Introduction
The study of human diseases has been traditionally a major focus of interest of biological anthropologists, since the very inception of discipline, as most of the initial contributors were physicians. Another significant reason is that diseases have played a major role as agents of natural selection to channelise human evolution and group differentiation in man. In this context, the contributions of Allison (1954) and Livingstone (1958) on distribution of sickle cell haemoglobin in Africa attained the status of classics in human population genetics. The present day distribution of haemoglobinopathies is explained as genetic adaptation to environment where malaria is endemic (Motulsky, 1960).

In medical science, anatomical and physiological dimension of human bodies have been separate from mind. Thus, in this cartesian model (Eisenserg, 1977), health was taken to mean as a disease free physical state. Though, great achievements have been made in the fields of diagnosis, chemotherapy and surgery, its philosophical orientation coercively divorced of biology from psychology and socio-cultural environment.

Medical anthropology, as an independent subfield of anthropology, is of recent origin; yet the roots of medical anthropology are as old as anthropology itself. The development of ideas and theoretical orientations in anthropology has a direct bearing on the style of enquiry in medical anthropology. Two fold definition by Foster (1979) highlights the “biocultural inter-relationship between human behaviour” and “health and disease”, as theoretical exercise and the utility of this knowledge in health related planning “through changing of health behaviour in direction believed to promote better health”.

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The long tale of man’s evolution, his achievements in the course, right from the moment, he first emerged from animal ancestry is remarkable. In this, biological variation played a major role in his successful adaptation to many different environments. Though all living men belong to the same species, i.e., *Homo-sapiens*, mankind is not geographically uniform. There is a diversity of human species which is clearly divisible into distinct *races* which are the major segments of the species. These races are the expressions of the pattern of diversity and are the manifestation of human variability. The description and explanation of variation is a challenge to a physical anthropologist who is concerned with its biological significance. Best physical anthropological classification of men is based on genetic characters (Boyd, 1942). Sources of variation lie in genetic differences and shuffling of genes, that are responsible for manifestation of each and every character and for different ethnic groups.

The principle of population genetics permits the scientific study of man in nature without the handicaps of small family size and lack of experimentally planned marriages. The recently developed haematological, serological, biochemical and other techniques and theoretical advances in human genetics have resulted in enormous progress in understanding human variation in the past few years.

Development in techniques to study human race has brought about many important changes in aims, perspectives and means employed to study the variation in human populations. Earlier population geneticists, used to study mostly the morphological characters, or metric traits which they believed were influenced by environment. Since then, the primary interest of anthropologists throughout has been to study
human variation. The process of development of genetically distinct population within a single species, sub-species, geographical races etc. during the course of generations is called micro-evolution. The evolutionary changes that have occurred in human beings from early times can be easily understood by biological variations. Similarly, the genetic variation that is discernible in different human populations and forms the basis of human variations is the result of human evolution which has led to varying gene frequencies in different breeding populations. Gene frequencies are simply the proportion of different alleles of a gene in a population. To obtain these proportions, we count the total number of individuals with various genotypes in population and estimate the relative frequencies of alleles involved. The main agencies leading to genetic variability among populations are natural selection, mutation, migration, genetic drift, and hybridization.

The hybridization and mutation introduce genes into a population and the natural selection determine what happens after they have been introduced.

Man’s genetic or biological changes are extremely slow. The organic evolution occurs through a process of selection acting on random genetic changes. There are chance differences that arise in genes or combination of genes (mutation and recombination), which produce a variety of effect on their carriers. It is important to note that differences among populations of the same species in different geographical regions are quantitative rather than qualitative variations not in actual genes present but in the relative frequencies of the different alleles. Races may be defined as populations that differ in the relative frequency of gene alleles. A human race, genetically determined, is a population which
differs significantly from other populations with regard to the frequency
of one or more genes.

Genetic evolution is slow since it must wait for a fortuitous accidental genetic change in Deoxyribose Nucleic Acid (DNA) before it can proceed and each change may take a considerable number of generations, before it is incorporated into the population (Strickberger, 1976). Genetics lies at the heart of the evolutionary problems because genes are the material links between generations and phylogenetic change depends on changes in the properties and frequencies of genes. However, the precision with which we can defuse the genotype, depends largely on the kinds of the characteristic, we choose to study. As far as anthropologists are concerned, population genetics is a vital aspect of their approach to genetic problems. The various groups of people are studied in order to compare their characteristics, with those of other in the groups. Populations which are in the habit of mating one another rather than with the members of other groups are known as breeding population.

The inheritance of most of the human attributes are a result of combination of several genes that may be affected by the environment in which the individuals grow. The pattern of mating determines the distribution of genes from one generation to the next. Mating in man is constrained by social as well as geographical barriers. People also tend to choose their mate from their social milieu as they have more opportunities to do so. Such behaviour may impede gene flow between groups. Societies also differ in the degree of relationship between mates that they encourage (Mckusick, 1978). It is generally agreed that we, all the living men, on this earth, belong to a single species Homo-sapiens,
but mankind, like most other animal species, is not geographically uniform. Within any population, individuals differ widely in various traits. Anthropologists have made repeated efforts to subdivide humanity into races or breeds, supposedly distinct from one another. The characteristics which are used to subdivide the human population into races, should fulfil the criteria given by Boyd (1950), namely:

1. It should be objective.
2. It must not be subject to modifications by environment.
3. It must be determined by one or a small number of genes.
4. It should be non-adaptive, for example they must not have any great selective value in evolution.
5. The criteria must not mutate at too high rates.

The reality is that these criteria are not satisfied and that the ideal phenotypes do not really exists. Every trait at disposal has disadvantages, and that is why it is wise to use not only one but several.

Using genetic data from living population for historical reconstruction. It is generally assumed that the frequencies of the relevant genes have remained stable or atleast, have changed slowly in relation to the time scale, which is being considered. It should be assumed that the value of comparative population research depends solely on its contribution to historical problems.

With the tremendous advancement in the field of medicine, new investigative as well as diagnostic tools are becoming available to aid the physician in the diagnosis and treatment of the disease. The knowledge of fundamental genetic principles is essential for optimal management of
neurological disorders. The contribution of medical genetics in the understanding of aetiopathogenesis of diseases have been phenomenal in recent years. Attitude towards preventing hereditary disease has changed tremendously and changing fast all over the globe (Maheshwari, 1993).

Epilepsy has been observed as far back as history records and throughout the ages people with epilepsy have made lasting contributions in many fields. The word epilepsy refers to a group of disorders characterized by recurrent epileptic seizures. Differential diagnosis first requires distinction between epileptic seizures and the many systemic, neurologic, and psychiatric disorders associated with paroxysmal behaviours that might be mistaken for epilepsy. The various forms of epilepsy and epileptic syndromes are defined by a constellation of signs and symptoms that include characteristic seizures types, other clinical features and family history. Electroencephalography (EEG) is an important aspect for classification of epilepsies (Engel, 1995). In the last few decades, there has been a significant progress in the diagnosis, classification and drug treatment of seizures in persons with different epileptic syndromes. Human epilepsy is classified as idiopathic, cryptogenic and symptomatic groups. The term idiopathic refers to a set of syndrome presumed to be genetic in origin (Jain et al, 1997). The idiopathic or primary epilepsies are specific inherited syndromes associated with characteristic seizures types but with no structural abnormalities and no other neurological deficits (Engel, 1995). In the primary generalised epilepsies the abnormality is generalized and affects the entire brain. This abnormality is normally not demonstrable or apparent and therefore routine investigations do not reveal any lesion.
Seizures that develop secondary to known causes are called symptomatic. Symptomatic or secondary or acquired type of epilepsy results from some local pathology within the brain. They have normal seizure threshold before the development of symptomatic epilepsy (Maheshwari, 1993). The most common cause of symptomatic seizures includes head injuries, brain tumours, encephalitis, cerebrovascular disease, malformations, hypoxia, drug and alcohol withdrawal as well as metabolic disorders.

It is now well accepted that there are important genetic influences in epilepsies (Anderson and Hauser, 1985; Sharma, 1986; Bird, 1987). An understanding of the basic principles of human inheritance patterns is necessary to appreciate the variety and complexity of genetic influences in the epilepsies. The various modes of human inheritance include single gene (Mendelian), chromosomal, mitochondrial and multifactorial / polygenic. Males and females are equally affected, the disease appears over multiple generations, and heterozygous mothers or fathers transmit the gene with equal risk to sons or daughters. Examples of autosomal – dominant disorders associated with epilepsy include tuberous sclerosis, benign neonatal convulsions, juvenile myoclonic epilepsy, and some forms of 3 Hz, spike and wave absence epilepsy (Leppert et al, 1989; Silvestri et al, 1989; Delgado-Escueta et al, 1990; Durner et al, 1991). Some examples of autosomal recessive disorders associated with epilepsy include phenylketonuria, Tay-sachs disease, pyridoxine dependency seizures, and Lafora body myoclonic epilepsy. Examples of X-Linked recessive disorders associated with epilepsy include Menkes’ syndrome, Pelizaeus-Merz Bacher disease, and fragile-X syndrome (Bird, 1994).
Although Huglings Jackson and other neuroscientists in the mid-nineteenth century recognised the existence of many different types of epileptic seizures, the fact that these diverse ictal events actually reflect a variety of underlying basic mechanisms has only recently been recognised. The international classification divides ictal events into those that are generalised (beginning simultaneously on both sides of the brain) and those that are partial (beginning in a part of one hemisphere) (Engel, 1995).

Ancient views of epilepsy often associated this disorder with supernatural forces that were usually believed to be evil. Those who proposed natural causes were still likely to prescribe treatments based on superstition rather than the scientific method. Consequently, persons with epilepsy were double victims, suffering both from their medical disorder and from the stigma and occult treatments imposed on them by society. At present, the word epilepsy is used to refer to a class of epileptic disorders, defined as chronic neurological conditions characterized by recurrent epileptic seizures (Engel, 1995). Many religious ceremonies are performed to please gods and goddess. Injuries are inflicted on the patients in an attempt to eliminate the influence of demons and devils. The patients are even made to smell the soles of torn shoes. Sprinkling of water on the eyes and face are practised by some to purify the air. If social and environmental problems are not taken care of, then good control of seizures cannot be achieved (Maheshwari, 1993).

The ancient belief by Indians to make an epileptic patient smell a shoe may actually have something to offer by way of epilepsy control. Serious research is going on in the developed world to use aromatherapy
as a method to treat epilepsy. The observation made by ancient Indians are now being studied in the west and certain perfumes developed for the same are under testing process.

For long, what was considered a disease effecting pork eaters, neurocysticercosis (formation of cysts in the brain leading to epilepsy and other complications) is affecting vegetarians also. The parasite found in pigs was long known to effect the brain and body of pork eaters, is now finding its way to the brain and body of vegetarians. Neurocysticercosis is the most common cause of symptomatic epilepsies in India (Sawhney et al, 1996).

The above account clearly indicate that epilepsy is a very heterogenous disease and prevalence of epilepsy is also not uniform in various human populations (Mathai, 1971; Sethi, 1974).

Keeping this in view, biomedical-anthropological investigations on epileptics are very relevant. The genetic variation with reference to four traditional anthropo-genetic markers: dermatoglyphics, PTC taste sensitivity, ABO blood groups and ABH secretions have been investigated with reference to epilepsy.

**ABO Blood Groups**

In 1900, the first observation of the agglutination of human red cell by serum belonging to the same species was made by Landsteiner. This opened a vast field for further such other discoveries of human blood groups. The discovery that the blood groups were inherited characters greatly increased their biological interest. It is now becoming increasingly clear that blood groups may have played an important role
in influencing the survival and fertility of different genotypes, which is of great importance and interest for anthropologists. Mourant (1954) raised the question whether infecting organism are the primary factors for the polymorphism of ABO blood groups.

Brues (1954), likewise thought that it would be difficult to explain the distribution of ABO blood group frequencies among various human races in the absence of any selective force. Later in 1960, Vogel et al developed the idea that the geographical distribution of the ABO blood types has been greatly influenced by the severe selection exercised by the pandemics such as plague and smallpox. According to the theory developed by Vogel and his co-workers (1960), individuals are immunologically handicapped in the production of antibody against viruses or bacteria which carry antigenic specificities similar to those of their own blood group substances. In other words, the closer the structural correspondence between ABH and microbial capsular antigens, the poorer the resources for resistance in the host. Further, by virtue of his normal blood group anti-A or anti-B antibodies, an individual may be equipped with built-in protection against a certain category of organisms. Mourant (1954) had suggested fairly early that the selective screening imposed by infectious disease in the past must have favoured certain immunological characters which might relate to blood group specificity.

If we compare the frequencies of the ABO blood groups, or phenotypes, in specific diseases with those in the total population, we find that they are not same. Where the frequency of a given blood group is higher in the disease state than in the general population, it suggests that people of that blood group are more susceptible to the
disease than those of other groups, and if the disease is one with substantial mortality before or during the reproductive period, it is natural selection, so that the disease may eliminate that blood group.

**ABH secretions**

It is well recognised that some people secrete their *ABO* blood group antigens in their body fluids while other do not. The blood group antigens are confined not only to the red blood cells, but are also widely distributed in human body like amniotic fluid, meconium, bile, saliva, etc. The secretion into saliva and other body fluids of *ABH* substances is controlled by a gene *Se* with dominant expression and independent of genes controlling the *ABO* blood groups. Its recessive allele in homozygous condition (*SeSe*) produces the non-secretion state. The antigens in the body secretions differ from red cell antigens in their chemical constitution. These are water soluble (glycoproteins) while those on red cells are alcohol soluble (glycolipids). The ability or inability to secrete is an inherited character, secretion being dominant to non-secretion. Thus, the saliva of group *A* secretor contains group specific substance *A*; that of group *B* secretor group-specific substance *B*; and that of *AB* secretor a substance with both specificities. Persons who are group *O* secrete *H* substance which is not group specific, being found also non-secretors of groups *A*, *B* and *AB*. The presence of *H* substance is detected by the use of anti-*H* serum. This is rarely found in man, but occurs naturally in eel serum and in the seeds of certain leguminous plants, notably Ulex europeus (gorse).

Race and Sanger (1950) pointed out the marked biological difference between those people who do and those who do not secrete
their antigens, and suggested that there might be selective differences between them.

Following the discovery of ABH substances in saliva by Yamakami (1926) and in other body fluids and glandular secretions (Hartman, 1941), a tremendous interest was generated in this genetic endowment. The genetics of the aberrant secretors is not yet clearly understood. Clarke et al (1960) considered that the secreted antigen is polygenic. McNeil (1957) had suggested different genes for the secretion of AB and H antigens. The real importance of secretor system as a disease marker was realised when Clarke et al (1956), showed that duodenal ulcer patients had more non-secretors of ABH substances than the secretors. Mourant et al (1978) discussed the role of ABO incompatibility causing spontaneous abortions. Sheppard (1953) suggested that selective differences between secretion and non-secretion of ABH substances might be particularly important in diseases of the gastrointestinal tract, as the concentration of antigens is particularly high in saliva and gastric juices. Working on this possibility Clarke et al (1956) made a significant discovery that duodenal ulcers are more common among the non-secretors of ABH substances than in secretors. This was later confirmed by Wallace et al (1958); Doll et al (1961); Newman et al (1961); Ball (1962); Pringle et al (1961) and Fodor and Urcan (1966). Mourant (1988) gave a comprehensive account of association between infectious and genetic markers and observed that apart from blood groups, secretor system has emerged as an important disease marker as a result of recent advancement in this field.

showed a marked deficiency of ABH non-secretors as compared with the general population in infants suffering from ABO haemolytic disease of the new born, thus suggesting immunization of mother via the muconium and amniotic fluid. Mourant et al (1978) discussed a consensus that ABO incompatibility of the mother with respect to the foetus is a predetermining factor not only for the ABO haemolytic disease of the new born but of spontaneous abortion. In relation to ABO haemolytic disease of the new born, Mourant (1982) stated that it suggests conflict of mother and foetal blood groups operating between tissue cells and this could be due to mother carrying or having carried a secretor foetus of ABO incompatible blood groups, indicating a selective loss of secretor foetuses and hence secretor genes. Bhalla (1990) developed the hypothesis of selection-relaxation to account for sustained high frequency of non-secretors in the advanced human societies in striking contrast to the low frequency of recessive gene (Se) diminishing to zero in many primitive human societies and its complete absence in apes and monkeys. Emphasizing the protective role of secreted antigens against deleterious effect of lectins on gastric mucosa, Bhalla (1990) postulated that “the ability to secretor ABH substances in saliva and other glandular secretions was, perhaps, of paramount adaptive significance at a stage, in the early history of man, when he was eating raw food (rich in lectins) like other animals. With the invention of fire making techniques, food habits of early man witnessed a radical change from raw to cooked food. This, in all likelihood, resulted in the reversal of negative selection pressure bearing upon the non-secretor allele leading to its establishment and maintenance in human population at frequencies much above the level of its mutation rate”.

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**PTC tasting ability**

An easily discernible hereditary character in the man is the ability or inability to taste a substance known as phenylthiocarbamide (PTC) or thiourea, which shows polymorphism at population level. Ability to taste PTC and related compounds is a well known example of polymorphic trait in human populations. Thiocarbamide in general are active goitrogenic substances (Greene, 1974). The ability to taste depends upon the level of di-idio-tryosene in saliva (Fisher and Griffin, 1965). To some people PTC has a distinctly bitter taste while to others it seems tasteless. Among tasters, some find the substance more bitter than others, some perceive only at the tip of the tongue, root of the tongue and so on. Fox (1931), Blakeslee and Salmon (1931) studied the problem of inheritance of the character and independently came to conclusion that taste variations were controlled by a single pair of autosomal genes and that the taster gene was completely dominant over the recessive allele.

Geneticists are in general agreement that it is controlled by a pair of alleles at one locus, and the ability to taste is mainly due to the dominant form of the gene. Blakeslee and Salmon (1931) and Synder (1955) showed that the ability to taste PTC was a dominant Mendelian character. Most investigators (Kalmus, 1957; Das, 1956) have found that females are more sensitive to the taste of PTC than males and can detect bitter taste in lower concentration than males (Sharma, 1967).

The PTC tasting ability is dependent not only on hereditary factors, but it is also influenced by environment. Considerable racial differences in the tasting sensitivity to PTC have been observed. It has been used in studies of relationship to several diseases, especially those of thyroid
gland (Boyd, 1950). Population geneticists with the help of this factor resolve two major problems, first it gives a chance for the study of genetic mechanism and secondly it also explains some of anomalies arising out of these complex hereditary purposes.

A number of studies have shown an excess of non-tasters among individual having adenomatous goitre (Harris et al, 1949; Kitchin et al, 1959; Azevado et al, 1965) and in addition to this, it was found that athyrotic cretins were significantly more likely to be non-tasters than normal controls (Sheppard and Gartler, 1960; Frazer, 1961). Non-tasters have also been shown to exhibit more susceptibility to dental caries (Chung et al, 1964), diabetes mellitus (Terry and Segall, 1947; Terry, 1960) and Saldanha (1956).

**Dermatoglyphics**

Dermal ridge configurations are inherited as a complex and polygenic trait. Inheritance of complex characters like these cannot satisfactorily be studied by conventional methods of pedigree analysis (Penrose, 1963; Pons, 1964; Weninger, 1965 and Mukherjee, 1966) and needs special techniques. Such characters are controlled by environment and their interaction. The dermatoglyphics include the analysis of the ridge configurations of the skin of the fingers, palms, soles and toes. It is a collective name for all these integumentary features and applies also to the division of anatomy which includes their study. These ridges are studded with sweat gland, but lack hair and sebaceous glands. The microscopic dermal ridges serve as friction pads providing better grip and grasp and also enhance tactile acuity. A detailed examination of these prints in thousands of individuals reveals that no two prints are
identical. These are usually formed in the human foetus in the thirteenth
week. After they are formed they do not change throughout one's
lifetime, unless they are permanently damaged beyond repairs,
by burning or cutting very deep. In case of superficial damage to the
epidermis, the prints are restored on the hand and foot regions as the
new skin develops. Abundant evidence has now proved that genetic
factors are operative in the inheritance of dermatoglyphic traits (Holt,
1968; Reed et al, 1975).

These dermal ridge configurations on fingers, palms and soles
must have aroused interest long ago, though when it was that men first
noticed them can never be known. There exist records that indicate
acquaintance with these patterns, long prior to the period of scientific
study.

In the normal population the major source of variation is genetical,
and racial and sex differences are well documented. Abnormal
dermatoglyphics findings have been reported in a number of
pathological conditions. Cummins and Midlo (1943), in a review of the
literature, found mention of epilepsy, schizophrenia, mongolism, mental
deficiency, neurofibromatosis, psoriasis, and congenital abnormalities
such as polydactyly and spine bifida. The development of
dermatoglyphic traits possibly occurs at a much earlier embryonic stage
than their microscopically observed morphological signs. Many authors
have attempted to correlate dermatoglyphics with disease. Cummins and
Midlo (1961) reported that morphological varieties of skin in pattern
types were distorted or damaged with the effect of certain diseases.
The role of dermatoglyphics in medicine and its correlation with
heritable is new and has opened new diagnostic avenues. Attempts have
been made by various researchers to correlate finger ball
dermatoglyphics with certain diseases, e.g., Phenylketonuria (Hirsch,
1965), Leukemia (Aleksandrawicz et al, 1966), Diabetes (Chakravorti,
1967), Cancer (Chorlton, 1970), Thalassaemia (Kumar et al, 1971; Kapoor
et al, 1998), Asthma (Bansal and Bector, 1975; Bhatnagar et al, 1983),
Pulmonary tuberculosis (Sidhu et al, 1977; Bhatnagar, 1980).
Chromosomal disorders (Hale et al, 1961), Fallot’s tetralogy (Bulsee et
al, 1983; Cascos, 1965), Congenital heart disease (Hale et al, 1961),
Schizophrenia (Moller, 1968; Eswaraiah, 1978), Wilson’s disease (Hodges
and Simon, 1962), Blind (Fatima et al, 1983; Jindal and Bansal, 1983),
Cataracts (Padma and Murthy, 1980), Manic depressive psychosis
(Narayanan and Mallikarjunaiah, 1983), Epilepsy (Shobha et al, 1994;

Moreover, dermatoglyphic features are known to be diagnostic aid
in various diseases conditions (Holt, 1968; Schaumann and Alter, 1976).
Numerous associations found between various genetic markers and
susceptibility to particular diseases besides throwing new light on the
mechanisms of man’s evolution are of substantial prognostic significance
and will probably influence treatment in time.

A study of these associations is important in understanding the
mechanism of disease resistance. The present study was undertaken with
the following objectives:

1. To study genetic variation for PTC, ABO blood groups, ABH
secretions and dermatoglyphic patterns of palms and finger balls
in epileptic patients and controls.
2. To investigate the role of the genetic markers in understanding the aetiology of disease and to investigate the heterogeneity of disease, if any.

3. To document certain myths / beliefs associated with epilepsy prevalent in the region.