuse as a result of the diminishing number of leprosy patients. Because of the stigma to patients, some prefer not to use the word "leprosy," preferring Hansen's disease. The term "leprosy" is still used by the U.S. Centers for Disease Control and Prevention and the World Health Organization. (Wikipedia, 2015)

3.4 Overview of Some Country

3.4.1 Rome

In the West, the earliest known description of leprosy there was made by the Roman encyclopedist Aulus Cornelius Celsus (25 BC – 37 AD) in his De Medicina; he called leprosy "elephantiasis". The Roman author Pliny the Elder (23–79 AD) mentioned the same disease. Although "sara't" of Leviticus (Old Testament) is translated as "lepra" in the 5th century AD Vulgate, the original term sara't found in Leviticus was not the elephantiasis described by Celsus and Pliny; in fact, sara't was used to describe a disease which could affect houses and clothing. Katrina C. D. McLeod and Robin D. S. Yates state that sara't "denotes a condition of ritual impurity or a temporary form of skin disease." (Dwivedi & Dwivedi 2007)

3.4.2 Persia (Iran)

The Persian polymath Avicenna (c. 980–1037) was the first outside of China to describe the destruction of the nasal septum in those suffering from leprosy.

3.4.3 Middle Ages

Numerous leprosaria, or leper hospitals, sprang up in the Middle Ages; Matthew Paris, a Benedictine Monk, estimated that in the early thirteenth century there were 19,000 across Europe. (Kutumbian, 2005) The first recorded Leper colony was in Harbledown. These institutions were run along monastic lines and, while lepers were encouraged to live in these monastic-type establishments, this was for their own health as well as quarantine. Indeed, some medieval sources indicate belief that those suffering from leprosy were considered to be going
through Purgatory on Earth, and for this reason their suffering was considered holier than the ordinary person's. More frequently, lepers were seen to exist in a place between life and death: they were still alive, yet many chose or were forced to ritually separate themselves from mundane existence. (Robbins, et al. 2009) The Order of Saint Lazarus was a hospitaller and military order of monks that began as a leper hospital outside Jerusalem in the twelfth century and remained associated with leprosy throughout its history. The first monks in this order were leper knights and they originally had leper grand masters, although these aspects of the order changed over the centuries. Radegund was noted for washing the feet of lepers. Orderic Vitalis writes of a monk, Ralf, who was so overcome by the plight of lepers that he prayed to catch leprosy himself (which he eventually did). The leper would carry a clapper and bell to warn of his approach, and this was as much to attract attention for charity as to warn people that a diseased person was near.

3.4.5 India

The Oxford Illustrated Companion to Medicine holds that the mention of leprosy, as well as cures for it, were already described in the Hindu religious book Atharva-veda. (Robbins, Tripathy, Misra, Mohanty, Shinde, et al., 2009) Writing in the Encyclopedia Britannica 2008, Kearns & Nash state that the first mention of leprosy is in the Indian medical treatise Sushruta Samhita (6th century BC). The Cambridge Encyclopedia of Human Paleopathology (1998) holds that: "The Sushruta Samhita from India describes the condition quite well and even offers therapeutic suggestions as early as about 600 BC" (Shinde, 2009) The surgeon Sushruta lived in the Indian city of Kashi by the 6th century BC, (Robertson, 2009) and the medical treatise Sushruta Samhita—attributed to him—made its appearance during the 1st millennium BC. The earliest surviving excavated written material which contains the works of Sushruta is the Bower Manuscript—dated to the 4th century AD, almost a millennium after the original work. Despite the existence of these earlier works the first generally considered accurate description of the disease was that of Galen of Pergamum in 150 AD. In 2009, a 4,000-year-old skeleton was uncovered in India that was shown to contain traces of leprosy. (Gould, 2005) The discovery was made at a site called Balathal, which is today part of Rajasthan, and is believed to be the oldest case of the disease ever found. This pre-dated the previous earliest recognized case, dating back to 6th-century Egypt,
by 1,500 years. (Kortschak, 2010) It is believed that the excavated skeleton belonged to a male, who was in his late 30s and belonged to the Ahar Chalcolithic culture. Archaeologists have stated that not only does the skeleton represent the oldest case of leprosy ever found, but is also the first such example that dates back to prehistoric India. This finding supports one of the theories regarding the origin of the disease, which is believed to have originated in either India or Africa, before being subsequently spread to Europe by the armies of Alexander the Great. In 1881, around 120,000 leprosy patients existed in India. The central government passed the Lepers Act of 1898, which provided legal provision for forcible confinement of leprosy sufferers in India. (McCurry, 2004)

3.4.6 China

Regarding ancient China, Katrina C. D. McLeod and Robin D. S. Yates identify the State of Qin's Feng zhen shi 封診式 (Models for sealing and investigating), dated 266-246 BC, as offering the earliest known unambiguous description of the symptoms of low-resistance leprosy, even though it was termed then under li 癬, a general Chinese word for skin disorder. This 3rd century BC Chinese text on bamboo slip, found in an excavation of 1975 at Shuihudi, Yunmeng, Hubei province, not only described the destruction of the "pillar of the nose", but also the "swelling of the eyebrows, loss of hair, absorption of nasal cartilage, affliction of knees and elbows, difficult and hoarse respiration, as well as anesthesia."

3.4.7 Japan

Japan has had a unique history of segregation of patients into sanatoriums based on leprosy prevention laws of 1907, 1931 and 1953, and hence, it intensified leprosy stigma. The 1953 law was abrogated in 1996. There are still 2717 ex-patients in 13 national sanatoriums and 2 private hospitals as of 2008. In a document written in 833, leprosy was described as "caused by a parasite which eats five organs of the body. The eyebrows and eyelashes come off, and the nose is deformed. The disease brings hoarseness, and necessitates amputations of the fingers and toes. Do not sleep with the patients, as the disease is transmittable to those nearby." This was the first document concerning infectivity. (Rafferty, 2005)
3.5 Scope

Leprosy damages the nerves in the cooler parts of the body, especially those near the skin that relate to the hands, feet and face. If treated during the early stages there will be no loss of sensation or paralysis but if the nerves are damaged, then feeling and movement will not return. Leprosy can affect people in many ways, not just physically. In some countries, largely due to myths and superstitions, there is a great deal of fear associated with leprosy people diagnosed with the disease can be stigmatised, rejected by their families and communities, they may lose their jobs and end up without a home or source of income. The Leprosy Mission cares for the whole person we are a holistic charity, focusing on the physical, social, spiritual, and psychological needs of leprosy-affected people.

Fig. No. 3.5: Places where superficial nerve trunks can be palpated

3.6 Division and Subdivision

3.6.1 Types of Leprosy

Fig. No. 3.6.1: Types of Leprosy
The current accepted classification for leprosy was made by Ridley and Jopling in 1966. It was based on clinical, histological and immunological criteria. It subdivided leprosy in groups: tuberculoid (T), borderline tuberculoid (BT), borderline (BB), borderline lepromatous (BL) and lepromatous (L). A minor form was added later: indeterminate (I). As it is well known, leprosy forms does not depend of the bacilli itself but on the immune response of the host. In LL macrophages are unable to kill bacilli; on the other hand, in BL there is an intense cell-mediated immunity response able to destroy bacilli. There is also a clinical presentation of leprosy without any skin lesions known as pure neuritic form (PNL) (Brakel, Nicholls & Das, 2005). There is also a condition known as “silent neuropathy” (SN). It is characterized by the impairment of sensory and motor functions without skin signs, nerve tenderness, pain, paraesthesia or numbness symptoms of neuritis. It is also called “quiet nerve paralysis” (Jardim & Antunes, 2003).

3.6.2 Paucibacillary (PB) Leprosy

- Skin lesions with definite loss of sensation
- Maximum of one nerve trunk enlarged

3.6.2.1 Indeterminate leprosy (IL)

This early form causes one to a few hypopigmented, or sometimes erythematous, macules. Sensory loss is unusual.

Most cases evolve from this state into one of the other forms, depending on the patient's immunity to the disease. Those with strong immunity may become cured of disease. In some, the disease may persist in this indeterminate form. In those with weaker immunity, the disease progresses to one of the other forms.

3.6.2.2 Tuberculoid leprosy (TT)
Skin lesions are few in number. Usually, one erythematous large plaque is present, with well-defined borders that are elevated and slope down into an atrophic center. The lesions can become arciform or annular, and they can be found on the face, limbs or elsewhere, but spare intertriginous areas and the scalp.

Another presentation involves a large asymmetric hypopigmented macule. Both types of lesions are anesthetic and involve alopecia.

Spontaneous resolution can occur in a few years, leaving pigmentary disturbances or scars. Progression can also occur, leading to borderline-type leprosy. In rare instances in which a patient is untreated for many years, the lepromatous type can develop. Neural involvement is common in TT; it leads to tender, thickened nerves with subsequent loss of function. The great auricular nerve and superficial peroneal nerves are often prominent.

3.6.2.3 Borderline tuberculoid leprosy (BT)

Lesions in this form are similar to those in the tuberculoid form, but they are smaller and more numerous. The nerves are less enlarged, and is less alopecia is present. Disease can remain in this stage, convert back to the tuberculoid form, or progress.

3.6.3 Multibacillary (MB) Leprosy

- Six more skin lesions with definite loss of sensation
- More than one nerve trunks enlarged
3.6.3.1 Borderline borderline leprosy (BB)

Cutaneous lesions consist of numerous, red, irregularly shaped plaques that are less well defined than those in the tuberculoid type. Their distribution may mimic those of the lepromatous type, but they are more asymmetric. Anesthesia is only moderate. Regional adenopathy may be present. Disease may remain in this stage, improve or regress.

3.6.3.2 Borderline lepromatous leprosy (BL)

Lesions are numerous and consist of macules, papules, plaques, and nodules. Annular punched-out-appearing lesions that look like inverted saucers are common. Anesthesia is often absent. As with the other forms of borderline leprosy, the disease may remain in this stage, improve, or regress.
3.6.3.3 Lepromatous leprosy (LL)

Fig No. 3.6.3.3: Lepromatous leprosy (LL)

Early cutaneous lesions consist mainly of pale macules. Later, infiltrations are present, with numerous bacilli. Macular lesions are small, diffuse, and symmetric. The skin texture does not change, and little or no loss of sensation occurs. The nerves are not thickened, and sweating is normal. The lateral eyebrows are affected by alopecia (i.e., madarosis), which spreads to the eyelashes and then the trunk. Scalp hair remains intact. Lepromatous infiltrations can be diffuse, nodules (called lepromas), or plaques. The diffuse type results in the appearance of a leonine facies. Neuritic lesions are symmetric and slow to develop. Eye involvement occurs, causing pain, photophobia, decreased visual acuity, glaucoma, and blindness. Testicular atrophy results in sterility and gynecomastia. Lymphadenopathy and hepatomegaly can result from organ infiltration. Stridor and hoarseness are a result of laryngeal involvement. Nasal infiltration can cause a saddle-nose deformity. Aseptic necrosis and osteomyelitis can occur with repeated trauma after joint invasion. Brawny edema of the lower extremities is a late finding. Unlike the other types of leprosy, LL cannot convert back to the less severe borderline or tuberculoid types of disease. Lepra reactions are complications that occur in 50% of patients after the initiation of therapy or, occasionally, before therapy.
3.7 Causes of Leprosy

Fig No. 3.7: Causes of Leprosy

Leprosy is caused mainly by Mycobacterium Leprae, a rod-shaped bacillus that is an obligate intracellular (only grows inside of certain human and animal cells) bacterium. M. Leprae is termed an "acid fast" bacterium because of its chemical characteristics. When special stains are used for microscopic analysis, it stains red on a blue background due to Mycolic acid content in its cell walls. The Ziehl-Neelsen stain is an example of the special staining techniques used to view the acid-fast organisms under the microscope.

It can spread only from some persons who have untreated leprosy, and only to other persons who have ‘low resistance’ to the disease. It is probably spread either through sneezing or coughing, or through skin contact. Most persons who come into contact with leprosy have a natural ability to resist it. Either they do not get it at all, or they get a small unnoticeable infection that soon goes away completely.

From the time a person is first infected with leprosy germs, it often takes 3 or 4 years for the first signs of the disease to appear. Leprosy is not caused by evil spirits, by doing something bad, by eating certain foods, or by bathing in river water, as some people believe. It is not hereditary and children of mothers with leprosy are not born with it. However, children who live in close contact with someone who has untreated leprosy are more likely to get it.

3.7.1 Transmission to Animals

3.7.1.1 Mouse

In 1960, Shepard first reported that the leprosy bacillus may multiply in the mouse footpad in a limited way and the era of experimental leprosy was thus ushered in (Shepard, 1960).
By suppressing the immune mechanism with a Thymectomy and whole-body 900 r X-ray irradiation (T/900 r), Rees and Weddell were able to increase the yield of leprosy bacilli per footpad up to 1000-fold. More than 200 strains of M. Leprae derived from patients from all over the world have produced an identical type of infection when inoculated into the footpads of mice. (Weddell, REES, 1968)

3.7.1.2 Nude Mouse

In 1976, Colston and Hilson, and Kohsaka et al. first reported on the growth of M. Leprae in the footpad of the nude (Athymic and with no hair) mouse. The nude (hairless) mouse is deficient in T-cells and is extremely sensitive to infection with M. Leprae. The nude mouse model can detect as little as 100 viable bacilli among an inoculum of 108 dead bacilli. A heavy infection of skin, ears, testes, liver, lymph nodes and spleen is present in the nude mouse. The organisms can grow up to a load of 1010 per gram of tissue. In addition to the subcutaneous or intradermal routes of inoculation, leprosy can be experimentally transmitted to nude mice through the nasal mucosa, either by aerosols or direct application (Bryceson, Pfaltzfraff, 1990)

3.7.1.3 Armadillo

In 1971, Kirchheimer and Storrs reported a disseminated experimental M. Leprae infection in the ninebanded armadillo. Some of the biological features of this animal are particularly interesting for leprosy investigations, namely: low body temperature (30–36°C) and a long life-span (12–15 years). Some 40% of the armadillos inoculated with M. Leprae develop leprosy-like systemic infection after about 1 year. The infected armadillo’s liver may contain as many as 1000 million bacilli. Some of the biological features of this animal are particularly interesting for leprosy investigations, namely: low body temperature (30–36°C) and a long life-span (12–15 years). Some 40% of the armadillos inoculated with M. Leprae develop leprosy-like systemic infection after about 1 year. The infected armadillo’s liver may contain as many as 1000 million bacilli.
(109) bacilli per gram of tissue. M. Leprae can also be transmitted to the seven-
and eight-banded armadillo species. The large quantities of M. Leprae which can
be obtained from infected armadillos are used for immunological studies. The
armadillo is also a suitable animal model for leprosy research. (Kirchheimer &
Storrs, 1971)

3.7.1.4 Monkeys

Gormus and his associates (USA) transmitted leprosy to 24 mangabey
monkeys, 7 rhesus monkeys and 5 African green monkeys. All of the monkeys had
the multibacillary type of leprosy and approximately 50% of them developed
squelch due to damage to peripheral nerves.

3.8 Symptoms of Leprosy

Leprosy primarily affects the skin and the nerves outside the brain and
spinal cord, called the peripheral nerves. It may also strike the eyes and the thin
tissue lining the inside of the nose. The main symptom of leprosy is disfiguring
skin sores, lumps, or bumps that do not go away after several weeks or months.
The skin sores are pale-colored.
Nerve damage can lead to:

- Loss of feeling in the arms and legs
- Muscle weakness
- Burning sensations in the skin
- Pale patches on the skin with loss of feeling
- Numbness and tingling of the feet and/or hands
- Weakness of eyelids, hands or feet
- Tender nerves
- Painless swellings or lumps, especially on the face and ear lobes
- Painless wounds or burns on the hands or feet.

It usually takes about 3 to 5 years for symptoms to appear after coming into
contact with the leprosy-causing bacteria. Some people do not develop symptoms
until 20 years later. The time between contact with the bacteria and the appearance
of symptoms is called the incubation period. Leprosy's long incubation period
makes it very difficult for doctors to determine when and where a person with
leprosy got infected. (Steaven, 2015)
3.9 Diagnosis of Leprosy

A diagnosis of leprosy is made if ONE of the following signs is positive:

- Skin patch with loss of sensation
- One or more peripheral enlarged nerves
- The presence of leprosy bacilli on the slit skin or nasal smear.

In addition the following chemical tests can be used to diagnose leprosy:

- Histamine test
- Lepromin test (Paolo Muthama, 2007)

3.10 Treatment of Leprosy

3.10.1 International Status of Leprosy

Until now, in the fight against leprosy, "multi-drug therapy (MDT)" has proved to be an efficacious and the most cost-effective weapon in the armamentarium of leprosy control programs across the world. Since 1985, the load of leprosy cases globally has reduced from more than 10 million to less than 0.2 million by 31 December 2007 and more than 14.5 million patients have been declared cured of leprosy through MDT. MDT emerged as a winner in the battle against the disease by the end of 2000, when the elimination of leprosy was declared at the global level. By the beginning of 2008, 119 of 122 endemic countries achieved the goal of elimination of leprosy i.e. less than one case per 10,000 population; however, three countries viz. Brazil, Nepal and Timor Leste have yet to eliminate leprosy. In the year 2007, five countries including India contributed more than 80% of the newly detected cases worldwide. (WHO, 2006)

3.10.2 National Status of Leprosy

Govt. of India started National Leprosy Control Programme in 1955 based on Dapsone domiciliary treatment through vertical units implementing survey education and treatment activities. It was only in 1970s that a definite cure was identified in the form of Multi Drug Therapy. The MDT came into wide use from 1982, following the recommendation by the WHO Study Group, Geneva in October 1981. Govt. of India established a high power committee under chairmanship of Dr. M.S. Swaminathan in 1981 for dealing with the problem of leprosy. Based on its recommendations the NLEP was launched in 1983 with the objective to arrest the disease activity in all the known cases of leprosy. However
coverage remained limited due to a range of organizational issues and fear of the
disease and the associated stigma. At this stage in view of substantial progress
achieved with MDT, in 1991 the World Health Assembly resolved to eliminate
leprosy at a global level by the year 2000. In order to strengthen the process of
elimination in the country, the first World Bank supported project was introduced
in 1993.

The 1st Phase of the World Bank supported National Leprosy Elimination
Project started from 1993-94 and completed on 31.3.2000. This Project involved a
cost of Rs. 550 crores of which World Bank loan was Rs. 292 crores. During this
phase, the prevalence rate reduced from 24/10,000 population in 1992 before
starting 1st Phase project to 3.7/10,000 by March 2001.

The 2nd Phase of World Bank Project on NLEP started for a period of 3
years from 2001-02. The project involve a cost of Rs. 249.8 crore including World
Bank loan of Rs. 166.35 Crore and WHO to provide MDT drugs free of cost worth
Rs. 48.00 crore. The project successfully ended on 31st Dec. 2004.

The National Leprosy Eradication Programme is being continued with
Govt. of India funds from January 2005 onwards. Additional support for the
programme is continued to be received from the WHO and ILEP organizations.
MDT is to be supplied free of cost as of now by NOVARTIS through WHO.

In the year 2001, after the global elimination was achieved, a target was
reset for the remaining 14 countries to achieve elimination on national basis by
December, 2005. India was one of these countries. The National Health Policy,
Govt. of India sets the goal of elimination of leprosy.

The National Leprosy Eradication Programme took up the challenge with
the active support of the State/ UT Governments and dedicated partners in the
World Health Organisation, the International Federation of Anti Leprosy
Associations (ILEP), the Sasakawa Memorial Health Foundation & the Nippon
Foundation, NOVARTIES, DANLEP (1986-2003) and the World Bank (1993-
2004).

As a result of the hard work and meticulously planned and executed
activities, the country achieved the goal of elimination of leprosy as a public health
problem, defined as less than 1 case per 10,000 population, at the National Level in
the month of December, 2005. As on 31st December 2005, Prevalence Rate
recorded in the country was 0.95/10,000 population. (NLEP,2010)
3.10.3 Pre-dapsone Era

Practice of using chaulmoogra oil (also called Hydnocarpus oil) for treatment of leprosy in India can be traced back to as early as 600 BC in Sushruta Samhita. It was extracted from the nut of a tree native to India and used to be administered as an ointment or by injection or by mouth. In the early 1870s, another remedy called "gurjon oil" was developed by Surgeon Dougall of the Madras Medical Service for treatment of leprosy. Dougall began prescribing the oil after reports of its successful use in leprosy patients in Venezuela. Gurjon oil was derived from the wood of a tree native to Andaman and Nicobar Islands and was rubbed on the skin. However, the use of gurjon oil was abandoned because patients reported that reaction with chaulmoogra oil were milder and left the skin softer as compared to gurjon oil. Despite the fact that there was little evidence to support the effectiveness of chaulmoogra oil, it continued to be the treatment of choice for leprosy in India until 1946 when Robert Cochrane started using dapsone. (Norton,1994)

3.10.4 Dapsone Era

Guy Faget used dapsone in leprosy patients on a pilot basis in 1941 at the National Leprosarium, Carville, USA. Two years later, Faget and colleagues reported that intravenous administration of sodium salt of diamino-diphenyl-sulfone (DDS) could arrest the progress of leprosy. Their findings introduced great hope for leprosy patients and marked the decline of use of chaulmoogra oil. Robert Cochrane and John Lowe started using dapsone in India and Nigeria, respectively. Later it was proved that DDS was the most active, least toxic form of sulfones against leprae bacilli, and it could be used in field conditions. The review of the literature suggests that the duration of dapsone monotherapy was not fixed and the patients were treated for a period varying from one to more than 20 years.(Lowe,1950)

Thus, introduction of dapsone simplified the treatment by paving the way for ambulatory treatment and changed the face of leprosy dramatically. Unfortunately, the reports of dapsone resistance started surfacing in the early 1960s. (Petti &Rees1964) The first report of primary dapsone resistance was documented in 1977. (Pearson,1977) This was followed by, footpad-proven secondary resistance being reported from an increasing number of countries.
worldwide with a frequency ranging from about 2-3%. With the realization of worldwide increase in dapsone resistance in *M. leprae* in the late '70s, dapsone monotherapy was no longer considered adequate for treatment of leprosy. The surveys sponsored by "Therapeutic leprosy" (THELEP) and others also proved that the epidemic of dapsone resistance was sabotaging the entire leprosy control efforts. Thus, there was a clear and urgent need for safe and practicable combined drug regimens effective in curing leprosy and preventing drug resistance under field conditions. (WHO, 1980)

### 3.10.5 Era of Newer Drugs

The 1960s and 1970s witnessed the discovery of various drugs including clofazimine and rifampicin for treatment of leprosy. The concept of MDT in leprosy arose after the availability of clofazimine and rifampicin and from the experience of rifampicin use in the therapy of tuberculosis. Based on the theoretical considerations, leprologists worldwide started using combined drug regimens for the treatment of leprosy on an experimental basis. Although, by late 1970, many countries including India had realized the importance of combined therapy as a definitive treatment of leprosy they included it in their national leprosy programs only in 1982 or later at the initiative of WHO. Since 1981, Moxafloxacin, macrolides and rifapentine have emerged as potential candidates for wonder drugs against *leprae* bacilli.

### 3.10.6 Multi-drug Therapy Era

In 1981, WHO took a monumental decision and recommended MDT for leprosy in the absence of systematic reviews, randomized control trials, cohort studies with comparators, or case control studies, which would support the use of WHO MDT over dapsone monotherapy. The development of the 1981 Study Group regimens was conceptually a product of THELEP work and discussions. Later, six case series assessing the effects of WHO MDT (monthly supervised rifampicin 600 mg and clofazimine 300 mg, plus daily unsupervised dapsone 100 mg and clofazimine 50 mg) for 24 months supported the WHO recommendations (1981). The original WHO recommendation was to treat the MB patients for two years or until skin smear negativity and to treat the PB patients with rifampicin and dapsone for six months. By 1985, these recommendations were adopted by almost
all countries. At the global level, the treatment of leprosy has not undergone much change since 1982, except for changes in the duration of the therapy. But in India, standard schedule of MDT has gone through many changes both in the duration of therapy and the criteria for classification of patients into paucibacillary and multibacillary for the purpose of therapy.

3.10.7 Role of Surgery

After having achieved the goal of elimination, it is felt that prevention of deformities and disabilities should be given importance so as to facilitate the social and psychological rehabilitation of persons affected by leprosy. Under the Disability Prevention and Medical Rehabilitation (DPMR) plan, a number of hospitals have been identified in India to provide reconstructive surgery (RCS) to the needy ones. It has been envisaged that the cases will be first screened by the medical officer at the Primary Health Centre and also by a Dermatologist/Medical Specialist at a district hospital to assess the suitability of the surgery. At the RCS unit of a tertiary level hospital, the surgeon and physiotherapy technician would further examine these cases in order to decide who would benefit from the surgeries. Also, there is a provision of cash incentive of Rs. 5000/- to be paid to patients below the poverty line for major RCS. However, what is lacking in the DPMR plan is the constitution of a team of specialists from orthopedics, plastic surgery, physical medical rehabilitation and ophthalmology specialties at each of these hospitals. This issue needs to be addressed so as to ensure effective delivery of quality services. (Convit, Aranzazu, Ulrich, Pinardi & Reyes, 1982)

3.11 Drugs in Leprosy

3.11.1 Antimicrobials

Antimicrobials are used to eliminate organisms. The first-line drugs are dapsone, rifampin, and clofazimine. Other antibiotics include minocycline, ofloxacin, and clarithromycin. For drug treatment purposes, infections are classified as paucibacillary or multibacillary. Paucibacillary disease can be treated with a combination of 2 drugs, whereas multibacillary disease requires triple-drug therapy. The length of treatment depends on the type of disease and the access to medicine. The recommendations of the World Health Organization (WHO) and those in the United States are both mentioned here.
3.11.1  Dapsone

Acts by blocking folic acid synthesis and is weakly bactericidal. Was widely used as monotherapy for leprosy until resistance developed. Now used as part of a multidrug regimen to treat leprosy.

3.11.1.2  Rifampin

Bactericidal for M Leprae. Inhibits DNA-dependent RNA polymerase, interfering with bacterial RNA synthesis. Part of multidrug regimen to treat leprosy.

3.11.1.3  Clofazimine

Red, fat-soluble, crystalline dye. Inhibits mycobacterial growth, binds preferentially to mycobacterial DNA. Has antimicrobial properties, but mechanism of action is unknown. Weakly bacteriocidal against M. Leprae. Has anti-inflammatory properties.

3.11.2  Corticosteroids

These are important anti-inflammatory agents used in the treatment of reactional leprosy. Corticosteroids are the reliable only in the treatment of reversal reactions. These medications can be used to treat leprosy reactions when a risk of neurologic deficits exists or when lesions occur in cosmetically important places. They can also be used to treat ENL.

3.11.2.1  Prednisone

May decrease inflammation by reversing increased capillary permeability and suppressing PMN activity. Stabilizes lysosomal membranes and also suppresses lymphocytes and antibody production.

3.11.3  Immunomodulators

These agents are used to modify the immune system response to diverse stimuli.

3.11.3.1  Thalidomide

Immunomodulatory agent that may suppress excessive production of tumor necrosis factor-alpha (TNF-alpha) and downregulate selected cell-surface adhesion
molecules involved in leukocyte migration. Can be used to treat recurrent or refractory ENL.

3.11.4 WHO Multidrug therapy (MDT)

Since 1995, WHO (World Health Organization) has supplied MDT free of cost to leprosy patients in all endemic countries.

**Fig No. 3.11.4: WHO Multidrug therapy (MDT)**

The drugs used in WHO-MDT are a combination of rifampicin, clofazimine and dapsone for MB leprosy patients and rifampicin and dapsone for PB leprosy patients. Among these rifampicin is the most important antileprosy drug and therefore is included in the treatment of both types of leprosy. Treatment of leprosy with only one antileprosy drug will always result in development of drug resistance to that drug. Treatment with dapsone or any other antileprosy drug used as monotherapy should be considered as unethical practice.

3.12 Side Effects of Anti-Leprosy Drugs

The anti-leprosy drugs related side effects are classified as minor and major as highlighted below:

3.12.1 Minor Side Effects

In the event of minor side effects, inform the DTLC or dermatologist on your next visit to the clinic. Continue MDT.

3.12.1.1 Slight itching

This is often caused by Dapsone and should be treated symptomatically with antihistamines.

3.12.1.2 Gastro-intestinal disturbances

These are mostly caused by Clofazimine and include nausea, vomiting, and abdominal pains. To reduce these, give the drug after a meal.
3.12.1.3  **Red urine**

This is caused by Rifampicin and is harmless. No significant action needs to be taken but there is need to reassure the patient.

3.12.1.4  **Red skin, eyes**

This is caused by Clofazimine and is harmless. No action is needed. The patient has no complaints at all apart from the cosmetic effect.

3.12.1.5  **Symptoms similar to severe flu**

This is caused by Rifampicin. Treat symptomatically and reduce the dosage to half until the symptoms have disappeared.

3.12.2  **Major Side Effects**

Refer the patient to the medical officer or DTLC as soon as possible and stop all MDT drugs, if major side effects present.

3.12.2.1  **Jaundice**

This is caused by Rifampicin. Stop all drugs immediately and refer patient to DTLC.

3.12.2.2  **Anaemia**

This is caused by Rifampicin and Dapsone. Rule out other causes of anaemia (parasites, malaria etc). Refer the patient to the medical officer or the DTLC.

3.12.2.3  **Exfoliate dermatitis**

This is caused by Dapsone. The skin is itchy, and later peels off. The patient is very ill. Stop drugs immediately and refer the patient to the medical officer or DTLC or to the nearest hospital.

3.12.2.4  **Fixed drug eruption**

This is caused by Dapsone. Stop Dapsone immediately. The eruption will slowly clear after stopping.
3.13 Document Classification

3.13.1 Classificatory Treatment

This Section is intended to take note of how the subject concerned and its divisions and its divisions and subdivisions are treated by different schemes of classification. The first point of interest is to find out the rank of the subject in the whole universe of subject, as recognized by different classificationists. Leprosy is the study of disease. So the place of leprosy as recognized by most of the classificationists is:

Universe of subjects

Technology (Applied science)
Medical science & Health
Medicine
Disease
Leprosy

3.13.2 Dewey Decimal Classification (21st ed.)

3.13.2.1 Introduction

Melvil Dewey, the profounder of Decimal Classification divides the Universe of Subjects into Ten Main Classes. Main Class 0 (zero) is used for general works on many subjects from many points of view, such as general newspapers and Encyclopedias deal with human knowledge. Main Classes 1-9 consist each of a major discipline or group of related disciplines as under;

000-Generalities
100-Philosophy, Parapsychology and Occultism, Psychology
200-Religion
300-Social sciences
400-Language
500-Natural sciences and Mathematics
600-Technology (Applied sciences)
700-The Arts fine and decorative arts
800-Literature (Belles-Letters) and rhetoric
900-Geography, History and auxiliary disciplines
The system permits further subdivisions to any degree desired, with a continued decimal notation, which consists of the addition, following any set of three digits 000-999, of a decimal point and as many more digits as may be required.

3.13.2.2 Treatment of Leprosy

Leprosy is treated as;

614.546 Leprosy
614.54 Miscellaneous disease
614.5 Incidence of and public measure to prevent specific diseases and kinds of diseases
614 Forensic Medicine; incidence of injuries, Wounds, disease; Public preventive medicine
610 Medical sciences, medicine
600 Technology (Applied sciences)

3.13.2.3 Comment

Dewey did not divide the Universe of Subjects, which should follow the categories of scholars. His aim was to devise a practical piece of machinery for the rapid and mechanical arrangement, filing and finding of books and literary material and he describes the system as a series of pigeon-holes into which material may be fitted. He even went so far as to declare that it mattered less in what pigeon-hole a book was put than that all books on the one and that one should be indexed.

A more serious defect in the eyes of many critics is the fact that the scheme always has a limit of nine main classes, nine main divisions, nine sections and so on. The limited number is due, of course, to the decimal numbering system and it must result in very long numbers for minute subjects. Dewey himself caused some of this congestion by using up nearly all his number sequence for new related subjects.

The Universe of Knowledge is ever growing. Its growth is accelerating. Any new idea or subjects has to be interpolated in the existing universe of subjects so as to preserve helpful sequence among them as viewed by the majority of specialists in the subjects concerned. It will, in turn, help to preserve the APUPA
pattern of arrangement among the compound subjects going with the Basic Subject judged by the requirements of a dynamic equilibrians.

The great merit of the system is in its simplicity and universal appeal of its notation, yet the comments of Bliss continue to echo in our ears that the “Decimal classification is disqualified as an functionally. It does not embody the natural, scientific, logical and educational order. It fails to apply consistently, the fundamental principles of classification.

3.13.3 Universal Decimal Classification (1961)
3.13.3.1 Introduction

UDC is the brain-child of two Belgians—Seneter Henrita Fontaine and Paul Otlet. It goes without saying that UDC is the result of remolding and expansion of the DDC. This was done under the auspices in detail of the Institute International de Bibliographic. The purpose was to adapt it for classifying micro documents and to develop a classified catalogue for them when DDC failed to serve the purpose.

Being the prototype of DDC, the UDC also divided the universe of knowledge into ten main classes more or less in the footsteps of DDC. The introduction of auxiliary tables, signs of combination and analytical subdivisions are special features of UDC. These are all intendeds to permit the subdivisions of the main class numbers in the brief details for use in classifying information for special purpose or special indexes. The abridged English edition of UDC highlights its aim in the following words: “UDC is essentially a practical system for numerically coding information, so designed that any item, once coded and fitted correctly, can be readily found regarded as a philosophical classification of knowledge, nor is the order of subjects of primary importance. What is of greater moment to a scientific classification is that the introduction of an auxiliary apparatus of connection and relation signs, lacking in the original Dewey system, has made UDC really Universal....”.

The UDC is a scheme for classifying the whole field of human knowledge. It can be applied both to the literature, which record knowledge, and to the catalogues, indexes etc., which refer to the literature. The whole field of human knowledge, regarded as unity, is divided into ten main classes denoted by decimal fractions as follows:
0 Science and knowledge, Organization, Computer Science, Information.
1 Philosophy. Psychology.
2 Religion. Theology.
3 Social sciences
4 Vacant
5 Mathematics and natural sciences
6 Applied sciences. Medicine. Technology
7 The arts. Recreation. Entertainment. Sport
8 Language. Linguistic. Literature
9 Geography. Biography. History

3.13.3.2 Treatment of Leprosy

Leprosy is treated as 616-002.73

616-002.73 Leprosy
616-002.7 Granulomatous and nodular inflammation
616-002 Inflammation, Irritation. Engorgement (hyperaemia).
Mucous congestion.
616-00 Morbid processes.
61 Medical Sciences
6 Applied sciences, Medicine, Technology.

3.13.3.3 Comments

The UDC is a scheme for classifying the whole field of knowledge, but primarily as a scheme for the treatment of masses of specialized material in large and specialist libraries. It can be applied both to the literature, which record knowledge, and to the catalogues, indexes etc, which refer to the literature. It enables to be arranged in such a way that all reference to information on a particular subject can be brought together and the information located with the minimum of searching. Finally UDC can be used for indexing, filing or shelving, preparing bibliographies and glossaries or simply as an aid to international communication. Many technical documents, such as British Standards and Indian Standards, contain UDC numbers but the system can also apply to reports, surveys,
conference papers and proceedings, articles, abstracts, newspaper clippings, bibliographies, catalogues, indexes as well as non-book material.

3.13.4 Colon Classification (7th ed.)

3.13.4.1 Introduction

Colon Classification (CC) is a general scheme for classification designed by the late Dr. S.R. Ranganathan. He experimented the application of the scheme during 1924 to 1932 and published its first edition in 1933. Since there have been seven editions namely edition 2 (1939), edition 3 (1950), edition 4 (1952), edition 5 (1957), edition 6 (1962/6) and edition 7 (1987). It is the first scheme entirely based on analytico-synthetic principle. This aim at analyzing first the subject field into constituent elements or facets and then constructing the class number by synthesis. In Dr. S. R. Ranganathan’s word, “The Colon Classification differs from DDC & the volumes of Congress Classification in some fundamental respects. It is their manifest aim to provide a ready made class number for most topics. Hence, such manuals consist, for the most part, of the schedules of classification and their schedules are by several times larger than that of CC. In the CC, however, ready made class numbers are not assigned to topics. The schedules in the CC may be said to consist of certain standard unit schedules. These standard unit schedules correspond to the standard pieces of Meccano apparatus. Even a child knows that by combining these standard pieces in different objects/ways many different objects can be constructed. So also by combining the classes in the different unit schedules in assigned permutation & combinations, the class numbers for all possible topics can be constructed. In this scheme, the function of the colon (:) is like that of the bolts and nut in a Meccano set”.

Ranganathan provides a set of independent tables for subjects, for relations, form and other classification factors, these tables like the parts of a Meccano set can be used for many constructions.

The purpose of adopting the synthetic method is to secure co-extensiveness of subject & class-mark, minuteness of classification is most of the subjects, individualization of every book in a library by assigning to each a specific class mark, infinite hospitality to new subjects, and maximum autonomy for the classifier.
3.13.4.1 Treatment of Leprosy

Leprosy is treated in CC7

L:4241 Leprosy
L:424 Infection Disease
L:42 Infection
L:4 Disease
L Medicine

3.14 Document Classification

3.14.1 Development of Subject

The origin of leprosy has always been a matter of uncertainty and an Indian or African origin for the disease has often been assumed based on historical sources that support an initial spread of the disease from Asia to Europe by the armies of Alexander the Great after 400 BC. Skeletal evidence for the disease was previously limited to 300-400 BC in Egypt and Thailand, till Robbins and colleagues reported on a case of leprosy in a skeleton showing changes associated with leprosy, buried around 2000 BC at the site of Balathal Rajasthan, India (Robbins, Tripathy, Misra, Mohanty, Shinde & Gray, 2009). Early written records giving clinical descriptions generally accepted as being true leprosy date from 600 BC to possibly as early as 1400 BC in India, where a disease called Kushta was distinguished from vitiligo (Monot, Honore, Ganier, Coppe’e & Lacroix, 2005). The ancient medical texts of Sushruta, Charaka and Vagbhata, compiled in the first to the sixth century BC, show that Indian physicians regarded leprosy as a disease that can be cured or alleviated. Sushruta Samhita (600 BC) recommended treating leprosy or kushtha, meaning “eating away” in Sanskrit with oil derived from the chaulmoogra tree; this remained a mainstay of treatment until the introduction of sulphones (Beowne, 1985). Documentation of lesions suggestive of leprosy like, numbness and loss of eyebrows in Chinese documents attest to the spread of the disease eastward to China and subsequently to Japan (Gussow, 1989). The disease was thought to have spread to the Middle East and westward to Greece by the conquering armies or the traders, as evident by the description of Greek physicians of a novel disease called elephantiasis graecorum. Subsequent spread to the Mediterranean basin and Western Europe may have been intensified during the Crusades by Romans (Trautman, 1990). In the Health
Protection era - from antiquity until 1830s, the dominant paradigm was disease prevention through enforced regulation of human behaviour and this was mediated via legislation, cultural practices and religious doctrines(Awofeso, 2011). Segregation was the main strategy for leprosy control in this era. In India, the Laws of Manu (1500 BC) mention various skin diseases translated as leprosy. The Laws prohibited contact with those affected by leprosy and punished those who married into their families. Ancient Indian society marginalized those with leprosy because of several factors: its chronic, potentially disfiguring nature; inconsistently effective therapy; association with sin; and the fear of contagion(Jacob & Franco, 2008). The Mosaic Law stated illness to be a punishment for sin and leprosy was considered to be the punishment for the most heinous sins or crimes. A purification ceremony and four sacrifices were essential before readmission to society was allowed(Short, 1953). Taboos, such as Chinese and African legends associating leprosy with necrophilia and incest, constituted a major action framework during the Health Protection era. The legacies of the Health Protection era in relation to leprosy control were largely negative, with erroneous knowledge about aetiology of leprosy resulting in stigmatisation and social exclusion of those diagnosed with the disease. Unfortunately, social stigma, alienation, and violence against sufferers of leprosy are attitudes that have continued through the ages up to the 20th century and these still exist, though in a diluted form. Feeny gives a number of examples of persecution acts in the early part of the past century. In Japan “no leprosy patients in prefecture” movement started in 1930 in which absolute isolation was supported by the social belief of that day; “leprosy is a shameful disease and the purity (absence of leprosy patients) of the nation should be maintained, thus justifying isolation”. In the United States, laws in some States allowed sheriffs and local health officials to arrest and confine anyone suspected of carrying the disease. In China (1937), 80 victims with leprosy, including women and children, were shot and thrown into a lime pit; and in Korea (1957), a mob beat 10 patients from a leprosarium to death18. Stigmatizing attitudes have even been incorporated into modern law, as demonstrated in India where the Motor Vehicles Act of 1939 forbade the granting of drivers’ license to leprosy sufferers and, until recently, the Indian Christian, Muslim, and Hindu Marriage Acts included leprosy as grounds for divorce(Brown, 2006).
### Table No. 3.14.2: Historical Development of Leprosy

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000 BC</td>
<td>In 2009, a 4,000-year-old skeleton found in Rajasthan, India contained traces of leprosy and is believed to be the oldest case yet discovered.</td>
</tr>
<tr>
<td>1400 BC</td>
<td>India: The laws of Manu, stated in the Vedas, included instructions for the prevention of leprosy, though it was not named.</td>
</tr>
<tr>
<td>600 BC</td>
<td>The first known true description of leprosy and its treatment with chaulmoogra oil in “Sushruta Samhita”, a treatise written in India.</td>
</tr>
<tr>
<td>30 AD(Approx.)</td>
<td>Jesus Heals People with Leprosy</td>
</tr>
<tr>
<td>0 AD(Approx.)</td>
<td>Birth of Jesus Christ</td>
</tr>
<tr>
<td>1084AD</td>
<td>Hospital of St. Nicholas Harbledown in England was founded by Archbishop Lanfranc for the treatment of leprosy-affected people</td>
</tr>
<tr>
<td>1200AD</td>
<td>&quot;Matthew the Parisian,” - Benedictine Monk, estimated that there were 19,000 leprosy hospitals or leprosaria across Europe.</td>
</tr>
<tr>
<td>1869</td>
<td>Wellesley Bailey visits India</td>
</tr>
<tr>
<td>1873</td>
<td>Dr. G. H. A. Hansen of Norway discovers M. leprae bacilli, indicating for the first time that leprosy was not a curse for evil deeds, as it was previously believed.</td>
</tr>
<tr>
<td>1874</td>
<td>“The Mission to Lepers in India” is Born</td>
</tr>
<tr>
<td>1892</td>
<td>Mr. &amp; Mrs. Watt of Guelph, ON, open their home to Wellesley Bailey. The Mission inspires strong Canadians support.</td>
</tr>
<tr>
<td>1940</td>
<td>DOCTORS USE DAPSONE FOR TREATMENT (Sadly, Dapsone only slowed leprosy progression)</td>
</tr>
<tr>
<td>1950</td>
<td>Mission surgeon Dr. Paul Brand pioneers surgeries for claw hand &amp; foot drop. His wife, Dr. Margaret Brand cares for patients with leprosy affected eyes.</td>
</tr>
<tr>
<td>Year</td>
<td>Event</td>
</tr>
<tr>
<td>------</td>
<td>-------</td>
</tr>
<tr>
<td>1982</td>
<td>The World Health Organization (WHO) recommends Multi-Drug Therapy (MDT) to cure leprosy. TLM adopts the use of this new successful drug blend.</td>
</tr>
<tr>
<td>2002</td>
<td>Over 12 million children, women and men have been cured of leprosy since 1985.</td>
</tr>
<tr>
<td>2003</td>
<td>About 4 million people live with the permanent effects of leprosy.</td>
</tr>
<tr>
<td>2010</td>
<td>The London Declaration commits to ending 10 Neglected Tropical Diseases including leprosy, by 2020.</td>
</tr>
<tr>
<td>2014</td>
<td>The Leprosy Mission Canada changes its name to effect:hope to reflect our belief that success starts with hope. Hope allows us to bring healing and love to every aspect of a person’s life, inspired by the compassion of Jesus.</td>
</tr>
</tbody>
</table>

### 3.14.3 Leprosy control programme

Leprosy eradication in India no data were available regarding the prevalence of leprosy prior to 1955. With the progress of National Leprosy Eradication Programme (NLEP), leprosy prevalence became clear and by mid-seventies, extensive data were collected. By 1980, a total of 40 lakh cases were recorded, giving a prevalence rate of 58 per 10,000 population. In 1982, there was a major advance in the treatment of leprosy. The most striking achievement of the programme remains the reduction of prevalence to elimination level.

The first attempt to deal with leprosy as a public health problem was taken up in 1952 by the Gandhi Memorial Leprosy Foundation (GMLF), an institution started under the Gandhi Memorial Trust. At that time, the only method to deal with the disease was to isolate leprosy patients in “leprosy homes” “sanatoria” or “asylums”, however, such places were very few and inadequate. Dapsone was the new drug that had just been introduced. The work first started in Sewagram (Wardha) in 1952, was subsequently replicated in 12 other centres of GMLF in...
different States. It soon became obvious that the SET programme, initiated by GMLF, was scientific, practical and a very effective method for control of the disease, and the Government of India took it up. The National Leprosy Control Programme (NLCP) was started in 1955 and the SET method became the standard procedure for leprosy control in the entire country. Later, the WHO also endorsed the method, and it was adopted worldwide.

The Enhanced Global Strategy for further reducing the disease burden requires endorsement and commitment from everyone working towards the common goal of reducing the disease burden due to leprosy and its detrimental physical, social and economic consequences to move closer to achieving the common dream of “world without leprosy”.

In 2005, the Government took another major step towards expansion of the NLEP. Leprosy work, which had been carried out so far as a vertical programme, was integrated into the general health services. There were no more special leprosy clinics. All hospitals, dispensaries and PHCs had to treat leprosy patients. Further, the field staff of PHCs had to take up case finding and follow up along with their regular duties. This era was also significant for leprosy control in the use of culturally appropriate depictions of people living with leprosy for leprosy fundraising and public awareness campaigns. Integration of leprosy into the general health service has greatly enhanced the scope of leprosy service. By integration, discrimination against leprosy has been set to be removed and the patients have access to the services of ophthalmologists, surgeons, physiotherapists, and general physicians. (Desikan, 2012).

3.14.4 National Leprosy Eradication Program (India):

Since Multi-Drug Therapy Introduction Initially, in India, on the basis of the recommendations of the "Indian Association of Leprologists", the GOI made slight modifications to the WHO MDT regimen for MB patients by adding an optional two-week of daily rifampicin at the start of treatment. However, this recommendation was withdrawn in 1990. The results of the trials conducted by UNDP/World Bank/WHO Special Program for Research and Training in Tropical Diseases through its Scientific Working Group on the Chemotherapy of Mycobacterial Diseases (previously THELEP) indicated that the rate of relapse
was less than one per 1000 person years irrespective of the duration of treatment i.e. less or more than 24 months. On the basis of these findings, duration of MDT was reduced to a fixed duration of 24 months in 1992. (WHO, 1994)

This was further shortened to 12 months in 1998 on WHO recommendations, which were based on the results mentioned below of the research work conducted across the world.

a. The combined effect of clofazimine, rifampicin and dapsone is greater than the bactericidal effect of rifampicin alone. Furthermore, addition of clofazimine and dapsone help in the elimination of rifampicin-resistant mutants in untreated MB patients.

b. Less than 24 monthly doses of MDT were as good as 24 or more doses of MDT in terms of efficacy.

c. A fixed 12-month MDT was effective in preventing relapse.

Till 1988, patients with bacterial index two or greater were categorized as MB and those with BI less than two were categorized as PB cases. From 1988 to 1995, all smear-negative patients were classified as PB on the basis of the recommendations of the WHO Expert Committee on Leprosy, and leprosy cases with more than 10 lesions (skin and nerve lesions involved) were classified as MB. However, all cases with positive skin-smear test were classified as MB, irrespective of the number of skin and nerve lesions. Since 1996, patients with more than five skin lesions are being classified as MB patients and patients with less than five skin lesions are considered as PB patients, for field conditions. However, there is provision for a slit-skin smear wherever facilities are available, and all smear-positive patients are included in the MB group.

At present, standard 12 months' fixed duration therapy for MB patients is being followed all over India and the duration of treatment of PB remains unchanged since its introduction.

3.15 Progress and Future Efforts on Leprosy

Although in the last two decades, the reported global prevalence of active leprosy infection has dropped by almost 90 per cent; yet a parallel drop in the incidence or new case detection has not been seen. From 1994 through 2011, the NCDR has persistently been more than 10 >100,000 new cases annually (WHO, 2012). The last three decades brought a tremendous and hard-
earned success in fighting leprosy, thanks to the impressive co-operation of various highly committed actors from civil society, government, and the private sector.

The Global Strategy (2006-2010) defines sustainability as ‘the capacity of a programme to maintain quality and coverage of services at a level that will provide continuing control and further reduction of a health problem at a cost that is affordable to the programme and the community’ (WHO, 2009). This is a major challenge for leprosy having changed from a well-supported, high priority specialized programme to one that is now mainly integrated within general health and social services. Radical re-thinking is necessary if we want to sustain early case detection, treatment, prevention of disability, and reduction in the consequences of leprosy including stigma. (Smith, 2010) Antileprosy work keeps aiming at rapidly pushing the disease further and further back. Thus, in this context sustaining exactly the same efforts as in the past is not enough and future success will depend on changing familiar patterns and approaches, keeping in mind the resources needed. (Lunau, 2010).

A second view might reveal, for instance, that the leprosy-related portion of a national health budget should not only be based on the current situation, weighing the leprosy burden against that of other diseases. There is a potential risk that this progress will lessen the perception of the benefit in continuing to spend resources on leprosy, as other competing priorities (e.g., human immunodeficiency virus/ acquired immunodeficiency syndrome, malaria, and tuberculosis) may appear to be of relatively greater importance. We should not forget the resurgence of drug-resistant tuberculosis in the USA after public health resources were diverted to other priorities. This implies the need for allocation of appropriate resources to leprosy to enable the care givers to manage permanent disabilities in one to two million individuals around the globe (Bennett, 2008). However, management of leprosy requires both treating the bacterial infection as well as minimizing the potential for permanent nerve damage and subsequent impairment. Thus today’s window of opportunity requires more resources than a short-term analysis would indicate.

Another possibility that needs to be considered is the paradoxical delay in treatment and subsequent increase in the severity of impairment. As a disease or condition becomes more rare, it takes a higher index of suspicion for a treating physician to appropriately diagnose or refer a patient for care. (Molyneux
Leprosy is rare in America and the average time from initial presentation to diagnosis is about two years and during this period of missed diagnosis, there is a risk of avoidable permanent tissue and nerve damage. We can assume that a lowered index of suspicion and delay in diagnosis may lead to increase in proportion of multibacillary cases and increased incidence of disability in some countries where even marked success in treating leprosy has occurred. (Saunderson, 2008)

Leprosy programmes have been slow to develop areas such as integration, multi-disciplinary research, involvement of people affected with and by leprosy, community-based rehabilitation and community participation. Many of these changes potentially threaten the position of those responsible for leprosy activities; we can be as isolated in our thinking and methods as people affected by leprosy. Research is a good example; leprosy research centres have become progressively isolated, using old technology, with little significant output. (Curtiss & et al., 2001) There is sometimes even criticism when leprosy researchers work in any other area than leprosy. Yet, the reality is that leprosy research is most productive when it is conducted in a multidisciplinary research environment which exchanges ideas, technologies and resources with other research areas. (Rodrigues, 2011)

Prevention of disability is one area that has been innovative, with self-care, community and family involvement, participation of groups of people affected by leprosy, and the use of available, affordable, acceptable appliances such as footwear. For sustainable prevention of disability the ownership of prevention of disability has to pass to people and communities. (Madhavan & et al., 2007) Advocacy must play an increasing role to bring about change. It involves influencing those who are responsible to ensure that leprosy is included in health care and social care, and that people affected are fully included in all aspects of society. Ever since John Snow produced his famous cholera maps of London in 1854, mapping of diseases has been recognized as an essential tool in public health. The latest tool for this purpose is the Geographical Information System (GIS) which besides mapping diseases, manages, analyses and presents data that are linked to geographical locations. Its specific strength is that it can visualise, establish relationships and analyse different features that share the same location. GIS has become an essential tool to be used with care and wisdom to establish the
burden of disease, identify risk factors, and to plan, monitor and evaluate control interventions. (Bakker, Scheelbeek & Van Beers, 2009)

3.16 Leprosy Patient Should Know

3.16.1 At diagnosis

- Leprosy is an infectious disease caused by bacteria not by a curse, witchcraft, or anything else.
- The patient may have infected several other people who may also develop leprosy. They should therefore encourage those people to get checked for leprosy when they develop patches.
- Leprosy bacilli are killed by MDT if the drugs are taken regularly for the recommended period.
- Much of the damage that had been done to nerves and tissues before the patient was started on MDT cannot be reversed.
- During (and after) MDT, patients are no longer infectious and therefore pose no danger at all to other family members or the community.
- In PB patients, patches will still be present when the MDT course is already finished. The patches will disappear slowly over a period of 1 - 3 years.
- Tablets need to be taken daily, as prescribed, and preferably at the same time each day.
- Drugs have to be collected from the clinic every four weeks. On the clinic day the patient takes Rifampicin and Clofazimine under supervision, and collects Dapsone and Clofazimine to be self-administered at home.
- Keep the drugs out of reach of children.

3.16.2 During MDT

A patient on MDT should report immediately to the clinic if one of the following happens:

- If patches start becoming red and swollen again
- If he/she notices sudden weakness of muscles
- If he/she notices that one or both of his/her eyes are red and painful
- If he/she notices pain in one of his/her limbs
- If he/she notices the appearance of red, swollen, tender nodules in the skin
- Additionally the patient should be advised about the following:
To take the drugs after a meal or in the evening just before going to bed if he/she feels any nausea after ingesting them.

To inform the staff at the clinic when they intend to travel. An adequate supply of drugs can then be given to cater for the period they are away.

To inform the staff at the clinic when they intend to move to another area. The clinic staff will then write a transfer letter and give advice on where they should continue treatment.

3.16.3 After MDT

Leprosy reactions can still develop after MDT. These reactions must not be treated with a new course of MDT but can be effectively treated with other drugs. Early reporting is absolutely necessary to prevent irreversible damage.

Patients should report as soon as they notice new patches or if old patches become thick and red. This may indicate that the disease has started again, or that a reaction is taking place.

Patients should report as soon as they notice/feel pain in their hands and feet.

3.17 Wound Prevention

The patient education should be individualized, case by case, and may be advised as follows:

3.17.1 Care for insensitive feet

Wear protective footwear throughout the day to avoid injury.

Avoid too much walking because this is the most common cause of a sole wound in an insensitive foot. So, take a ride on a bicycle when you can; send others in your place; if you must go, stop often, rest your feet and watch where you step.

Learn from any earlier wounds to your feet so that you do not make similar mistakes again.

Avoid heat. Sit with your feet protected, when you sit close to a fire.

Avoid sitting on your lower legs when you sit on the ground because this may cause pressure ulcers.
3.17.2 Daily foot inspection
- Inspect insensitive parts of your feet and legs and also to look for signs of injury, dryness, cracks, and swellings. A small mirror is useful for inspecting the sole of your feet.
- Feel for warm spots: this may warn of injury, and to press for tenderness caused by infection in the deeper layers of the sole of the foot.

3.17.3 Care of dry feet
- Soak for 20 minutes twice daily in salty water, then to rub oil into the skin. This helps to keep the skin of your feet moist and prevents cracks.
- Trim and rub off any callus.

3.17.4 Care of wounds at home
- Remove the cause, e.g. a nail or small stone in a shoe.
- Soak the wound in soapy or salty water for 20 minutes, at least once a day, or more frequently when the wound is discharging. Remove dirt gently.
- Cover the wound with a bandage. This can be made of old clean cloth.
- Rest the foot.

3.17.5 Care for insensitive hands
Generally apply the same kind of care as for the feet. Hands are most frequently damaged during cooking (burns) and manual labour as a result of too much friction.

3.17.6 Care for eyes with lagophthalmos
Patients with lagophthalmos (inability to close the eye) need special attention. Patients should be advised to:
- Wear sunglasses
- Check their eyes daily in the mirror for redness and foreign bodies
- Bind a pad of clean cloth over the eyes at night
- Avoid rubbing the insensitive eye.
3.18 PubMed: A Introduction

PubMed comprises more than 26 million citations for biomedical literature from MEDLINE, life science journals, and online books. Citations may include links to full-text content from PubMed Central and publisher web sites.

3.18.1 A Brief History of NCBI’s Formation and Growth

The establishment of the National Center for Biotechnology Information (NCBI) in November of 1988 occurred primarily through the convergence of three independent but related actions. They were:

- 1984-86—Advocacy groups convened meetings on Capitol Hill to educate legislators and their staffs on the value of supporting genomic research.
- 1986—NLM’s Long Range Plan was completed; it contained a recommendation that a new NLM Division be created to manage and process molecular biology information.
- 1987—The House Select Committee on Aging, Chaired by Senator Claude Pepper, introduced a Bill to establish the NCBI.

In 1984, the Delegation for Basic Biomedical Research began briefing sessions on the Hill, using Nobel winners like Dr. James Watson and Dr. David Baltimore to inform legislators about the importance of genomic research as a new and integral part of the advancement of scientific research. These briefing sessions were thought to be critical in creating an atmosphere in Congress that was receptive to the creation of a biotechnology information center like that of the NCBI.

Also in 1984, Dr. Donald A. B. Lindberg became the director of the NLM, and soon thereafter led the National Library in a major long-range planning effort. Over 100 leaders in the biomedical community participated in this rigorous process, forming 5 panels covering the principal domains of NLM. Of particular note was panel 3—titled “Obtaining Factual Information from Data Bases”—which would prove to be the source from which the idea for NCBI was initially conceived.

The germination of the idea for a center emanated in great part from Dr. Allan Maxam, a professor of biological chemistry at Harvard who was a pioneer of molecular genetics and served as a key member of the 1986 NLM Long Range Planning Panel. He instructed his fellow panel 3 members on the importance they
should assign to the field of biotechnology and informed them of the country’s need to harness the large volume of data that would be generated by the oncoming genetic revolution in science. The Planning Panel, and the NLM Board of Regents, embraced the idea of the need for an organization that could serve as both a repository and distribution center for the growing body of genomic and genetic knowledge and also serve as a unique resource for developing new computer analysis and communication tools for managing molecular biology information.

The newly formed Friends of the NLM saw this as an opportune time to approach the Congress on the need for a biotechnology information center and sought out Senator Claude Pepper, a major champion for medical research. Realizing his congressional colleagues would need to be educated about the benefits of biotechnology research, Senator Pepper asked that NLM develop a document that could be used to explain the need for a center. The resulting document, known as “Talking One Genetic Language: The Need for a National Biotechnology Information Center,” formed the background for the introduction of the initial bill (H.R.5271) to create the NCBI as part of the NLM. The bill was introduced late in the congressional session, and no action was taken on it, but it was reintroduced in the following session by a determined Senator Pepper.

On March 6, 1987, Senator Pepper, as Chairman of the House Committee on Aging, introduced his new bill (H.R.393) to establish NCBI, stating that the center would deal “with nothing less than the mystery of human life and the unfolding scroll of knowledge, seeking to penetrate that mystery, which is life itself.” The hearing had a compelling slate of 15 witnesses, including senior federal and academic health officials as well as five patients who had benefitted from biotechnology.

Although the bill encountered a number of legislative obstacles, Senator Pepper kept the effort alive by securing an appropriation of $3.85 million to begin the biotechnology information program at NLM. During this timeframe, Dr. Daniel Masys, director of the Lister Hill Center for Biomedical Communications, and his branch chief, Dr. Dennis Benson, initiated NLM’s early biotechnology information activities.

The following year Senator Pepper, with the help of Congressman Henry Waxman and Senators Edward Kennedy and Lawton Chiles, incorporated H.R.393 into the NIH reauthorization bill known as the Health Omnibus Extension Act
P.L.100-607. It passed the Congress and was signed into law by President Reagan on November 4, 1988.

Following enactment, Senator Pepper, in ceremonies conducted in the Capitol’s Mike Mansfield Room, said about biotechnology and the new center: “I hope and pray it’s going to realize the dreams that many of us have cherished for a long, long time, by being able to prolong the lives and promote the health and happiness of human beings.”

The act stipulated the following functions for the new National Center for Biotechnology Information:

a. design, develop, implement, and manage automated systems for the collection, storage, retrieval, analysis, and dissemination of knowledge concerning human molecular biology, biochemistry, and genetics;

b. perform research into advanced methods of computer-based information processing capable of representing and analyzing the vast number of biologically important molecules and compounds;

c. enable persons engaged in biotechnology research and medical care to use systems developed under paragraph (1) and methods described in paragraph (2); and

d. coordinate, as much as is practicable, efforts to gather biotechnology information on an international basis.

Beginning with a modest budget of $8 million and a dozen staff members, NCBI began its journey to become a national resource for molecular biology information. Dr. David Lipman, a key developer of the FASTA algorithm, was recruited from NIDDK and was appointed as NCBI Director. Along with the support of his three key appointees—Dr. Dennis Benson, Chief, Information Resources Branch; Dr. David Landsman, Chief, Computational Biology Branch; and Dr. James Ostell, Chief, Information Engineering Branch—Dr. Lipman rapidly grew the center into a major information hub in the molecular biology revolution. NCBI is now a leading source for public biomedical databases, software tools for analyzing molecular and genomic data, and research in computational biology. Today NCBI creates and maintains over 40 integrated databases for the medical and scientific communities as well as the general public. There are over 3 million visitors daily to its website, approximately 27 terabytes of data downloaded per
day, and the number of users as well as downloads increases dramatically each year.

Listed below are some of the major milestones from the many that have occurred over the past quarter of a century:

- **1990—BLAST**—the Basic Local Alignment Search Tool (BLAST) is introduced; optimized for speed, the sequence comparison algorithm quickly finds similar sequences to one’s query.
- **1991—Entrez**—The search and retrieval system for NCBI’s linked databases is introduced in CD form, allowing users to easily find related information from different databases.
- **1992—GenBank at NCBI**—NCBI assumes responsibility for GenBank, a database of nucleotide sequences, and collaborates in its development with international partners at the European Molecular Biology Laboratory (EMBL) and the DNA Data Bank of Japan (DDBJ).
- **1993—Network Entrez**—Network Entrez, a client-server version of the CD-ROM, is introduced, bringing Entrez to the Internet.
- **1994—NCBI Website**—NCBI establishes its own website, mounting initially BLAST, Entrez, dbEST(Expressed Sequence Tags), and dbSTS (Sequence Tagged Sites).
- **1995—Genomes**—This new resource organizes information on genomes, including sequences, maps, chromosomes, assemblies, and annotations.
- **1995—Bankit**—The online tool is introduced to facilitate submissions to GenBank.
- **1996—OMIM**—NCBI mounts the Online Mendelian Inheritance in Man (OMIM), a directory of human genes and genetic disorders.
- **1997—PubMed**—NCBI introduces PubMed, a freely accessible bibliographic retrieval system to the entire MEDLINE database. The new service is launched at a Capitol Hill event by Vice President Al Gore and the ranking Labor/HHS Appropriation Subcommittee members, Senator Tom Harkin (D-IA) and Senator Arlen Specter (R-PA), highlighting its significance.
- **1998—New NIH Disease-Based Services**—Collaborations with NIH Institutes for Disease-Based Services are established such as CGAP, the
Cancer Genome Anatomy Project, to identify the human genes expressed in different cancerous states.

- **1999—Human Genome**—Human Genome Project researchers completely sequence the first human chromosome (#22) and deposit the sequence data at NCBI. A working draft of the entire human genome is completed the following year and made freely accessible from NCBI.

- **1999—Suite of Genomic Resources**—NCBI releases a number of resources to support comprehensive analysis of the human genome, including: **LocusLink**—key descriptors of genetic loci; **RefSeq**—a non-redundant set of human reference sequences; and **dbSNP**—a collection of data on human genetic variation.

- **2000—PubMed Central**—NCBI debuts its free full-text digital archive of biomedical and life sciences journal literature. PubMed Central (PMC) serves as an online counterpart to NLM’s extensive print journal collection and is in keeping with the National Library’s legislative mandate to collect and preserve the world’s biomedical literature.

- **2000—GEO**—The Gene Expression Omnibus database is launched in response to community interest in a public repository for data generated from high-throughput microarray experiments.

- **2001—Bookshelf**—The new Entrez database is introduced to provide free access to books and documents in the life sciences and healthcare fields.

- **2002—WGS**—GenBank begins including Whole Genome Shotgun sequences, which are generated by a semi-automatic technique.

- **2003—DTDs**—NCBI Introduces Document Type Definitions (DTDs) for archiving and exchanging journal content.

- **2003—Entrez Gene**—The Entrez Gene database (formerly known as LocusLink) is developed to supply key connections between maps, sequences, expression profiles, structure, function, homology data, and the scientific literature.

- **2004—PubChem**—The PubChem database is released, providing information on the chemical structure and biological activities of small molecules.

- **2005—NIH Public Access**—NIH develops a Public Access Policy to provide scientists, researchers, and the general public with access to the
published results of NIH-funded research through NCBI’s PubMed Central. NCBI develops a NIH Manuscript Submission System that allows researchers to submit their published papers to PubMed Central.

- **2005—My NCBI**—NCBI introduces the My NCBI tool, which retains user information and database preferences to provide customized services for many NCBI databases.

- **2006—dbGaP**—NCBI launches the database of Genotypes and Phenotypes (dbGaP) to archive and distribute the results of studies that investigate the interaction of genotypes and phenotypes. Studies include Genome-Wide Association Studies (GWAS), medical sequencing, and molecular diagnostic assays, among others.

- **2007—Genome Reference Consortium**—The Consortium of NCBI, EBI, Sanger Institute, and the Genome Institute is created to improve the sequence quality and accuracy of the human reference genome. It takes on the task of improving the reference sequences for other model organisms, including the mouse and zebra fish, often used as models for human disease.

- **2008—Discovery Initiative**—NCBI embarks on a program to help users better explore the myriad of data contained in NCBI’s resources. Automated methods are employed to surface related data that may not be apparent to the user in their original search query but which could lead to serendipitous discoveries.

- **2008—Public Access Becomes Mandatory**—Congress enacts legislation mandating that scientists submit final peer-reviewed journal manuscripts that arise from NIH funding to PubMed Central. The policy requires that the papers be made public on PubMed Central no later than 12 months after publication.

- **2008—1000 Genomes Project**—NCBI archives and distributes data from this international public-private consortium, which aims to build the most detailed map available of human genetic variations. In 2012, NCBI improved the accessibility of these data by collaborating on an effort to make them available on the cloud through Amazon Web Services.
- **2010—dbVar**—NCBI establishes the dbVar archive of large scale genomic variation data and associated defined variants with phenotypic information.

- **2010—My Bibliography**—NCBI introduces the My Bibliography tool to simplify the process for gathering one’s published articles and other materials. The tool, which is connected to NIH’s grants management system, also assists researchers in complying with the NIH Public Access Policy.

- **2011—PubMed Health**—The service is introduced to provide information for consumers and clinicians on prevention and treatment of diseases and conditions, with an emphasis on reviews of clinical effectiveness research.

- **2012—Genetic Testing Registry (GTR)**—NCBI, in collaboration with NIH, launches the GTR to address the need for information about genetic tests for healthcare providers, researchers, patients, and others. The database provides information about the availability, validity, and usefulness of genetic tests.

- **2013—Clinvar**—NCBI creates the ClinVar database to aggregate information about sequence variation and its relationship to human health. The database includes submissions from outside research and testing groups as well as internal data drawn from such sources as dbSNP, dbVar, dbGap, and Gene Reviews.

- **2013—PubReader**—NCBI develops a new presentation style that optimizes reading of PMC articles through a browser on a desktop, laptop, or tablet computer.

  An in depth account of the history of NCBI and NLM can be found in a published version of the Joseph Lieter NLM/MLA lecture presented at the annual meeting of the Medical Library Association in 2007 (1).

### 3.19 About PubMed

PubMed is a free resource developed and maintained by the National Center for Biotechnology Information (NCBI), a division of the U.S. National Library of Medicine (NLM), at the National Institutes of Health (NIH).

PubMed comprises over 22 million citations and abstracts for biomedical literature indexed in NLM’s MEDLINE database, as well as from other life science
journals and online books. PubMed citations and abstracts include the fields of biomedicine and health, and cover portions of the life sciences, behavioral sciences, chemical sciences, and bioengineering. PubMed also provides access to additional relevant websites and links to other NCBI resources, including its various molecular biology databases.

PubMed uses NCBI's Entrez search and retrieval system. PubMed does not include the full text of the journal article; however, the abstract display of PubMed citations may provide links to the full text from other sources, such as directly from a publisher’s website or PubMed Central (PMC).

### 3.19.1 History of PubMed

PubMed was first released in January 1996 as an experimental database under the Entrez retrieval system with full access to MEDLINE. The word "experimental" was dropped from the website in April 1997, and on June 26, 1997, free MEDLINE access via PubMed was announced at a Capitol Hill press conference. Use of PubMed has grown exponentially since its introduction: PubMed searches numbered approximately 2 million for the month of June 1997, while current usage typically exceeds 3.5 million searches per day.

PubMed was significantly redesigned in 2000 to integrate new features such as LinkOut, Limits, History, and Clipboard. PubMed began linking to PubMed Central full-text articles and the Bookshelf's initial book, Molecular Biology of the Cell. The Entrez Programming Utilities, E-Utilities, and the Cubby (My NCBI subsequently replaced the Cubby) also were released.

In 2002, the PubMed database programming was completely redesigned to work directly from XML files, and two new NCBI databases, Journals (now the NLM Catalog) and MeSH, were created to provide additional search capabilities for PubMed.

### 3.19.2 Advantages of Searching with PubMed

PubMed is the interface used most often to search MEDLINE at Mount Sinai. In addition to being free, PubMed offers several advantages over other versions of MEDLINE (ex. OvidSP, ISI Web of Knowledge):

- Automatic Term Mapping: PubMed automatically includes synonyms and Medical Subject Headings in your search. As a result, the retrieval from a
search in PubMed is often more comprehensive than the retrieval from the same search in other versions of MEDLINE.

- Quicker access to newly published articles: PubMed obtains data before commercial versions of MEDLINE.
- A fast, intuitive search interface: PubMed is designed to facilitate efficient information retrieval by end users: scientists and clinicians.
- Note: Although MEDLINE/PubMed links to the full-text of many articles, it does NOT contain or search the full-text of journal articles.

### 3.20 Conclusion

Literature search refers to the process in which people use tools to search for literature relevant to their individual needs. In the context of this review, tools are Web-based online systems; literature is limited to the biomedical domain; and typical user information needs include, but are not limited to, finding the bibliographic information about a specific article, or searching for publications pertinent to a specific topic (e.g. a disease). With the ease of Internet access, the amount of biomedical literature in electronic format is on the rise. As of 2010, there are over 20-million citations indexed through PubMed, a free Web literature search service developed and maintained by the National Center for Biotechnology Information (NCBI). PubMed is as part of NCBI’s Entrez retrieval system that provides access to a diverse set of 38 databases. PubMed currently includes citations and abstracts from over 5000 life science journals for biomedical articles back to 1948. Since its inception, PubMed has served as the primary tool for electronically searching and retrieving biomedical literature. Millions of queries are issued each day by users around the globe, who rely on such access to keep abreast of the state of the art and make discoveries in their own fields.

Although PubMed provides a broad, up-to-date and efficient search interface, it has become more and more challenging for its users to quickly identify information relevant to their individual needs, owing mainly to the ever-growing biomedical literature.

The data collected from PubMed on Leprosy during 2003-2012 is been analysed by using various bibliometric tools and presented in the Chapter IV.
Reference


http://library.mssm.edu/tutorials/advantages.html


http://vussc.info/communicable-diseases


