Tuberculosis and leprosy today, are the major health problems in the tropical and subtropical world. Both are chronic diseases, caused by Mycobacteria and are, our greatest enemy which have afflicted the world from centuries.

Leprosy was the first human bacterial disease recognized by a Norwegian Scientist Armauer Hansen working in Bergen in 1873. He identified the bacteria microscopically in the tissues of patients with leprosy. This was at a time when human diseases due to infection were not accepted and this discovery preceded by years of Koch's discovery of tubercle bacillus.

The origin of leprosy is shrouded in the mists of antiquity. Its earlier spread is also a matter of surmise. WHO has made several attempts to collect global data on the leprosy situation. The estimated number of cases in the world today is around 12 millions distributed over several countries. This disease however, has been present in the Indian subcontinent for several centuries. Information regarding its extent and distributions have been meagre until recent years. The 1931 census gave the number of leprosy cases as 1.5 million. Since then antileprosy work has been intensified specially in the post independence, periods after 1948, more particularly after the National Leprosy Control Programme which was launched by the Government of India in 1955. The estimated number of cases have consequently been rising in successive decennial population counts. The main factors responsible for progressive increase were; rapid increase in population, increased case findings and increased voluntary reporting due to community awareness. The approximate number of cases in the country is 4 million based on the 1981 census population of 683.9 million. The lepromatous rate ranges between 10-25% in different areas and the deformity rate is roughly 20%. About 400 million people live in hyperendemic and moderately endemic areas and are exposed to the risk of leprosy. In the country 72 districts have been identified hyperendemic for leprosy with a prevalence rate of about 10/1000. The magnitude of the problem is therefore, vast and consequent need to achieve the control over the disease gains a greater urgency in the context of attaining our social objective of health for all by 2000 AD.
Leprosy is normally a very chronic disease. The severe forms tend to deteriorate with time, and the most severe and contagious forms last for life, without much affecting the life span. The life expectancy of patients in some cases is shortened by a few years specially in the lepromatous forms. In more than one third of untreated or advanced cases, leprosy results in disabilities which increase with time and become permanent. These disabilities affect mainly the extremities and the face including the eye, resulting in serious impairment of working capacity and disruption of the social life of the patient, who becomes an outcast in the society.

The disabilities and deformities of leprosy patients have in many cultural systems, resulted in the belief which may even be held today by health workers, that the disease has necessarily incurable consequences. The degree of social ostracism resulting from this attitude makes the patients themselves convinced that their exclusion from the community is justified. A similar feeling of guilt may even be shared by the patient's families.

Leprosy afflictions go through phases. The greater part of the population having contact with patients with multibacillary forms of leprosy may be infected. The defense (Immune status) in most cases will limit and arrest the multiplication of leprosy bacilli before signs of the disease appear. In some individuals, the disease will progress to clinical forms of leprosy. The earliest recognised form of the disease is indeterminate leprosy. This is an early unstable form in which majority of the patients will control the infection leading to spontaneous healing without treatment (Lara and Nolasco, 1956). Some patients with indeterminate leprosy will progress to more persisting disease with a spectrum from pauci-bacillary tuberculoid (TT-BT) leprosy to multi-bacillary lepromatous (BL-LL) leprosy. The last form is the most infectious and has the longest incubation period. An average period of four years ranges from exposure to the appearance of clinical signs of the disease.

Sulphone therapy in leprosy was started in 1941 and it was believed that leprosy could be controlled with the help of this drug. The appearance of Dapsone (DDS) resistant leprosy, and the need to continue DDS treatment for life long in lepromatous leprosy patients, has led to a failure in the control of leprosy by DDS alone in most areas of the world with leprosy. Today on a world scale leprosy might be on the increase (Lara & Nolasco, 1956).
The screening programmes to detect cases of leprosy started long back in the leprosy endemic areas. But the progress has been and still is hampered by difficulties encountered in the diagnosis of early leprosy and leprosy in the incubation period. It is increasingly important that an early diagnosis of leprosy be made since antileprosy treatment can limit the spread of leprosy and it is much easier to treat leprosy in earlier stages than in the advanced state. Epidemiological studies of leprosy have been and still are difficult to carry out for want of methods to identify human beings infected with leprosy bacilli in the absence of full blown symptoms. The real problem has been to detect the early cases.

Koch's delayed hypersensitivity (1890) test (tuberculin) has helped enormously in calculating the percentage of people infected with tuberculosis who later develop the disease. On the same pattern lepromin test was evolved by Mitsuda in 1919, but this test has not provided any useful information excepting that it differentiates between tuberculoid and lepromatous form of the disease. Therefore, a very important tool in diagnosing leprosy becomes practically useless and other methods have to be looked upon. In the past several years other tests have been used with little success. Mention may be made of complement fixation test where the antigen or antibody may be detected through serological tests but this test in leprosy has not proved to be useful. Rubino test and antigen-antibody detection in gels by Ouchterlony or Oudin methods have been used in the past with no success. During recent years several other tests have been devised and used with some success. These tests comprise of cross-immunoelectrophoresis, fluorescent antibody test, radioimmune assay and enzyme linked immunosorbent assays. These tests have provided certain very useful information and may be used in detecting cases suffering from leprosy.

Crossed Immunelectrophoresis test has been developed and tried to a limited extent. Rojas Epinosa et al. (1976) using this technique demonstrated the presence of anti-mycobacterial antibodies in the sera of patients suffering from lepromatous leprosy. The antigen was prepared from isolated Mycobacterium leprae. Harboe et al. (1977) using this test showed that lepromatous sera contain over twenty antibodies reacting with Mycobacteria.
Harboe et al. (1978) developed radio-immunoassay with the use of radiolabelled *M. leprae* antigen prepared from purified sonicated *M. leprae*. A method was developed by Buchanan for the early detection of systemic infection in armadillos through a sensitive ELISA test which detects antibody to arabinomannan. This test appears to be a simpler one and is stated to be specific.

Studies on enzymatic pattern and protein estimations have been carried out in isolated cases. These studies indicated that there is certainly an appearance of serum enzymatic changes in early phases of the disease. The protein values have also indicated gross abnormalities in different cases. But the changes in the level of enzymes or proteins have not been correlated among themselves. Since these changes are non-specific and may appear in several other diseases a positive correlation has to be established by using some specific parameters of study along with these non-specific ones. Serum immunoglobulin patterns have been studied in leprosy at times, but these specific changes have not been analysed in relation with other parameters.

A readily available and chemically characterized leprosy specific antigen would be a valuable tool for diagnosis and toxonomic studies. The vaccine programme at IMMLEP has recently led to an unexpected breakthrough in finding *M. leprae* specific antigen. The Wellcome Foundation in the course of removing large amount of bacteria from armadillo tissues for making trial vaccine, produced liters of left over supernatants, containing soluble molecules from the sick animal tissues. This was reacted with serum from leprosy patients on the remote chance that it might contain surface molecules or antigens specific to *M. leprae* which would bind with patients antibodies. The solution contained the antigen, which was at a higher concentration than in the bacteria themselves. This indicated that it is actively secreted by the bacteria. The antigen was identified as a simple universal glycolipid having three sugar molecules with a fatty component of *M. leprae* cell wall. It has been synthesized at National Institute for Medical Research, London and is presumed to work as an artificial vaccine costing much less than the materials obtained from Armadillos. Scientists at Seattle have modified the antigen and bound it to plastic, plates to conduct ELISA. When serum containing antileprosy antibody
is added to antigen coated dishes, antibodies are bound to the dishes. A colour producing test for bound antibodies then reveals that sera contained anti-\textit{M. leprae} antibodies. This test seems to be absolutely specific for leprosy antibodies, and will not react with blood from people with related mycobacterial infection.

Despite the sensitivity and specificity attached to this antigen it would be difficult to procure it since the source of \textit{M. leprae} itself is limited and is constantly dying out due to mass treatment. Therefore, some alternatives have to be thought of. If some antigenically related atypical mycobacteria, which is capable of detecting the same or appropriate percentage of cases of leprosy like that of \textit{M. leprae} glycolipid, is sorted out the problem of scarcity of antigenic availability may be abbreviated.

This laboratory has been working from the past several years on an antigenically related strain of \textit{Mycobacterium} designated \textit{M. habana}. Its possibility to detect antileprosy antibodies in humans through cell sonication or in particulate state may provide some useful information regarding the diagnosis of leprosy.

This study has been undertaken for:

1. Detection of \textit{M. leprae} specific antibodies through ELISA test using particulate \textit{M. leprae} antigen as control.

2. Comparison of \textit{M. habana} antigen through ELISA test to detect \textit{M. leprae} antibodies in the sera samples of leprosy patients.

3. Patterns of Immunoglobulin in the sera of leprosy patients.

4. Serum enzyme profile during various phases of leprosy along with contacts and healthy individuals.

5. Total serum protein and albumin/globulin estimations in the sera samples of various grades of leprosy and its comparison with tuberculosis patient sera.