CHAPTER-III

Lymphatic Filariasis:
Disease and Treatment
The Disease

Disease is primarily a biological problem, which reflects its multidimensional tribulations in the biological as well as socio-psychological life of human being. Human being is a social animal whose health conditions influence his socio-economic and bio-psychological life. Health is always treated as wealth. The World Health Organization (WHO) defines health as a state of complete physical, social and mental well-being and not merely the absence of disease or infirmity. In recent years, this statement has been amplified to include the ability to lead a "socially and economically productive life" (Park & Park, 1998). A good health makes a person behave in normal ways, work in normal ways and keep him free of any infection or disease (Manelkar, 1999). Therefore, good health not only determines a person's socio-economic and bio-psychological identity but also an identity as a social entity. Health regulates not only human biology but also determines socio-psychological harmony of society. Though, an individual is an integral part of social structure and system, the output of the society depends upon the output of its members. The functional output and socio-economic performance of the individuals and society have been influenced by the health status of the individuals and the community. The "individual's productivity" influences the "mass productivity" of society. Thus a healthy person is an asset for the society and a diseased person is a burden to the family as well as to the society. On the other hand in some diseases due to social stigma, patients maintain segregated life instead of an integrated life. This type of segregation creates distortions in the integrated social system of the society. There are some diseases like paralysis, leprosy, mental disorder, blindness, downs syndrome and lymphatic filariasis (LF), which cause temporary or permanent disability among the human beings. Due to disability manifestation, these diseases generate problems not only for the patients but also for their family members and the society as a whole. Filariasis is one such disease, and is an important public health and socio-economic problem in tropical countries. The disability manifestation of filariasis creates "occupational inertness" instead of an "occupational alertness". Beyond that permanent disability many filariasis-infected people are facing temporary disability due to filarial fever. This
manifestation of the disease also creates not only work loss but also economic loss to their family. So the complex disease manifestation of filariasis creates a vicious circle of poverty-disease-poverty among the people, because mostly the poor people are victims of this disease. Conditions related to poverty facilitate the infection and progression of disease. On the other hand the disease condition accelerate the process of poverty. Hence, poverty vis-a-vis filariasis must be understood in a holistic manner.

LF is an important public health and socio-economic problem. More than 120 million individuals worldwide are estimated to suffer from LF caused infection with mosquito borne filariasis nematode Wuchereria bancrofti (WHO, 1994). Filariasis is a global problem. It is prevalent in 73 countries worldwide with over 120 million people infected (WHO, 1996). LF is endemic in 80 countries situated in the tropical and subtropical belt; an estimated 70 per cent of infected cases are concentrated in India, Nigeria, Bangladesh and Indonesia (WHO, 2001). It is estimated that 751 million people live in endemic areas. 91 per cent of the 128 million cases of LF are caused by Wuchereria bancrofti. While Brugia malayi and Brugia timori infection account for the other 9 per cent. Wuchereria bancrofti has the widest distribution and occurs in Africa, the Caribbean, Latin America and many islands of Western and South Specific Ocean. It is responsible for loss of 5 million disability adjusted life years (DALYs) per year. Among 120 million people estimated to be infected with LF in the world, 48 million live in India alone. In India, about 412 million people are at risk of filarial infection (Sharma et al., 1995) and about 45 million people are estimated to be infected with the most wide spread species Wuchereria bancrofti (Ottensen & Ramachandran, 1995). The problem of LF is increasing every year due to gross mismanagement of the environment. Due to poor knowledge about the disease, the disease is spreading day-by-day. The population exposed to the risk of infection was 25 million in 1953, 64 million in 1958, 136 million in 1968, 236 million in 1976 and 304 million in 1981. The disease is endemic all over India except in Jammu and Kashmir, Himachal Pradesh, Punjab, Haryana, Delhi, Chandigarh, Rajasthan,

\textsuperscript{2} Wuchereria bancrofti is the parasite of the disease lymphatic filariasis.
Nagaland, Manipur, Tripura, Meghalaya, Sikkim, Arunachal Pradesh, Mizoram and Dadra and Nagar Haveli. However, recent studies carried out during the past two decades indicate that areas previously known to be free from filariasis show evidence of low degrees of transmission. The most infected areas are found in Uttar Pradesh, Bihar, Andhra Pradesh, Orissa, Tamil Nadu, Kerala and Gujurat (Park & Park, 1998). It is estimated that 25.05 million individuals are exposed to the risk of filariasis out of which 6.4 per cent belong to Orissa. In Orissa, the rural population is more exposed to the risk of filariasis (22.2 million) than that of urban population (2.85 million) (WHO, 2001).

Bancroftian filariasis is prevalent in both urban and rural areas. Although, mortality is uncommon, morbidity associated with this infection is considerable and lifelong. Since mortality is uncommon, LF escapes the attention of planners, governments and the media. All rural and many urban areas in India lack anti-filarial measures and only 11 per cent of the endemic population is protected by the National Filaria Control Programme3 (NFCP, 1995).

Burden of Disease

The burden of diseases has been estimated by the measure “Disability Adjusted Life Years” (DALYs) by World Bank Development Report (World Bank, 1993). In 1990, communicable diseases caused 59 per cent of burden due to death and disability among the world's poorest 20 per cent population. Among the world's richest 20 per cent on the other hand, non-communicable diseases caused 85 per cent of burden due to death and disability. A raised baseline rate of communicable disease decline between 1990 and 2020 would increase life-expectancy among the world's poorest 20 per cent around ten times as much as it would the richest 20 per cent (4.1 Vs. 0.4 years). However, the poorest 20 per cent would gain only around a quarter to a third as much as the richest 20 per cent from a similar increase in non-communicable diseases (1.4 Vs. 5.3 years). As a result, a further decline in communicable diseases would decrease the poor-rich

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3 National Filaria Control Programme (NFCP) is a lymphatic filariasis control programme launched by the Government of India.
gap in 2020, but an accelerated rate of overall decline in non-communicable
diseases, the poor-rich gap would widen (Gwatkin et al., 1999).

The global burden of the filariasis is only 0.23 per cent of the total burden of
parasitic and infectious diseases. It has been recognized that social and economic
burden caused by LF is poorly understood and remain un-quantified (Evans et al.,
1993). It has been estimated that on average 23.4 man-days are lost due to
Bancroftian filariasis and an overall 162.12 million man-days are lost per year in
India due to acute filarial attacks (Sabesan et al., 1992). If the cost of treatment
including hospital and surgical procedures were also to be included, the actual
loss would have been manifold. Acute disease is likely to be many times more
prevalent than chronic (Pani et al., 1995). The adverse effects caused by the
disease are not well documented, because those have serious socio-economic
impact on the society by significantly lowering the productivity and economic
loss resulting from its chronicity (Ramaiah et al., 1997; Ramu et al., 1996;
Gyapong et al., 1996, Evans et al., 1993). There is inadequate understanding of
the disability, social stigma and psychological affects resulting from chronic
diseases but more severely affected people are probably socially restricted as well
as physically burdened. The number of individuals worldwide with overt physical
disabilities resulting from Bancroftian filariasis is about 40 million (WHO, 1994).
Indirect economic losses result from social and physical confinements of people
with chronic conditions. The costs incurred by individuals as well as by the health
system are also substantial. The World Health Organization (WHO, 1994) also
identified the need to asses the spectrum of social and economic affects of LF.
Due to paucity of information on the economic burden of the disease steps have
been taken to estimate the economic costs of the disease in different parts of the
world (Ramaiah, 2000).

History of Filariasis

The preponderance of evidence indicates that LF existed in the ancient tropical
world. One can find description about elephantiasis in the works of many ancient
Greek and Roman authors including Celsus, Galen, Aretaens, Caelius, Aurelianul
Pliny and Plutarch. Many of them have described this disease as leprosy, which came to be unknown as *elephantiasis graecorum*, to distinguish it from another form of elephantiasis with a different appearance, *elephantiasis arabum*, probably Bancroftian filariasis (Savitt, 1993). Ancient descriptions of a medical condition resembling Bancroftian filariasis exist not only in the writings of Greco-Roman scholars but also in the writings of scholars from India, the Nile Delta and Polynesian Islands such as Fiji and Society Islands (Adams, 1844, 1846, 1847; Bhishegranthaed, 1911; Castellani, 1919; Hoepli, 1959; Foster, 1965, Laurence, 1967, Sasa, 1976) (quoted by Savitt, 1993). The historian B.R. Laurence argues that Bancroftian filariasis actually originated in South-East Asia and spreaded with the migration of people to the South Pacific Islands (especially Polynesia) and to Africa (Laurence, 1968, 1977) (quoted by Savitt, 1993). Other argue that filariasis came to the new world, most likely as a result of African slave trade, conducted by White Europeans, brought concentrations of infected black Africans to slave depots in West Indian Islands like Barbados, where they were sold and redistributed to other West Indian Islands or to the North and South American main-lands (Savitt, 1993). A legacy of this black African slave trade in the United States was the establishment of a focus of Bancroftian filariasis at Charleston, South Carolina and the surrounding "Low Country", which survived until the early twentieth century (Savitt 1977; Chernin, 1987; Reynolds and Sy, 1989; quoted by Savitt, 1993).

Western acquaintance with LF began with the advent of colonialism in the eighteenth and nineteenth centuries and the exposure of western physicians to exotic tropical diseases in the colonies. One of the earliest western descriptions of filariasis is by Jean-Nicolos Demarquary, a French physician working in the Somma district. He came across with a patient originally from Havana, Cuba and reported the matter in the Gazette Medical de paris, that an operation has been performed on the young man to get rid of a tumor occupying the left scrotal sac (which he refers to as “Galctocele of vital”) (Demarquary, 1863, quoted by Rajan, 2000). He writes that on tapping the scrotal sac, he obtained a milky white fluid and that he made a microscopic study of that still warm fluid. His sketches leave little doubt that he saw microfilariae in the fluid. In 1868 Wucherer described a
species of worm in the urine of patients with tropical haemoturia in Brazil and clearly distinguished them from *Billarzia (Schistosoma)* hematobium. Lewis, a Welsh physician stationed in India, appears to have performed some remarkable studies on LF, and it is an egregious oversight that no major filarial parasite is eponymously named after him (Manson, 1899, quoted by Rajan, 2000). In 1870, he noted that the same worms were present in the blood of some patients. Lewis made accurate inferences on the nature of the disease, the relationship between microfilariae and adult worms and the possible mechanism of lymphoedema in affected individuals. In some of his works, he refers to his new discoveries on “filariae” based on an unintentional follow up of a patient two and a half years after initial contact and convinced that the infection is chronic in nature. With great accuracy he correlated the presence of these “filariae” with lymphatic obstruction and chyluria. It is clear that his work had an impact on other workers in the field, since both Manson and Bancroft refer to his work in their description of LF (Rajan, 2000).

In India filariasis is known since 600 B.C. The Indian Physician Susurta⁴ in his valued book *Sushruta Samhita⁵* referred to a disease called *Slipada* (*Si*-elephant, *Pada*-leg). The description of this condition appears to include diseases other than LF; it is nonetheless clear that at least some of patients exhibiting *Slipada* are cases of LF (Rajan, 2000). The clinical manifestations of filariasis was depicted in the stone carving of the famous sun temple “Konark” built in 200 A.D. in Orissa, which is located at distance of 50kms. from the present study area. The stone carving of elephantoid leg indicates that the disease existed in this area since 200 A.D. In the medieval Oriya literature poet Dinakrushna Das has mentioned about filariasis in his writings. So, it appears that the presence of the disease filariasis in Orissa is very old.

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⁴ Susurta was the ancient ayurvedic physician of India.
⁵ *Sushruta Samhita* was an ayurvedic text book written by Susruta.
Biology of the Disease

Perhaps the most ugly disease of the human kind is the clinical entity known as elephantiasis. The horribly swollen parts of the body afflicted with this condition have been known since antiquity. The ancient Greek and Roman writers linked the thick ended and fissured skin of infected persons to that of the elephant, although they also confused leprosy with this condition. Majority of filariasis is caused by *Wuchereria bancrofti* followed by two other species namely *Brugia malayi* and *Brugia timori*. Bancroftian filariasis (*Wuchereria bancrofti*) is the most wide spread of the filariasis of humans, extending throughout central Africa, the Nile Delta, Turkey, India and South East Asia.

*Morphology*

Adult worms are long and slender with a smooth cuticle and blunt rounded ends. The head is slightly swollen and bears two circles of well-defined papillae. The mouth is small; a buccal capsule is lacking. The male is about 40mm long and 100μm wide. The vulva is near the level of the middle of the esophagus.

Adult *Wuchereria* live in the major lymphatic ducts of humans, tightly coiled into nodular masses. They are normally found in the afferent lymph channels near the major lymph glands in the lower half of the body. Rarely, they invade a vein. The females are viviparous, producing thousands of Juveniles known as microfilariae. Microfilariae are not as differentiated as a normal first-stage juveniles and are sometimes considered advanced embryos. The microfilariae of *Wuchereria bancrofti* retain the egg membrane as a sheath. The sheath is rather delicate and close fitting but can be detected where it projects at the anterior and posterior ends of microfilariae. When stained, several internal nuclei and primordial of organs can be seen in the microfilariae. The local of these and the presence or absence of a sheath is used to identify the several species of microfilariae found in humans.

The microfilariae are released into the surrounding lymph by the females. Some may wonder into the adjacent tissues, but most are swept into the blood through
the thoracic duct. Throughout much of the geographical distribution of this
disease, there is a marked periodicity of microfilariae in the peripheral blood;
that is, they can be demonstrated at certain times of the day, whereas at other
times they virtually disappear from the peripheral circulation. The maximum
number usually can be found in nighttime. For this region, night-feeding
mosquitoes are the primary vectors of the *Wuchereria* in areas where microfilarial
periodicity occurs. During the day, the microfilariae are concentrated in blood
vessels of the deep tissues of the body, predominantly in the pulmonary vessels
proximal to the pulmonary arterioles (Spencer, 1973). Causes of the periodicity
remain obscure, but they apparently do not involve daily release of a new
generation of progeny by the adult female. Stimuli, such as arterial oxygen
tension and body temperature, probably are involved. Administration of pure
oxygen to a patient during peak microfilariamia can cause the microfilariae to
localize in the deep tissues. Reversal of the patient's sleep schedule causes
reversal of periodicity, so microfilaraemia can cause the microfilariae to localize
in the deep tissues that microfilaraemia becomes diurnal. The adaptive value of
periodicity is difficult to explain. Although, it is clearly advantageous for the
microfilariae to be present in the peripheral blood when the vector is likely to be
feeding, however, it is not understood is there any value in being absent when
the vector is not feeding. Some strains of *Wuchereria* have a normal diurnal
periodicity. The periodicity is unimportant clinically, but it has significant
diagnostic and epidemiological implications.

In certain areas, a strain of *Wuchereria* is common that shows a diurnal
periodicity, referred to as sub-periodic. The morphology of the adults is identical
to that *Wuchereria* producing periodic microfilariae; most investigators believe
that only one species is involved although some designate the sub-periodic type
as a separate species named *W. pacifica*. Daytime feeding mosquitoes are the
major vectors of the sub-periodic strain.

Microfilariae are ingested by the mosquito along with its blood meal. They lose
their sheath in the first 2 to 6 hours in the insect's mouth, after which they
penetrate the gut of the host and reach the thoracic muscles. The first cuticular
molt occurs about 2 days later. The second stage juvenile is a short, sausage-shaped worm (Sausage stage) in which most of the organ systems are present. Within 2 weeks the second molt takes place. The juvenile is now an elongate, slender filari form third stage and development ceases. The filari form juveniles are 1.4 to 2mm long and are infective to the definitive host. They migrate throughout the hemocoes, eventually reaching the labium, or proboscis sheath, from which they escape when the mosquito is feeding. They enter the skin through the wound made by the mosquito. After migrating through the peripheral lymphatics, the worms settle in the larger lymph vessels, where they mature (Schmidt & Roberts, 1981).

**Pathology and Symptomatology of Filariasis**

Filarial symptoms are caused by the adult worms, living as well as dead and degenerating. Microfilariae, apparently cause slight or no pathology, although they have been tentatively associated with tropical pulmonary eosinophilia granulomatos of the spleen, and allergic reactions following their destruction by drugs. The adult worms lie in the dilated lymphatics or in the sinuses of the lymph nodes. The pseudotubercular granulomatous reaction around the trapped worms becomes pronounced on their death. It occludes the small lymphatics, narrows the larger ones, and ultimately walls off the necrotic tissues surrounding the degenerating worms. The early cellular reactions and oedema give way to vascular and lymphatic hyperplasia and fibroblastic proliferation. Finally, there is absorption and replacement of the parasite by hyalinized, or even calcified, scar tissue. The lymphatics became varicose, collateral branches open up, and lakes of lymph develop in the sinuses of the lymph nodes. The living and dead worms and microfilariae evoke an infiltration of eosinophilic leukocytes in the inflamed tissues. Because Bancroftian filariasis may run its course over many years, it varies greatly in its clinical manifestations. It is possible to classify broadly the results of filarial infection into the asymptomatic, inflammatory and obstructive types (Nutman, 2000).
Asymptomatic Filariasis

In endemic areas children are exposed to infection at an early age, and by the age of 6 years they exhibit microfilariae in their blood without experiencing symptoms referable to their infection. On physical examination the patient may exhibit a moderate generalized enlargement of lymph nodes, especially of the inguinal lymph region. Blood examination discloses numerous microfilariae and a low-grade eosinophilia of the adult worms die. The microfilariae disappear without the patient's being aware of the infection.

Inflammatory Filariasis

The inflammatory filariasis infection is an allergic phenomenon due to sensitivity to the products of the living and dead adults worms. Superimposed streptococcal and fungal infections may be involved. Recurrent attacks are characterized by funiculitis, epididymitis, orchitis, retrograde lymphangitis of the extremities, and localized areas of swelling and redness of the arms and legs. Fever, chills, headache, vomiting and malaise may accompany these attacks, which last from several days to several weeks. The lymphatic of the legs and genitalia are chiefly affected. In males acute lymphangitis of the spermatic cord (funiculitis) with tender, thickened cord, epididymitis, orchitis, and scrotal oedema are common. Similar acute attacks may occur at monthly or longer intervals in patients with or without elephantiasis. Usually the affected extremity becomes red, hot, and very painful. Therapy with antimicrobial drugs is usually unsuccessful, suggesting a verminous rather than bacterial etiology. Abscesses of the pelvis of the kidney, epididymis, retroperitoneal tissues, inguinal nodes, and iliopsoas muscles may result from the dead and degenerating worms. These abscesses may be sterile, but frequently pyogenic bacteria are present. The acute granulomatous reaction in the lymphatics due to the worms and their toxic products, which is manifest by local inflammation and systemic allergic symptoms, gradually merges into a chronic proliferative overgrowth of fibrous tissue around the dead worms that produces lymphatic obstruction, recurrent attacks of lymphangitis, and eventually the elephantiasis.
Obstructive Filariasis

Elephantiasis through lymphoedema is the dramatic end result of filariasis. Many mistakenly believe that it is the inevitable termination of every filarial infection, but fortunately, the grossly enlarged scrotum, breast, or leg occur in a few individuals. Elephantiasis has been reported in 1 to 70 per cent of infected natives in various parts of the world, many of who are exposed to infective mosquitoes from birth. Obstructive filariasis develops slowly, usually follows years of continuous filarial infection, and is preceded by chronic oedema and often by repeated acute inflammatory attacks. In the chronic stage the cellular reaction and oedema are replaced by fibroblastic hyperplasia. There is absorption and replacement of the parasite by proliferative granulation tissue, and extensive lymph vessels are produced. The high protein content of the lymph stimulates the growth of dermal and collagenous connective tissue and gradually over a period of years, the enlarged affected parts harden, producing chronic elephantiasis. The site of the obstructive inflammation determines the parts of the body affected. Obstruction of the thoracic duct or the median abdominal lymph vessels may affect the scrotum and penis of the male and external genitalia of the female, while infection of the inguinal glands may involve the extremities and external genitalia. Elephantiasis is uncommon in persons under 30 years of age. There is little correlation between the presence of microfilariae in the blood and elephantiasis, since microfilariae disappear after the death of the worms. Rupture of the lymphatics of the urinary bladder or kidney may produce chyluria, those of the tunica vaginalis, hydrocele or chylocele, and those of the peritoneum chylous ascites. The most common features are hydrocele and lymphangitis of the genitalia and recurrent attacks of lymphangitis with fever and pain. Recurrent lymphangitis and even elephantiasis may be accentuated or in some cases even produced by superimposed streptococcal infection (Brown, 1975).

Clinical Manifestations of Lymphatic Filariasis

The disease passes through different stages showing different clinical conditions.
**Microfilaraemic Stage**

In *Wuchereria bancrofti* endemic areas, the overwhelming majority of infected individuals have few overt clinical manifestations of filariasis despite the presence of large numbers of circulating microfilariae in the peripheral blood of the body. The prevalence of microfilaraemia is higher among men when compared to women (Brabin, 1990). It is also found that the prevalence increases with age and usually reaches a plateau between 20-30 years of age. Usually these microfilaraemic people are clinically asymptomatic. It is clear from the earlier studies that "asymptomatic microfilaraemic stage" is not as benign as initially believed although the majority of individuals appear to remain asymptomatic for years with relatively few progressing to either the acute or chronic stages (Kumaraswami, 2000).

**Acute Manifestation of Lymphatic Filariasis**

The acute clinical manifestations of filariasis are characterized by recurrent attacks of fever associated with inflammation of the lymph nodes (adenitis) and/or lymph vessels (lymphangitis) termed adenolymphangitis (ADL) (Kumaraswami, 2000). Acute disease is essentially an inflammatory condition involving lymphatics and lymph nodes. It is best described as acute ADL, though the affected local sites could be different. The clinical signs include pain, tenderness, redness, local swelling, local warmth (sometimes local abscess) and other commonly associated findings include fever, oedema, constitutional complaints and localized or ulcerated abscesses.

The most common presentation is that of a cord-like structure associated with retrograde lymphangitis in the lower or upper limbs. In the scrotal area or the breast it may present as a painful palpable nodule. Funiculo-epididymoorchitis is the usual presenting feature of acute attacks of lymphangitis involving the male genitalia. The patient is in pain and, on examination; the scrotum is painful and red with testicular tenderness. The systematic reactions are mild and distal oedema is rare. Recurrence of these attacks at the same site is common (Kumaraswami, 2000).
Chronic Manifestation of Lymphatic Filariasis

In bancroftian filariasis, the occurrences of the major signs of chronic disease are hydrocele, lymphoedema and elephantiasis. The most common are hydrocele and swelling of the testis, followed by oedema of the entire lower limb, the scrotum, the entire arm, the vulva and the breast in descending order of frequency (Pani et al., 1990; Pani et al., 1995).

Lymphoedema/Elephantiasis

In the LF endemic area, lymphoedema of the leg occurs more frequently in women than in men (Lammie et al., 1993, Barry et al., 1971; Gyapong et al., 1994; WHO, 1994). The differential diagnosis of oedema of the leg includes not only lymphoedema, but also chronic venous insufficiency, deep vein thrombophlebitis, lipoedema, tumour, and a variety of other conditions (Olszewski et al., 1993; Pflug, 1976; Ruschhaupt and Graor, 1985; Ter et al., 1993). Lymphoscintigraphy may sometimes be useful in distinguishing lymphoedema from other causes of leg oedema (Ter et al., 1993). There is no clinical or laboratory marker currently exists that reliably confirms the filarial aetiology of lymphoedema secondary to individual patient (Addiss & Dreyer, 2000).

Acute attacks influence the classification of lymphoedema. Episodes of ADL may also result in lymphoedema, but this is usually transient. The degree of obstruction caused by the inflammatory response at the site of the dead adult worm and the extent of collateral lymphatic circulation determine the magnitude and duration of lymphoedema (Addiss & Dreyer, 2000).

In case of filariasis, swelling of the limbs is most common feature. These lymphoedema or swelling of the limbs have been graded by World Health Organization (1992).

Grade 1: Pitting oedema of the limb that is reversible on elevating the limb.

Grade 2: Pitting/Non-pitting oedema that is not reversible on the limb. Skin is normal.
Grade 3: Non-pitting oedema of the limb with non-reversible on elevation. 
Skin is thickened.

Grade 4: Non-pitting oedema with fibrotic and verrucous skin changes 
(elephantiasis).

**Hydrocele**

Hydrocele is the commonest clinical manifestation in many endemic communities 
with 40-50 per cent of the males affected. When the parasites are lodged in the 
testicular lymphatics, it results in accumulation of fluid in the tunica vaginalis 
testis causing hydrocele. In some case of hydrocele, the patient may not 
experience the ADL attacks and therefore, the progression seems to be a passive 
phenomenon. Clinically speaking repeated episodes of adenolymphangitis 
involving the testicular lymphatics (epididymorchitis) may precede the 
development of hydrocele. World Health Organization (1992) has recommended 
different grading for hydrocele for field implication.

- Grade 1: Smaller than tennis ball.
- Grade 2: In between grades I and III.
- Grade 3: Bigger than the patient’s head.

**Other Manifestations of Filariasis**

There are many other clinical manifestations being attributed to filariasis. These 
include tropical pulmonary eosinophilia (TPE), chronic funiculitis, chyluria, 
lymph varix, nephritis, chylocele, chylous ascites, chylous diarrhoea, 
endomyocardial fibrosis, pericardial effusion, mono-arthritis (knee), etc. Except 
TPE, chronic funiculitis and chyluria, others are seen rarely. Some individuals 
with adult worms produce allergic reaction against microfilariae and hence 
contain no microfilariae. This condition is called TPE. The symptoms of TPE 
includes paroxysmal nocturnal cough with low eosinophilia count and impaired 
lung function. Chyluria is a condition where patients complain of passing 
milking white urine due to admixture of chyle in urine. Some patients complain
of associated frank hematuria also. The condition is usually painless and may be intermittent. The condition is believed to result from blockage of the retroperitoneal lymph nodes below the cisterna chilii with consequent reflux and flow of intestinal lymph directly into renal lymphatics. Microfilaria may be detected in urine or blood in few cases.

**Treatment and Control of the Disease**

Generally, the goals of treatment for LF are to prevent, reverse or halt progression of disease and to break the transmission of the parasite. For almost half a century, the main drug of choice in the treatment of filariasis is diethylcarbamazine (DEC). Other drugs or drug combinations may be more effective for interrupting transmission at the community level (Addiss & Dreyer, 2000).

**Tools for Diagnosis of the Disease**

Infection of filariasis can be confirmed by detecting microfilaria or filarial antigen in peripheral blood or by detecting the characteristic movement of the adult worm known as the "filarial dance sign" on ultrasound examination. The later method is not available widely for patient use, as it needs costly instruments.

The most commonly practiced method is detection of microfilariae in peripheral blood of the body (Eberhard and Lammie, 1991). For epidemiologic screening 20-60 µl of capillary ("fingers prick") blood can be dried on a slide, stained with Giemsa, and examined under the microscope for the presence of microfilariae.

**Treatment of Lymphatic Filariasis**

The pathogenesis of LF has been changed in due course of the time. This changing knowledge is a pivotal implication for the treatment. There are many factors, which are operating the pathogenesis of LF, but four factors are most important. Those are: the living adult worm, inflammatory responses caused by death of the adult worm, secondary bacterial infections and microfilariae. It is now clear that the living adult worms cause extensive lymphangiectasia, even in
asymptomatic persons. Because lymphatic vessels dilatation is diffuse and not restricted to the site of the living adult worm, it is hypothesized that worms release diffusible substances that directly or indirectly cause lymphangiectasia (Dreyer & Piessens, 1999). Again, dilatation of the lymphatic vessels leads to dysfunction of lymphatic and the chronic clinical manifestations of LF like lymphoedema and hydrocele. Death of the adult worm causes an acute inflammatory response that is manifested clinically an adenitis and lymphangitis. On the other hand secondary bacterial infections cause an acute syndrome of dermatolymphangioadentitis (Olszewski, 1996; Olszewski et al., 1993).

The treatment for three major clinical forms of LF is discussed below.

Asymptomatic or Sub-Clinical Lymphatic Filariasis

Early treatment of asymptomatic persons is necessary to prevent further lymphatic damage. DEC, which has macrofilaricidal and microfilaricidal properties, is the drug of choice. Because the macrofilaricidal action of DEC does not seem to reverse existing lymphatic damage, the goal of DEC treatment would be to prevent further adult worm associated lymphatic damage and dysfunction. The microfilaricidal activity of DEC clears the blood of the parasite, and reverse filaria associated haematuria and proteinuria.

Acute Manifestations of Lymphatic Filariasis

The acute manifestations of the LF are adenitis and lymphangitis, which results in considerable pain, suffering and decreased ability to perform work (Sabesan et al., 1992; Gyapong et al., 1996). The aetiology of "acute attacks" has been a debatable subject (Dreyer and Piessens, 1999). The ADL attacks attributed to bacterial infections, immunologic resources to a variety of filarial antigens and release of substances from, or death of, the adult worm (Moore et al., 1996; Montestruc et al., 1960). Recent clinical observations reveal that much of the confusion has resulted from combining two distinct entities: "true" filarial adenolymphangitis (AFL) and acute dermatolymphangioadentitis (ADLA) (Dreyer and Piessens, 1999; Dreyer and Noroes, 1997).
Acute Filarial Adenolymphangitis (AFL)

During the period of acute episode, anti-filarial treatment is not recommended because it may provoke additional adult worm death and exacerbate the inflammatory response. During the acute episode, supportive treatment is recommended, including rest, postural drainage, particularly if the lower limb is affected; cold compresses at the site of inflammation; and antipyretics and analgesics for symptomatic relief. After the resolved of acute attack if the patient remains microfilaria or antigen positive, DEC may be given to kill the remaining adult worms. If these tests are not available, the patient can be treated with a single 6mg/kg dose of DEC and monitored for local adverse reactions that would indicate death of remaining adult worms.

Chronic Manifestations of Lymphatic Filariasis

The chronic manifestations of LF include lymphoedema, adenopathy, and urogenital disease, but lymphoedema is most common in filariasis endemic regions.

Lymphoedema

Lymphoedema occurs most frequently in the lower extremities, but it also can involve the arms, breast, scrotal wall, penis and rarely the vulva. The principle of the treatment of lymphoedema of the leg is same for lymphoedema in other sites.

Data from different countries indicate that elephantiasis and lymphoedema of the leg are reversible with a treatment regimen that emphasizes hygiene, prevention of secondary bacterial infections and physiotherapy (Shenoy et al., 1995). This regimen is similar to that now recommended for treatment of lymphoedema in Europe, Australia, and the United States (Foldi et al., 1989; Boris et al., 1994), where it is known by a variety of names including complex decongestive physiotherapy and complex lymphoedema therapy.

Patient education is most important in lymphoedema treatment, both to alter fatalistic beliefs about the inevitable progression of the disease and to foster motivation. In the disease endemic areas where hygienic conditions are often
The most important component in preventing acute bacterial attacks is meticulous attention to hygiene and skin care and aggressive treatment of skin lesions, especially candidiasis. To reduce and manage the lymphoedema different methods are useful. Exercise, elevation of the limb, compressive bandaging and gentle massage are helpful in this direction. During acute attack only elevation is advised and other measure are not recommended.

Various surgical procedures for lymphoedema have been attempted since early in the 20th century (Matas, 1913; Watson, 1953; Miller, 1977; Jamal, 1981). But the results of these have generally been unsatisfactory (Foldi et al., 1989; Mortimer, 1990). According to Addiss and Dreyer (2000) the role of surgery in the treatment of lymphoedema of the limbs, particularly that associated with LF, is limited. In case of advanced elephantiasis, plastic surgery may be warranted after leg volume has been reduced and the health of the dermal and subcutaneous tissue has been restored (Foldi et al., 1989).

**Hydrocele**

Hydrocele is the most common manifestation of bancroftian filariasis in male population. When the bancroftian parasites are lodged in the lymphatics of testis, it accumulates fluid in the tunica vaginalis testis and causes hydrocele. A majority of these patients do not give any history of ADL attacks in their lifetime. Therefore, the progression seems to be a passive phenomenon.

The definitive treatment for hydrocele is surgical; a variety of techniques have been recommended in medical science (Lord, 1964; Rodriguez et al., 1981; Wannas, 1983; Dandapat et al., 1984; Albrecht et al., 1991). Those patients are found to be infected with *W. bancrofti* they should be treated with DEC⁶ (Addiss & Dreyer, 2000).

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⁶ DEC is Diethylcarbamazine, the main drug available against lymphatic filariasis.
Control Measures of Lymphatic Filariasis

Currently there are two ways for the control of LF. Those are based on (a) Chemotherapy and (b) Vector Control.

From the last many years of experience with DEC, it has been found that even after the full regimen of treatment, some microfilariae still persist in the body. Due to this and other reasons like toxic effects it has not been possible to prevent the spread of filariasis by the administration of DEC alone. Chemotherapy must therefore be supplemented by an effective vector control programme, if the disease transmission has to be effectively prevented (Park, 1998).

Chemotherapy

At present Diethylcarbamazine (DEC) is the main drug available against LF. This drug is both effective and safe. The dose of DEC that is most generally accepted for the treatment of bancroftian filariasis is 6 mg/kg body weight per day orally for 12 days. This amounts to a total of 72 mg of DEC per kg of body weight. For Brugian filariasis recommended doses range from 3 to 6 mg of DEC/kg body weight per day, up to a total dose of 18-72 mg DEC/kg body weight (WHO, 1984).

After oral administration DEC is rapidly absorbed reaching peak blood levels in 1-2 hours. It is also rapidly excreted; the blood half-life is only 2-3 hours in alkaline urine and about 10-20 hours in acid urine. DEC causes rapid disappearance of microfilariae from the blood circulation. It is quite effective in killing microfilariae.

Filaria Control in the Community

Mainly there are three reasons for which filariasis never causes explosive epidemics (a) The parasite does not multiply in the insect vector, (b) The infective larvae do not multiply the human host and (c) The life cycle of the parasite is relatively long, 15 years or more. These factors favour the success of control programme (WHO, 1984). DEC is still today the only drug available for
chemotherapeutic control of filariasis. The administration of DEC can be carried out in various ways.

**Mass Therapy**

In this strategy, DEC is given to almost every-one in the community irrespective of whether they have microfilaraemia, disease symptoms or no symptom of infection. Generally it is accepted that mass therapy is indicated in highly endemic areas (WHO, 1984).

In many of the pacific Islands, mass treatment control projects using DEC have markedly reduced prevalence of *W. bancrofti*. But in India, the result of mass chemotherapy during 1958-60 has met with little success. This was due to poor population compliance. For mass chemotherapy to be accepted, a good rapport must be established with the community before the treatment begins. Therefore intensive health education is require for the general public for this.

**Selective Treatment**

Here, DEC is given only to those who are microfilariae positive. This type of selective treatment is more suitable for the low endemic region.

In Indian scenario the current strategy is based on detection and treatment of human carriers and filaria cases. The recommended dose in Indian programme 6 mg DEC per kg of body weight daily for 12 doses, to be completed in 2 weeks (i.e. 6 days in a week). Dosing once a week or once a month has also been recommended (WHO, 1967), but it is operationally difficult and not practicable excepting in individual patients who are highly motivated. In endemic areas, treatment must be repeated at specific intervals, usually every 2 years. This is partly because, despite remarkable antimicrofilarial properties, expected microfilaria clearance with DEC is incomplete at times even after adequate treatment. The other reason is that people living in endemic areas are exposed to reinfection (Park, 1998).
**DEC Medicated Salt**

DEC medicated salt approach is a special form of mass treatment using very low doses of the drug over a long period of time. Common salt medicated with 1-4 g of DEC per kg has been used for filariasis control in some endemic areas of *W. bancrofti* and *B. malayi* particularly after an initial reduction in prevalence has been achieved by mass or selective treatment microfilariae carriers. Treatment should be continued for at least 6 to 9 months. In the Lakshadweep Islands, this regimen has been shown to be safe, cheaper and effective (WHO, 1984). Meyrowitsch *et al.* (1996) recently showed that DEC salt was more effective than single dose DEC in reducing the prevalence of microfilaraemia. The combination of the long life of the adult parasite for several years and infectiousness of a patient with low parasitaemia represents a serious obstacle to control programmes based on chemotherapy alone (Temu and McMahon, 1981).

**Vector Control**

Vector control is a beneficial measure for disease control where mass drug administration fails or mass DEC administration is impractible, steps may be taken there to break the chain of transmission. To break the chain of transmission several vector control measures are there which can be applicable techniques for controlling mosquitoes. As LF is a *Culex* mosquito vector borne infectious disease, it is always wiser to take steps to check the mosquito breeding.

**Antilarval Measures**

Adequate sanitation and underground wastewater disposal system are the ideal method to destroy the mosquito-breeding place.

The anti-larval activities comprise the following:

1. Chemical Control

   - Mosquito Larvicidal Oil: Mosquito larvicidal oil (MLO) is active against pre-adult stages. It has been the main chemical used to control *Culex quinquefasciatus* for some time. Since it has proved to be less efficient
under field conditions and more expensive than other chemical preparations, it is being replaced by pyrethrum oil, temephos and fenthion.

- **Pyrosene Oil-E**: This is a pyrethrum based emulsifiable larvicide. The emulsion concentrate contains 0.1 to 0.2 per cent pyrethrias by weight and is required to be diluted with water before use. The emulsion is diluted in the ratio of 1:4.

- **Organophosphorous Larvicides**: During the past 10 years, organophosphorous larvicides yield successful results. However, the vector mosquito has developed resistance to many of these chemicals. The frequency of application is once weekly on all breeding places (NFCP, 1979).

2. Removal of Pistia Plant

The breeding place for mansonia mosquitoes is aquatic vegetation, i.e. pistia plant. So to control that mosquito, certain herbicides such as phenylene 30 or shellweed killer D may be useful for destruction of that aquatic vegetation (NFCP, 1979).

3. Minor Environmental Measures

Larvicidal operations are complemented by minor engineering operations such as filling up of ditches and cesspools, drainage of stagnant water, adequate maintenance of septic tanks and soakage pits, etc. Management of the environment is the most effective approach to the problem of controlling mosquito breeding (Park, 1998).

*Anti-Adult Measures*

The vector mosquitoes of filariasis have become resistant to DDT, HCH and dieldrin. The use of these compounds for indoor residual spraying, tried earlier has been discontinued. Pyrethrum as a space spray still holds promise. It is useful as a temporary means of personal protection but has no practical value in present day vector control programmes (NFCP, 1979).
**Personal Prophylaxis**

Though mosquito control is a complex procedure, it is wiser to take preventive measure to avoid mosquito bites by using mosquito nets. Screening of homes can be done to reduce the man-mosquito contact.

**Global Elimination Programme of Lymphatic Filariasis**

The strategy of the Global Programme to Eliminate LF has two components: firstly, to stop the spread of infection (i.e. interrupt transmission) and secondly, to alleviate the suffering of affected individuals (i.e. morbidity control). In 1993, LF designated as an eradicable disease (CDC, 1993) and a strong start was made in 1997 when the World Health Assembly passed a resolution calling for "...the elimination of lymphatic filariasis as a public health problem..." As per WHO developed strategies – by the end of 1999 documentation had completed on safety of co-administered drugs. By the end of 2000, 15 million people at risk, covered by active LF programmes, meetings for programme managers established in all regions and sampling techniques for certification of LF validated. By the end of 2001, 30 million people at risk, covered by active LF programmes, training curricula completed and mapping completed in more than half of endemic countries in all regions. As per WHO plan, by the end of 2002, 50 million people at risk, covered by active LF programme, and first ten countries certified LF free. By the end of 2003, 100 million people at risk, covered by active LF programmes and twenty countries certified LF free. In near future by the end of 2004, 200 million people at risk, will be covered by active LF programmes, thirty countries will be certified LF free and review will be done on regional eradication of LF.

While the best current understanding of the science of eliminating LF has been used to construct practical targets and strategies for action, it is clearly recognized that as the elimination effort develops, the scientific information will change and accordingly, the recommended practices must also change.

The first strategy of WHO is to stop the spread of infection (interrupting transmission). By doing mass drug administration to entire risk populations it can be effectively interrupt transmission of LF by reducing the number of
parasites in the blood to levels below which the mosquito vectors can no longer transmit infection.

**Treatment of Choice for Mass Drug Administration**

- Use of once-yearly treatment with single-dose of two drugs given together (albendazole plus either ivermectin or DEC) for 4-6 years.

- Exclusive use of table/cooking salt fortified with DEC for 1-2 years.

**Relieving and Preventing Suffering and Disability**

*The Principles Underlying the Strategy*

Lymphatic damage induced by filarial infection (frequently beginning in early childhood) causes lymphatic dysfunction that paves the way for lymphoedema, elephantiasis, hydrocele and other clinical manifestations. The most significant factor in producing lymphoedema and elephantiasis, and compounding the damage caused by filarial parasites, is bacterial "super infection" of the skin; these infections cause severe, febrile syndromes in patients while they destroy the delicate lymphatic vessels and exacerbate both the progression of disease and the frequency of clinical symptoms. Prevention of bacterial super infection removes some of the factors responsible for disease progression and permits partial resolution of clinical damage; this prevention can be effected through simple but regular and sustained washing and skin care. For most hydrocele surgery is the treatment of choice: for other urogenital manifestations of filarial disease, optimal surgical procedure must still be defined.

*The Treatment of Choice*

Intensive local hygiene to educate both patients and health workers that intensified local hygiene, bandaging, limb positioning and regular exercise can relieve suffering. Prevent exacerbation and progression of symptoms, and even reverse some of the clinical damage already sustained.

*Integrated Approach*

The control of the transmission of the disease filariasis a complex challenge. None of the single method can bring a fruitful result so an integrated approach
is needed to control filariasis using all the above strategies and approaches in optimum combination.

In filariasis control four major breakthroughs have occurred. The first of these is the development of safe, single dose, annual drug treatment. It has been proved that a single dose of DEC is very effective even two years after treatment. A single dose ivermectin has proved to be equally effective. A combination of single dose of both drugs reduced microfilaraemia more than 95 per cent 2 years after treatment. Secondly, intensive local hygiene on the affected limb with or without the use of antibiotic and antifungal creams, has been shown to have dramatic effects by halting the progression of or even reversing elephantiasis and lymphoedema. Thirdly, DEC medicated cooking salt has been introduced in India. The carefully controlled addition of very low concentration of DEC has been long been recognized as an effective means of eliminating LF infections in communities. Finally, there has been the development of insecticide sprays and polystyrene beads to seal latrines and roof-top water storage tanks to eliminate or reduce populations of urban *culex* mosquitoes (WHO, 1996).

**Study in Filariasis: A Review**

Lymphatic filariasis has emerged as an increasingly important public health problem in several areas around the world (Albuquertne *et al.*, 1995; Harb *et al.*, 1993). An estimated 120 million persons in Africa, Southern Asia, the Western Pacific Islands, the Atlantic coast of South and Central America, and the Caribbean are affected; of these, estimated 44 million persons suffer from lymphoedema of the extremities or urogenital disease (WHO, 1994). The physical debilitation, social stigmatization and economic losses associated with LF made the LF a leading cause of disability and it is the second leading cause of permanent physical disability in human next to mood disorders (WHO, 1995). LF is the most common cause of lymphoedema and hydrocele, where the nocturnally periodic forms of *Wuchereria bancrofti* transmitted by *Culex quinquefasciatus* is responsible for 95 per cent of the disease burden and *Brugia malayi* transmitted by *Mansoniasia* mosquitoes for the remaining 5 per cent,
together accounting for 40 per cent of the infected people in the world (Michael et al., 1996). This disease is geographically limited to economically backward countries of the world hampering their socio-economic progress considerably (Ottesen, 1994).

The Global Programme has identified the Indian sub-continent as the region in which the greatest numbers of people are at risk of LF: of the total 514 million, 454 million are in India alone. The WHO reported that 295 million (5.74 per cent) of sub-continent’s population is at risk population (WHO, 2003). The physical incapacitation associated with ADL episodes emphasizes the significance of LF as a major public health problem of substantial socio-economic consequences. However, the prevalence of disease in different communities and different geographical regions varies. *Wuchereria bancrofti*, the parasite for bancroftian filariasis has been reported in various parts of the world. ADL episodes are more frequent in the age group of 40 years and above. Individuals with chronic manifestations seemed to be more vulnerable to ADL attacks with 62.2 per cent acute episodes compared to those with hydrocele and “normal exposed”. ADL episodes ranged from one to five and the mean duration of episode was 8.6 days. In 72.5 per cent of the episodes the affected individuals were incapacitated and unable to do their normal activities for an average duration of 3.7 days (Gasarasi et al., 2000). Again, among the children of aged 1-15 years, microfilariae prevalence is significantly higher (Meyrowitsch et al., 1995). Prevalence of hydrocele in adults (more than 20 years of age) is more compared to elephantiasis (Onapa et al., 2001). The total disease attributable to filariasis is significantly higher in male than female due to the occurrence of hydrocele in male. While the prevalence of chronic signs are clearly age-dependent (Pani et al., 1991). On the other hand the prevalence both microfilaraemia and elephantiasis in the urban area is higher than that of the rural area. In both the areas, prevalence of microfilaraemic generally increases with age, to a maximum in those aged 20-29 years and then declines within most age groups. The prevalence of microfilaraemia and elephantiasis are higher in male than that of female. However the prevalence of microfilaraemia in female is higher than that of male (Sharma et al., 1999). However, it has been observed
that microfilaraemic (Mf) rate, filariasis disease and Mf rate by sex are not different (Kumar and Dash, 1994). Recent study in Orissa also reports that the prevalence rate is more in case of male compared to female. Again the prevalence of various forms of the disease is more age dependent in both sexes. About one-seventh of men and women of higher age groups suffered from chronic debilitation forms of the disease (Babu et al., 2001). There is also a dynamic progression through the grades of lymphoedema and that the frequency of ADL episodes is positively associated with this progression (Pani et al., 1995).

Studies in endemic areas indicate that far from there being any simple direct relationship between microfilaraemia and disease status, it is possible to find some individuals with microfilariae in their blood but no disease and indeed with all other combinations of infection and disease status (Hairston & de-Meillon, 1968, Hairston & Jaclowski, 1968; Beaner, 1970; Bryan & Southgate, 1976; Denhson & Mc Greevy, 1977 and Pani et al., 1991)

Though filariasis is a major public health problem in various tropical countries still people do not know the reasons behind filariasis. Studies from different parts of the world reveal about people’s poor knowledge about filariasis. Study results from Thailand show that community members are well aware of filariasis, and they believe, it has been endemic since the villages establish over one century ago. Most villagers moreover know who has enlarged legs, and the local term for this condition is teen-to. Although local filariasis personnel introduced the term “elephantiasis” into the village during recent surveillance activities, community members remain unfamiliar with this “foreign” term. Further while filariasis is endemic and despite control programme efforts, most villagers appear unsure of its etiology, mode of transmission and means of prevention. With regard to filariasis etiology, most villagers understand the illness to be either an inherited condition or one that can be contracted through poor blood and air circulation. Lack of blood vessels across the arches of the feet, excessive physical labour, drinking or bathing in water from the “unpure” plu, supernatural forces, or personal contact when a person has a fever etc. are the factors behind the cause of LF (Rauyajin et al., 1995).
Studies from Northern Ghana report that people attribute supernatural causes to filariasis. According to people, elephantiasis of the leg is usually attributed to spiritual causes. It is believed that during "war dances" which are normally performed at funerals, Juju men from different clans display their spiritual powers by throwing "spiritual medicines" on the ground. Any unsuspecting person who steps on these could get elephantiasis of the leg (Gyapong et al., 1996). It has also found in Haiti that the most common cause of elephantiasis people perceived is that by walking barefoot and stepping on a bug, worm, or microbe in the ground or in water, while most thought that trauma to the testis is the cause of hydrocele but did not relate mosquito bite to filariasis (Eberhard et al., 1996). In India also very small proportion of people in endemic areas know that filariasis is caused through mosquito bites and maximum unaffected individuals are either uncertain or feel that filariasis is not preventable. But the clinical manifestation of filariasis such as ADL, lymphoedema of limbs and hydrocele are well recognized by the community members and described by local terminologies (Ramaiah et al., 1996; Nayak et al., 2001). People believe that hydrocele is considered to have no link with the other disease manifestations but is believed to be inherited and to some extent enhanced by hard work, excessive drinking of palm-wine and sexual activities (Ahorlu et al., 1999). Similar beliefs about LF disease have been reported from other endemic areas of the world, such as the Philippines (Lu et al., 1983), Thailand (Rauyajin et al., 1995), India (Bandyopadhyya, 1996; Ramaiah et al., 1996), Haiti (Eberhard et al., 1996, Coreil et al., 1998) and Ghana (Gyapong et al., 1996b). In all endemic area, very small proportions of people know the role of mosquito in the spread of filariasis. In Orissa also very small proportion of people know that filariasis is caused through mosquitoes. The other ways of spread of elephantiasis, cited by the people are fate or evil spirits, heredity, impure drinking water, heavy physical work, climate, etc. (Nayak et al., 2001).

Although, the disease LF does not result in immediate mortality, the associated morbidity is believed to cause significant disability, with severe social and economic consequences to its victims (WHO, 1992). The spectrum of disease ranges from periodic, recurring attacks of localized inflammation, tenderness and
pain, often accompanied by fever, nausea and vomiting, known as acute adenolymphangitis (ADL), to chronic symptoms including lymphoedema, elephantiasis and hydrocele. Acute attacks are often debilitating, preventing those affected from performing their normal activities for a period of time. Chronic disease has also been assumed to be debilitating, leading to a restriction in the duration of activity and perhaps, to changes in activity patterns (Evans et al., 1993). Little information exists on the social and economic impact of the disease (Evans et al., 1993). Although some estimates of the prevalence of the chronic symptoms do exist, their association with acute attacks and their impact on daily activities also remain to be systematically documented. However, little data on the expenditure incurred by affected households or individuals loss of work time, etc. have been generated. Another attempt to quantity the impact showed that LF account for a relatively low proportion (0.23%) of the global burden of infections and parasitic disease in terms of Disability Adjusted Life Years (DALYs), but the accuracy of this estimate must be doubled, given the lack of reliable data on which to base the calculation (Anon, 1993).

Study from South India state that the disease hampers about 66 per cent of patient's occupational activities. They either work fewer hours or alter their activity. Some have completely given up their job. Domestic chores of most of the female patients are also impeded. Most of those affected try to avoid travel. The disability is work in patients with acute disease (Ramaiah et al., 1997). The incidence of ADL, which lasted an average of 5 days, is found to be very high in northern Ghana. Most of those affected are unable to perform their normal activities for much of ADL episode (Gyapong et al., 1996). Studies from south India state that nearly one-third of the acute adenolymphangitis (ADL) episodes do not seek treatment and for 27 per cent of the episodes they consult health personnel, undergo treatment and pay for it. On average, the ADL patients spend Indian Rupees (INR) 2.35 (US$ 0.07) per episode on treatment, but expenditure is as high as (INR) 32.11 (US$ 0.92) among those who pay. Doctor's fees and medicines constitute 83 per cent of the total treatment costs. Patients with multiple and longer duration episodes and with better living conditions spend relatively more on treatment. The proportion of patients who spent money on
treatment is smaller in poorer households, but their treatment costs form a relatively higher proportion of their income than those of middle and high-income households. The ADL episodes curtail economic and domestic activities. In 87 per cent of the episodes, the affected are not able to attend any economic activity compared to 37 per cent of the episodes in the case of controls. Patients spent only $0.68 \pm 1.91$ hours on economic activity during the ADL episodes, which is significantly lower than that among healthy controls (Ramaiah et al., 1998).

Chronic cases treatment studies from South India showed that about three-quarters of the patients seek treatment for filariasis at least once and 52 per cent of them pay for treatment, incurring a mean annual expenditure of (INR) 72 (US$ 2.1). Doctor's fees and medicines constitute 57 per cent and 23 per cent of treatment costs respectively. The proportion of people seeking treatment is smaller and treatment costs constitute a higher proportion of household income in lower income groups. Most patients do not leave work, but spend only $4.36 \pm 3.41$ h per day on economic activity compared to $5.25 \pm 3.52$ h work by healthy controls; the mean difference of $0.89 \pm 4.20$ h per day is highly significant ($P<0.01$). This loss of work time is perpetual, as chronic disease manifestations are mostly irreversible. As estimated 8 per cent of potential male labour input is lost due to the disease. This study results clearly show that the chronic form of LF inflicts a considerable economic burden on affected individuals (Ramaiah et al., 1999). Studies from Tamil Nadu revealed that direct treatment cost per year per patient is found to range from (INR) 30 to 101 among patients with different manifestations. Income foregone (indirect cost) annually by each patient, which is a function of frequency and duration of ADL range from (INR) 182 to 702. ADL episodes among filariasis patients alone cost a minimum of Rs.4515 million for the nation every year. Cost benefit analysis of filariasis control programme in India showed that the benefits in terms of savings on treatment and work loss due ADL alone exceed the cost by 24 per cent. The per capita cost of the National Filaria Control Programme was calculated to be (INR) 2.6 per annum (Krishnamoorthy, 1999).
These economic burden estimates have some limitations. The most important is that data collected from the studies carried out in South India have been extrapolated to the whole country. In addition, figures on the prevalence of chronic disease used in calculations are based on epidemiological surveys carried out several decades ago (Michael et al., 1996). Quantitative information on the economic burden and costs of control on other countries is very sparse (Evans et al., 1993). However, a few studies in Africa have demonstrated that filariasis reduces the productive capacity of those affected, and that the health care system; in addition the patients incurs considerable expenditure on the surgical treatment of hydrocele (Wijers and Kinyanjui, 1997; Wegera et al., 1979; Muhondwa, 1983). A recent study in Ghana has shown that as much as 7 per cent of the potential male labour input could be lost because of chronic filariasis (Gyapong et al., 1996). These data suggest that the cost benefit of filariasis control in these countries is also likely to be high (Ramaiah et al., 2000).

Besides economic burden, people with LF also experience socio-phychological problems (Ramaiah et al., 2000) and patients having big hydrocele suffer in sexual disability (Dreyer et al., 1997). Patients suffering from hydrocele face a greater psychosocial burden. Unmarried men in particular find it difficult to get a spouse with their condition, and various degrees of sexual dysfunction are reported amongst married men and hydrocele causes damage to male identity (Gyapong et al., 2000). Women with elephantiasis have treated as un-wanted wife and their disfiguring creates embarrassment, shame, cultural constraints and psychological problem (Bangopadhya, 1996).

The control of LF has suffered for a long time from lack of research and funding. However, during the last decade, significant research advances have led to a better understanding of the severity and impact of the disease and to the development of efficient tools for diagnosis and strategies of treatment/control (Nicolas, 1997). The development of feasible control measures, which can be applied on a community basis, the successes of recent programmes and increasing political commitment led the 50th World Health Assembly in May 1997 to pass a resolution identifying as a priority “the elimination of LF as a public health problem” (WHO, 1997).
The ultimate aim of filariasis control is to eliminate or reduce the amount of acute and chronic filarial disease. Despite a sketchy understanding of the mechanisms of filarial pathogenesis, it has been demonstrated empirically in many places that drastically reducing or eliminating transmission prevents or greatly reduces the incidence of clinical diseases in the community. These reductions are obvious when the prevalence of microfilaraemia is kept at 1-2 per cent. The efforts on morbidity of only moderate reductions in transmission, and the long term effect of individual treatment without interfering with transmission are the focus of current research on filariasis control strategies; studies now under way should provide answers within the next few years. At present, however, control activities aim to interrupt transmission (Evans et al., 1993).

Transmission may be affected by (1) controlling the reservoir of infection through chemotherapy; and (2) reducing or eliminating contact between the vector and the human populations, either by reducing the size of the vector population or by modifying human behaviour. Chemotherapy and changes in risk-related behaviour (e.g. use of bed nets, cleaning up of breeding sites around houses) are the most likely means of benefiting individuals in the absence of community wide programmes, but these options have yet to be proven effective.

Three characteristics of filariasis are of particular significance for its control. First, there is no animal reservoir of infection for the prominent species, *Wuchereria bancrofti*, and in only a small part of its range is there an animal reservoir for *Brugia malayi*. Unlike many other parasitic infections, which infect animals as well as humans, eliminating LF from the human population eradicates the disease. Second, filarial worms are long-lived. Although the evidence is only indirect, a lifetime of 7 years or more is suggested for adult worms, and at least one year (possibly 2-3) for Mf. Control programme must, therefore, be long-lived. Third, in most places, *Wuchereria bancrofti* is nocturnally periodic-transmission takes place largely in the evening and at night -- and blood samples for testing must be taken at night (not withstanding the DEC provocative test that has been used in some places during the day). Local conditions may also be important.
Where vector control is concerned, effectiveness may be influenced by the predominant species of mosquito vector, and its breeding, traveling and biting habits, the "efficiency" with which the vector transmits filariasis may be important, though comparative data from around the world are scare on this topic. The degree of in-and out-migration of the human population may also affect control efforts (Evan et al., 1993).

Control of *W. bancrofti* parasitism should rely on community based distribution of filariacidal drugs and whenever feasible, in vector control as a complementary tool. Three drugs have been shown to be safe and effective for large-scale application in filariasis control programmes. Diethylcarbamazine (DEC), used for 50 years (Ottesen, 1985) and recently ivermectin and albendazole. Fundamental shifts have occurred in chemotherapy strategies (Nicolas, 1997).

Decision-making in filariasis control programmes (especially when to stop) has for a long time suffered from the lack of sensitive and convenient tools to monitor *W. bancrofti* parasitism in humans. In consequence, several programmes have been stopped too early, leading to re-emergence of endemicity a few years later. This happened in French Polynesia, where control programme with DEC, started in the 1950s, led to a sharp reduction of microfilarial prevalence (Perolat et al., 1986 quoted by Nicolas, 1997). However, ten years after interruption (in 1982), the incidence of the parasitism (microfilaraemia) reached pre-control levels (20-30%) microfilaraemia in some islands, indicating that the adult worms had persisted (Cartel et al., 1992). Drug treatment should kill both microfilariae (for interrupting transmission) and adult worms to prevent re-emergence of the parasitism. Treatment strategies have been simplified and now rely on ingestion of single doses of drugs once or twice a year instead of the formerly recommended 14 day course of DEC, which has no more effect than single-dose treatment (Cao et al., quoted in WHO, 1997). After its success in tried *Onchocerca volvulus* control in Africa, ivermectin was tried against *W. bancrofti* in French Polynesia (Roux et al., 1989) and shown to be microfilaricidal (Ottesen and Campbell, 1994). DEC and ivermectin, after ingestion, kill microfilariae within 1-4 hr and keep the MF level low until 6 months after treatment, but a major
outcome is the discovery that treatment using 2 drugs is significantly more effective than single-drug treatments (Moulia-Pelat et al., 1994). Likewise, a study carried out in Sri Lanka showed that albendazole, a drug used against intestinal parasites, has a weak microfilaricidal effect when administrated alone but reduced the microfilaraemia by 99 per cent for a year, when combined with ivermectin, as with ivermectin plus DEC (Ismail et al., in WHO, 1997).

Control programmes should now be based on the treatment of the entire human population in a given endemic area (to avoid false-negative diagnosis). Mass community treatment should be applied yearly, for 4-6 years, with a combination of DEC 6 mg/kg and ivermectin 400 μg/kg. The later also being active against other parasites such as intestinal worms, lice and scabies (de Silva et al., 1997).

In India in the past five years, several steps have been initiated to move from control to elimination of LF. The most important of these steps is the introduction of annual single-dose mass treatment with the traditional drug DEC, in 11 districts with a population of 32 million across six states (Ottesen, 1997). There are plans to extend the elimination programme to 100 districts over the next five years (Das et al., 2001). Such efforts are being complemented with the introduction of albendazole, which yields "beyond-filariasis benefit’s including enhanced nutritional benefits and effects on intestinal helminth infections and other parasites" (Ottesen, et al., 1999). With this background, the present study makes an attempt to understand the bio-cultural dimension of LF in rural coastal Orissa.