CHAPTER II

REVIEW OF LITERATURE
REVIEW OF LITERATURE

This chapter summarizes major contributions of significant studies and articles in dermatoglyphic study. It evaluates current knowledge, pointing out major methodological flaws or gaps in research, inconsistencies in theory and findings.

Brief history of dermatoglyphics

The patterned traceries of fine ridges on fingers, palms and soles have aroused interest long ago. There exist records that indicate acquaintance with these traceries on dermatoglyphics, long prior to the period of scientific study. One of the most telling fragments of this unwritten history is one aboriginal Indian carving found at the edge of Kejimkujik Lake in Nova Scotia. Within the outline of a human hand, scratched in stone, are lines roughly representing dermatoglyphics and flexion creases. The thumb bears a spiral whorl. This petroglyph is generally credited with an age of at least several hundreds of years. Its significance lies in the fact that the maker, though living under primitive conditions, has become familiar with dermatoglyphics and flexion creases and was inspired to engrave a picture of them (Cummins and Midlo, 1961).
Pottery making people even in remote time showed an interest in finger prints. Such prints are often conspicuous and the patterns may be more compelling of attention in the clay than on the skin. The fragment of a clay lamp with a clear finger print (dating back to the fourth or fifth century of the Christian era) was excavated in Palestine by the late Dr. Bade. Numerous objects recovered at the site bear prints of the same potter. Thumb prints found on contracts over two thousand years ago show that the Chinese have long used finger prints for signature and identification. Finger prints along with palm prints known as “Panja” were used for some centuries in India. Since 17th century this field has been explored by Western workers in a planned scientific way.

The study of dermatoglyphics has fascinated researchers since the first work done by Purkinje in 1823. Finger prints as a device for personal identification was introduced in a district in India in the 1870s by Sir William Herschel. In 1880, Herschel and, independently, Henry Faulds brought them to public attention in England as a potential method for identifying criminals. The use of fingerprints acquired a scientific basis with the work of Francis Galton. Galton identified and studied the basic issues that must be addressed in order that fingerprints can be an efficient and reliable method of criminal
identification. Someone who has not looked closely at a finger print might suppose that identification would be accomplished by a subjective evaluation of the gross pattern type (arch, loop or whorl). But while these gross features were indeed useful for rough classification, Galton stressed that identification was accomplished precisely only through attention to the minutiae of the prints – tiny islets and forks in the ridges.

From examples furnished by Herschel and others, Galton was able to establish that human finger prints were remarkably stable from early youth to advanced age, even to after death. Galton devised taxonomic methods starting with a set of basic patterns, a method that permitted pigeonhole storage in a way that survived to the computer age (Stigler, 1995).

Galton and Wilder were the pioneers who studied the hereditary basis of dermal pattern. It is agreed that dermatoglyphic features are polygenic or muti-factorial in inheritance along Mendelian lines. Once they get established, they remain permanent in the entire life of the individual. The ridge patterns formed before birth never changes in life and after death they may change as a result of decomposition. Though a temporary disfigurement may be brought
about by cuts, burns and skin diseases, the ridges resume their original appearance.

The study of dermatoglyphics gained prominence with the work of Harold Cummins. The word “dermatoglyphics” was coined in 1926 to describe what until then had been referred to as epidermal ridge configurations. Until Cummins’ pioneering work in the study of this particular human variation, little was known or understood of the embryology of dermatoglyphics, its population wise variability or its significant associations with chromosomal aberrations and other congenital defects. Cummins explored every area of dermatoglyphics with thoroughness.

Simultaneously with finger printing for identification, anatomists, physical anthropologists and geneticists collected data which are the basis for development of this field. Medical interest in dermatoglyphics began in 1943. Distortion of the dermal pattern was first recognized in Mongolism by Harold Cummins, 20 years before the condition was recognized as being due to chromosomal aberration. This stimulated many workers to investigate other cases of dermatoglyphic patterns.
Dermatoglyphics have been analysed in several groups of conditions including single gene disorders and syndromes of unknown cause. Trisomy 21, 18 and 13 are syndromes in which diagnosis can be made frequently on the basis of the prints alone. But in trisomy 8 or Ullrich-Turner syndrome prints alone are not diagnostic. Dermatoglyphic markers have been reported in several diseases like blood pressure (Reed, 1995) Diabetes mellitus (Ziegler et al 1993) Alzheimer’s disease (Weinreb, 1986) Epilepsy (Nair, 1996 and Mondal, 2000) congenital adrenal hyperplasia (Borger et al 1986) congenital heart defects (Nair, 1986) etc. Cytogenetic and dermatoglyphic studies were performed on a group of 197 institutionalized patients with severe mental and physical handicaps in order to evaluate contribution of chromosomal aberrations on the etiology of the condition by Kodama (1982). Significant differences were observed in several dermatoglyphic characteristics including Simian crease, finger tip pattern, mean a-b ridge count, hypothenar pattern and hallucal pattern. When such studies were made, several overlapping were observed. A specific pattern itself cannot identify any anomaly.

Over the last 70 years, dermatoglyphics have been extensively studied in a wide variety of conditions. A convincing body
of evidence now exists that altered dermatoglyphic profiles are associated with a number of environmental congenital disorders, such as congenital rubella and foetal alcohol syndrome. Dermatoglyphics may be viewed as 'fossils' of late first and second trimester foetal development. Because both the brain and dermatoglyphic features are derived from the ectoderm, there is a rationale for using dermatoglyphics as indirect markers of developmental disturbances occurring during the first half of pregnancy.

Fluctuating asymmetry (FA) is an indicator of developmental homeostasis. Developmental homeostasis is the capacity of an individual genotype to produce a proper well formed phenotype in the face of insults that occur in the course of development. Developmental stability of various animal species, including man has been assessed by the magnitude of FA in bilateral morphological traits. Severe stress at some critical stage of development can produce abnormal phenotypes. Increased FA implies disturbances in developmental homeostasis at the molecular, chromosomal and epigenetic levels according to Parsons (1992).

In lower animals, FA of sternopleural bristle number in *Drosophila melanogaster* grown from first instar larvae was
substantially greater at 30° C than at 25° C. At 30° C, which is close to lethality, variability was high. In addition, a number of severely abnormal flies formed at 30° C, confirmed the poor developmental homeostasis of flies at this temperature, as reported by Parsons (1992).

In two species of sand lizard Lacerta, egg clutches were taken from females from natural population and the embryos were developed at constant temperatures, 20, 25, 30 and 35° C. Variation of scale characters assessed by FA and the mean numbers of scale disturbances indicated maximum developmental stability at 25° C. FA based upon mandibular and maxillary first molars of Wistar rats increased under cold stress, audiogenic stress and protein deprivation, especially when two of the three stresses were combined together. These examples show that FA is useful for monitoring environmental stress.

When nucleic acid molecules are incorporated into cells in the process of forming transgenic organisms, the extra macromolecules imply an energetic cost and a general destabilization of metabolic processes. This is a situation of genomic stress. Transgenic pigs with high growth rates have simultaneously a high incidence of gastric ulcers, arthritis, cardiomegaly, dermatitis and
renal disease. FA in adults is a trait which reflects the total environment during development and which can monitor the disruption of developmental homeostasis. Both recombination data and observations on stress sensitive mutants suggest that detailed FA studies on the effects of combinations of environmental and genomic stresses would be useful for environmental monitoring.

Any major genetic perturbation can be stressed at the genome level by disrupting normal physiological processes, thus destabilizing metabolic excesses and unforeseen phenotypic effects occur. In humans, increased dental FA was found in individuals with Down syndrome (Townsend, 1983). Thus when the total chromosome number is upset, the FA has increased in Down syndrome.

The findings of increased FA in cases of familial congenital malformations and genetic disorders suggest a defective stabilization that resulted in the defect together with an increased FA according to Livshits and Kobyliansky (1987). Increased dermatoglyphic asymmetry can be related to a non-specific distortion at an early stage of embryonic development (Pechenkina et al, 2000)
THE PRESENT DERMATOGLYPHIC STUDY

The study included five disability groups. The disability groups studied include children with autism, children with Cerebral palsy, children with deafness and dumbness, children with Down syndrome and children with learning disability. The investigator intends to give a brief description of each disability group and critically evaluate the available dermatoglyphic studies relating to each disability group.

The inability to carry out certain activities is termed disability. A disability has been defined as “any restriction or lack of ability to perform an activity in the manner or within the range considered normal for a human being. According to census 2001 the major disability group—mental retardation is 21 crores and in Kerala, it is about 8 lakhs. The disability groups thus become a liability for society.

Autism

Autism is a life long developmental disability that prevents individuals from properly understanding what they see, hear and otherwise sense. This results in severe problem of social
relationships, communication and behavior. Individuals with autism have to painstakingly learn normal patterns of speech and communication and appropriate ways to relate to people, objects and events in a similar manner to those who have had a stroke.

Autism has been mystifying scientists for more than half a century. Complex behavioral disorder shows a wide variety of symptoms, most of which usually appear before a child turns three. Till recently, in most cases, detection and diagnosis came about only around the time the child went to school and found it difficult to cope with normal teaching methods.

At least 16 of every 10,000 babies are born with autism or one of its related disorders. Ever since autism was first identified in 1943 by Leo Kanner, scientists have made several studies in autism. Autism is found throughout the world, among all races, nationalities and social class. Boys are affected more often than girls in the ratio of 4:1 (Stopford, 1987). By examining the inheritance of the disorder researchers have shown that autism runs in families, though not in a clear-cut way. Siblings of people with autism have a 3 to 8 percent chance of being diagnosed with the same disorder. This is much greater than the 0.16% risk in the general population. Studies of twins
in the UK confirm that autism has a heritable component but suggest that environmental influences play a role as well. In monozygotic twins, who share the same genes, when one twin has autism, the second twin has only a 60% chance of being diagnosed with the same disorder. This indicates that other factors must modify the genetic pre-disposition to the disorder. Several environmental risk factors are already known. *In utero* exposure to rubella or to birth defect causing substances such as ethanol and valproic acid increase the chance that autism will develop. People with certain genetic diseases like Phenyl Ketonuria also have a greater chance of developing autism. About 5% of the thalidomide victims had autism, which is about 30 times higher than the rate among general population. The study of thalidomide victims with autism showed that many cases of autism are initiated very early in gestation (Rodier, 2000).

Elevated levels of fluctuating asymmetry have been associated with autism (Martin and Manning, 1999). A child with autism has a few physical anomalies characteristic of the disorder. The corners of the mouth are low compared with the centre of upper lip, tops of ears flop over, ears are a bit lower than normal and have an almost square shape. Along with minor physical anomalies, neurological anomalies are also reported (Rodier, 2000). The nerve
dysfunction in people with autism reflect nearly a brain injury that not only affects the cranial nerves, but has secondary effects on later brain development.

In a study of autistic children, 12.5% showed fragile X etiology for autism (Chetan, 1995). Wing (1980) has suggested that infantile autism is congenital in approximately 80% of the cases. Dermatoglyphics associated with congenital defects are significant markers of pre-natal events. Dermatoglyphics formed at approximately the 10th week of embryonic development (Penrose and Ohara, 1973) and are therefore probably most sensitive to environmental and genomic stresses acting in the early gestational period (Naugler and Ludman, 1996 b). Since both genetic and environmental factors are reported to cause autism, the present study included an analysis of the relationship between dermatoglyphic patterns and autistic syndrome. Aberrant dermatoglyphic patterns could represent a subset of minor physical anomalies, from early intra-uterine development and detectable in early childhood, that would serve as markers in the diagnosis of autism.

A study of dermatoglyphic analysis of autistic children showed a significantly higher number of arches and whorls and a
significantly lower number of ulnar loops (Riveria, 1981). In a study of dermatoglyphics of Autistic Basque children by Arrieta et al. (1990), significant differences was observed between the digital dermatoglyphics of autistic boys and the control group. There was no significant difference between autistic girls and control group. Autistic children of both sexes had a higher frequency of aberrant palmar creases. When a discriminant analysis of the same study was conducted by Arietta et al. later in 1993, the results showed that the discriminant function obtained seems to fit better in females than in males. The researchers also found that ridge hypoplasia and ridge dissociation are two types of imperfect ridge formation that arise as a result of disturbances in embryonic development. Congenitally imperfect ridge formation, ridge hypoplasia and ridge dissociation was studied in a sample of autistic children and control group. The study showed that the incidence of these anomalies is higher in girls than in boys.

Studies in the 1960s and early 1970s were skeptical about any substantial role for genetic factors in the etiology of autism (Rutter, 2000). Evidences from studies of twins (Wahlstrom et al. 1989) with autistic disorder have shown a higher concordance in monozygotic than dizygotic pairs. Evidence from both twin and
family studies indicate strong genetic influences and the likelihood that they applied to a phenotype that was much broader than the traditional diagnostic category of autism (Rutter, 2000). Medical and chromosomal findings also indicated genetic heterogeneity.

Present study aims at identifying any dermatoglyphic marker for autism. The investigator hopes that autism could be identified at a very early stage, using a non-invasive technique like dermatoglyphics, and early treatment would have a better chance of shaping brain development and might lead to a better outcome.

Cerebral palsy

Cerebral palsy is a disorder of movement and posture appearing in the early years of life. It is caused by damage to or lack of development in a small part of the brain controlling movement and posture. It has been described as a lively mind in a disobedient body.

The term Cerebral palsy covers a wide range of types and severity of disability. Some people are so mildly affected that there may be no obvious disability; others may be very seriously handicapped. Often the damage to or lack of development in the areas of the brain causing Cerebral palsy also affects other parts of the brain, resulting in other types of disability such as mental handicap,
visual impairment and loss of sensation. There is often lack of control of tongue and lips, visual and perceptual disorders, loss of tactile discrimination, spatial disorders and seizures as well as abnormal behavioral patterns. The incidence of Cerebral palsy is said to be between 2 to 2.5 percent per 1000 birth, with no distinctions of sex, race, maternal age or social background (Stopford, 1987).

Anything that can cause brain damage before, during or soon after birth can cause Cerebral palsy. Before birth, maternal infections, chronic diseases, physical trauma or maternal exposure to toxic substances or X-rays may damage the brain of the foetus. During the birth process the brain may be injured, especially if labour or birth is difficult or complicated. Premature birth, anoxia, high fever, infections, poisoning, hemorrhaging and related factors may cause harm to the brain following birth. In short, anything that results in oxygen deprivation, poisoning, cerebral bleeding or direct trauma to the brain can be a possible cause of Cerebral palsy (Hallahan and Kauffman, 1978).

Cerebral palsy is generally not a hereditary condition and it is unusual for 2 cases to occur in the same family (Stopford, 1987). Cases in which genetic factors cause Cerebral palsy are very rare. In
same cases of genetically determined biochemical disorders associated with mental retardation, the child may show evidence of brain damage or Cerebral palsy. Study by Simsek et al. (1998) suggest congenital and prenatal factors to have a role in more than 50% of Cerebral palsy cases.

Dermatoglyphic analysis might be useful to explain the occurrence of Cerebral palsy at different stages of gestation. Studies in this subject are scanty. Diagnostic values of dermatoglyphic features of patients with Cerebral palsy was studied by Simsek et al. (1998) and compared with control. It was found that arch, radial loop and whorl prints have increased and ulnar print has decreased in boys investigated which was significant. No difference was found between girls with Cerebral palsy and control group. No clear distinction occurred between the two groups with respect to palmar flexion lines. There was a notable decrease in the counts of a-b ridges of the investigation group as compared to those of the control group. It was significant in boys but not in girls. In Cerebral palsy, remarkable differences in comparison with controls were found in dermatoglyphic features (Simsek et al., 1998).
A study of asymmetry in Cerebral palsy has not been reported so far. The role played by genetic factors in the etiology of Cerebral palsy is limited. A dermatoglyphic asymmetry, if shown, can indicate the period in which the change has occurred. If a dermatoglyphic patterns typical for Cerebral palsy is obtained, it can lead to early and accurate detection. Usually the diagnosis is not made until the child is at least one year old and the cause is very often not identified. Treatment at an earlier age is more successful and deformities of the limbs can be prevented.

**Deaf and Dumb**

The term deaf and dumb is of common usage in the human society. Generally a deaf person is also mute because he fails to learn speech in his childhood.

The deaf and mute persons may be divided into 2 main divisions.

a) Those who are born or have become deaf at such an early age that speech has not developed naturally or has not got much time to be established.

b) Those who have become deaf in later years after acquiring speech and languages in a normal manner and those with partial impairment of hearing capacity.
The causes of deafness are various in nature. Some of these causes are closely related to the causes for blindness, such as small pox and veneral diseases. Records of different hospitals in India reveal that the diseases like malaria, mumps, septic tonsils, otitis media, chronic rhinitis, adenoids etc are the common causes for deafness. Accurate data about the oral and auditory handicapped in India is not available (Ray, 1984).

In view of the complexity of the hearing mechanism it is not surprising that deafness can result from a wide variety of genetically determined abnormalities. Inheri'ed deafness which is manifested in infancy has profound consequences for the affected child and the family. In the absence of hearing, normal speech cannot develop and even after years of special schooling, vocal communication is still impaired.

Deaf children have diminished contact with other people and outside world. The range of information which they acquire is limited. A severe hearing defect drastically impedes communication with others and places stress upon interpersonal relationships, social isolation, unhappiness and depression often results. So hearing
disability is often associated with diminished academic achievement (Beighton, 1983).

Inherited deafness is usually present at birth, but some types of deafness become manifested and progress later in life. Information concerning a significant causative agent is not always forthcoming. Many of the children have unrecognized genetic deafness. The prenatal causes of inherited deafness are infections (rubella) and maternal drug therapy (quinine, thalidomide etc). Deafness is also a major component of well over 100 Mendelian syndromes.

Dermatoglyphic studies conducted in children with deafness and dumbness are only few. Ingole and Shah (2004) made a study of dermatoglyphics in congenital deaf and mute. The study reported an asymmetry of 'atd' angle of a greater degree in affected group as compared to control group. Borate et al (2004) reported a study of palmar dermatoglyphics in congenital deaf. The study discusses the possibility of using dermatoglyphic parameters as a predictive anatomic tool to diagnose the disease at an earlier age. A related study was reported from Bulgaria by Tornjova and Randelova (1994). They made a comparative study of normal Bulgarian children
aged 3 to 18 years and children with visual, auditory and mental insufficiency. The relative frequency of pattern types of the digits differed in the three groups. The differences were particularly noticeable on the 2\textsuperscript{nd} and 4\textsuperscript{th} digits.

A dermatoglyphic profile was carried out in 108 cases of congenital profound Sensori-neural Hearing Loss (SNHL) with delayed development of speech and language. The study was reported from India (Chaturvedi and Kumar, 1993). The study showed that no pattern existed in a constant fashion to identify children with hearing loss.

The aim of present study is to see whether the dermatoglyphics in deaf and dumb children differ from that of the control group. The study also includes the FA of finger pattern and a-b ridge difference.

**Down syndrome**

Chromosomal anomalies are one of the major causes of mental retardation. Human congenital anomalies are many; however, Down syndrome (DS) is the commonest. It occurs in all social and ethnic groups all over the World. The first detailed and systematic description was done by Langdon Down in 1866. All people with DS
are not identical even with respect to the extra genetic material. The DS frequency is one in 650-1000 live births (Roy, 1998). The round head, open mouth, stubby hands, slanting palpebral fissures and short stature creates an unforgettable clinical picture.

In the majority of cases with Down syndrome, the abnormality is due to non-disjunction which produces an extra-chromosome on 21 pair. It is therefore called trisomy 21. In a very small proportion (2%) of persons with Down syndrome, the abnormality of chromosome is due to translocation (because the chromosome material has transferred its location). The total chromosome number in such cases is 46. If non-disjunction occurs after the first cell division, only part of the cells will have 47 chromosomes and the condition is called mosaicism.

A cytogenetic study of Down syndrome subjects and their parents in ICMR, Bombay revealed 86% pure trisomy 21, 6.67% mosaic trisomy 21 and 6.67% translocation trisomy 21. Parental chromosomal analysis showed normal karyotypes. Origin of extra chromosome was detected as maternal in 75.3% and paternal in 24.57% using chromosomal polymorphism study with GTG banding (Seema et al., 2002).
Among the newborns 0.3% of live births are aneuploid with the most common abnormalities being trisomy 21 and sex chromosome trisomy, with an increasing incidence in still births occurring at 20 weeks of gestation and term. When a cytogenetic study of 345 mentally retarded patients with major and minor malformations attending the genetic clinics at Manipal Hospital was evaluated, chromosome abnormality was observed in 29% of which 22% case had free trisomy 21 (Divya et al., 2002). In another study at J.J. Hospital, Bombay, out of 269 cases of Down syndrome anomaly, 77% cases were having free trisomy 21 with a male to female ratio of 5.6:4.4. Eight (8) % cases were of mosaic Downs' and 3.3 % were of translocation.

The incidence of Down syndrome rises dramatically with maternal age (Erickson, 1979). About 40% of Down syndrome victims are born to women over 40 years of age. The greater incidence among older mothers may be associated with the long delay in completion of meiosis. Potential eggs in the ovary begin the meiotic process before birth and proceed to primary oocyte stage. They then remain dormant until shortly before ovulation occurs. Cells of a woman 40 years of age have thus remained in an inactive stage for about twice as long as those of a woman 20 years of age. A period of
forty years may certainly be long enough to permit weakening of the spindle fibers or other structures necessary for the proper disjunction of chromosomes. Also, older female often undergo dramatic hormonal changes with possible side effects on cellular division (Strickberger, 2002). This long period of quiescence may explain at least partially why non-disjunction occurs more frequently in older than in younger mothers.

In a study of association of chromosomal anomalies with age and birth order showed that the incidence of chromosome aberrations is higher when the age and birth order of parents are higher (Roy, 2002). A reduction in maternal age was reported from a study in Hyderabad. The mean maternal age calculated was 25.4±6.37 and mean paternal age was 33.0±6.67 years. The Down syndrome incidence was prevalent in 20-36 years of age group in mother and in fathers it was between 25 to 39 (Rao et al., 2001). A preponderance of younger parents having Down syndrome was also reported by Seema et al. (2002). The possibility of paternal age effect alone is small (Erickson, 1978). An analysis of Down syndrome live births in Sweden 1968-70 by single year maternal age interval was carried out. A gradual increase in rates of Down syndrome with maternal age was
observed up to about age 30 to 31 and steep increase was observed thereafter (Hook and Fabia, 1978).

A study of spontaneous abortions has reported an interesting pattern of association of maternal cigarette smoking and maternal age with trisomy. Smoking was negatively associated with trisomy in younger mothers, those under 30, but positively associated with trisomy in older mothers (Kline et al., 1983). The increased frequency of Down syndrome in women below 35 years of age has been noticed in industrial and densely populated areas (Rao et al., 2001).

Characteristic dermatoglyphic abnormality was recognized in Down syndrome patients many years before the demonstration of chromosomal basis of the disorder (Reed, 1991). Down in 1909 first noted the increased frequency of a single transverse palmar crease (Simian Crease) in Down syndrome. Penrose in 1931 first reported an increase in the occurrence of a single flexion crease of the little finger in Down syndrome. Based on the significant differences in the frequencies of dermal configuration for "Mangoloid embeciles" compared with control, Walker developed the
first practical diagnostic index for Down syndrome patients using only
dermatoglyphic features (Walker, 1958).

The frequencies of the various dermal ridge patterns on
the finger of Mongols differ from those of the general population
(Holt, 1964). Fingerprints show less variation, there are fewer whorls
and contrary to the usual tendency for the frequency of arches to
increase as whorls diminish, fewer arches. There is also a reduction in
the frequency of radial loops, which occur chiefly on digits IV & V,
instead of digit II. The deficiency of all these patterns is compensated
by an increase in ulnar loops which tend to be high and L-shaped. The
findings were first described by Cummins (1939) for an American
sample of Mongols. The same was confirmed by Walker (1958) for a
Canadian sample.

The dermatogram - a dermatoglyphic monogram based
upon 4 pattern areas was compared with 3 other indices for the
diagnosis of Down syndrome - correctly diagnosed 79% of Down
syndrome patients (Reed and Christian, 1976). The dermatogram was
developed for use in a clinical setting as a rapid screening mechanism
for Down syndrome. Data collected from 70 cytogenetically
confirmed Indian DS patients and their parents were compared with
230 controls. Significant differences were noted in the TFRC, abnormal dermatoglyphic features like high axial tri-radius, transverse palmar crease, Sydney line, pattern on hypothenar and inter-digital areas in higher frequencies among DS patients and their parents, than in controls (Rajangam et al., 1991). Mostly ulnar loop pattern was observed in DS patients (Rajangam et al, 1995). Dermatoglyphic analysis carried out has proved that they are invaluable in their clinical value, in selecting patients of DS for cytogenetic studies.

As Down syndrome can occur due to trisomy 21, translocation or mosaicism, dermatoglyphic studies were focused on whether there is any dermatoglyphic variation in these different groups causing DS. In a study by Banerjee et al. (1991) dermatoglyphic features showed significant differences between trisomic Downs, their parents and control population. Non-trisomic Downs also differed from their parents and the control population, but the magnitude of differences has not reached the level of significance. Schmidt et al. (1981) have pointed out the possibility of undetected 21 trisomy mosaicism in both the younger and older mothers as a possible cause of DS progeny. Younger parents with abnormal dermatoglyphic features might have a higher risk of having a DS child, as these parents may be undetected mosaics for trisomy 21 line.
Ayme et al. (1979) while discussing the merits of dermatoglyphic studies in parents of DS children, suggested that changes in the dermatoglyphics may be more pronounced in the parent, who had given the extra chromosome 21. Abnormal dermatoglyphic features have been reported for cytogenetically confirmed Indian Downs and their parents by Rajangam et al. (1991). The proportion of individuals with an increased chance of mosaicism is appreciably greater in a sample of both the mothers and the fathers of 'Mongol' children, as compared with the parents of 'non-Mongol' children (Loesch, 1981). In the cytogenetically diagnosed patients with mosaic DS, a highly significant co-relation was observed between the % of trisomic cells and the presence of traits characteristic of this syndrome in the dermatoglyphic patterns (Rodewald et al., 1980). Dermatoglyphics and the analysis carried out so far prove that they are important in their clinical value, in selecting patients of Down syndrome for cytogenetic analysis and in detecting hidden mosaics in unscreened population. The dual approach using both dermatoglyphic and cytogenetic studies may aid in identifying persons with an enhanced risk of having affected children, and thus assist in counseling these families (Schmidt, 1981).
A comparison of dermatoglyphic features of Down syndrome patients from different racial origins was made to see whether the phenotypic resemblance of Down syndrome patients extended to include their dermatoglyphic characteristics also (Quazi, 1977). In another study of Cuban Mongols, the dermatoglyphic patterns were very similar to those observed in other countries (Borbolla et al., 1980). Similar results are also reported from Jewish Down patients (Katznelson, 1982) from Japanese Down syndrome patients (Shiono and Azumi, 1982). It seems that the peculiar dermatoglyphic patterns of patients with Down syndrome is not affected by ethnic influences (Borbolla et al., 1980).

The typical phenotypic and dermatoglyphic characters of Down syndrome patients all over the World requires explanation. Waddington (1942) postulated that the genotype is buffered in such a way that development is canalized and ordinarily proceeds along evolved developmental tracks. Developmental pathways vary in their stability depending on the degree of canalization. Some are more readily displaced from the normal phenotype by genetic and environmental trauma than are others (Shapiro, 1975). The extra chromosome in Down syndrome results in a generalized decrease in canalization of development. Morphogenetic hypothesis have been
forwarded by Morris (1991) to explain the dermatoglyphic variations found in Down syndrome. Slight variations of developmental timing in either pad or ridge morphogenesis could account for the dermatoglyphic features of Down syndrome. Certain aspects of developmental timing are under strong genetic influence and individuals with abnormal genotypes which affect general ontogenic processes (as in the presence of extra chromosome 21) might exhibit certain characteristic dermatoglyphic traits (Morris, 1991). Corresponding to the low developmental stability, fluctuating asymmetry has been elevated in Down syndrome patients compared with fluctuating asymmetry in healthy controls (Katznelson et al., 1999).

Several reports on dermatoglyphics of Down syndrome victims are from abroad. Reported cases from India are only a few. A study of dermatoglyphics of Down syndrome cases from Kerala is not available. The present study includes dermatoglyphic features of Down syndrome, a study of relationship between Down syndrome and maternal age and a special focus on fluctuating asymmetry.
**Learning Disability**

The term "Learning Disability" indicates limited ability in learning. It was in a parents meeting as early as in 1963 in New York City that Samuel Kirk proposed this term as a compromise in the face of the confusing variety of labels that were then being used to describe the child with a particular type of learning problem. Over the years, a variety of labels have been used to describe the reading disabilities including "dyslexia", "reading backward" "learning disability" etc. The term "dyslexia" was used extensively for many years to describe a reading disability syndrome that often included speech and language deficits and right and left confusion. When it became evident that reading disorder is frequently accompanied by disabilities in other academic skills, the use of the term "dyslexia" diminished, and general term such as "learning disorder" began to be used.

The Federal Government of USA has defined Learning Disabilities in Public Law 94-142 as follows "Specific Learning Disability means a disorder in one more of basic psychological processes involved in understanding or in using language, spoken or written, which may manifest itself as an imperfect ability to listen,
think, speak, read, write, spell or to do mathematical calculations.”
The term does not include children who have learning problems which
are primarily the result of visual, hearing or motor handicaps, of
mental retardation, of emotional disturbances or of environmental,
cultural or economic disadvantages (Hallahan & Kauffman, 1978).

Each child, adolescent or adult with a Learning disability
is unique and each shows different combination or severity of
problems. A person with learning disability is an individual who has
one or more deficits in the essential learning process (Suresh, 1998).
So children with learning disability is a heterogeneous group having
various degrees of language deficits, perceptual deficits or other
cognitive abnormalities. These disorders are globally present. In
several Western Countries learning disability and developmental
language disorders have been detected quite early and adequate
training and rehabilitation strategies have already been implemented.
In USA, the Federal Government considers them as a special category
like physically handicapped from 1975 onwards.

The picture is different in India. Though child language
disorders have gained recognition among professionals and
educationalists, adequate rehabilitation program and special education
facilities are yet to be devised. The magnitude of the problem and its incidence and prevalence in our country is not yet fully understood. There has been swift development in the educational program over the past 50 years in Kerala. But the success rate in many qualifying examinations like SSLC and Plus Two remains poor. A high failure rate in these examinations may be related to the prevalence of child language disorders and learning disability (Suresh, 1998).

The results of a preliminary survey conducted by the Sree Chitra Tirunal Institute for Medical Sciences and Technology (SCTIMST), Thiruvananthapuram shows that 9.5% of children in the age group of 0 to 12 year have delayed acquisition of speech and language due to some causes (Suresh, 1998). In a clinical study from a community mental health program in primary schools in Kerala, of all the identified problem children, 12% had learning disability (Mathew and Kumar, 1998). Three to four times as many boys as girls are reported to have reading disability among children in schools and clinically referred samples (Kaplan and Sadock, 2000).

At present the identification and diagnosis of learning disabilities is done only after the child experiences considerable amount of failure in academic learning. This would have developed
its own negative consequences like frustration, fear of failure, aversion towards schooling etc. This emphasizes the need for early identification of the learning disabled and providing remedial facilities for them. Pre-primary school stage (3 - 6 years) is more convenient for identifying learning disabled children.

Dermatoglyphic studies on learning disabled are few. In a study by Jamison (1988) Palmar dermatoglyphic prints of 261 dyslexics was compared against 707 controls. Dyslexics of both sexes were found to exhibit greater complexity in terms of ridge count and pattern location than controls, particularly on the left hand. He reported higher a-b ridge count, wider 'atd' angles on both palm and higher frequencies of pattern in left fourth inter-digital area. The study supported the hypothesis that some causative factor relating to the development of dyslexia is operating during the time period in which dermatoglyphic features are formed. Abnormal palmar flexion creases have been reported by Rosa et al (2000) in a group of children with idiopathic intellectual disability.

Naugler and Ludman (1996b) made a study of 49 developmentally delayed children and 51 control group to explore the usefulness of FA as a risk marker. Only 2 dermatoglyphic characters
were studied, the finger print concordance and a-b ridge symmetry. To assess FA as a risk marker, Odds ratios were used in addition to traditional group comparisons (Naugler and Ludman, 1996a). The study showed that children with a finger print concordance of 3 or fewer are over twice as likely to have a diagnosis of developmental delay. Children with an asymmetry of 4 or greater between the a-b ridge counts of left and right hand are approximately twice as likely to have a diagnosis of developmental delay. Jameela et al (2002) reported asymmetry of finger pattern and a-b ridge count in a study of learning disabled children from Ernakulam district.

RELATED STUDIES IN GENERAL

The chromosome study in general mental retardation has shown the prevalence ratio being 2.5 to 5 per thousand and overall incidence of congenital malformation among newborns is about 1.5 to 2 percent (Rao, 1998). Previous studies by several groups have revealed a definite chromosomal association in the subjects with a definite degree of mental retardation. So dermatoglyphic studies have focused on conditions with chromosomal abnormalities.

A genetic disease which was first noticed because of several dermatoglyphic peculiarities was the Down syndrome. In
1939, long before the chromosomal basis of Down syndrome was established, Cummins pointed out characteristic differences in dermatoglyphic features in patients with Down syndrome, compared to the normal population. Dermatoglyphic studies of Down syndrome have been discussed in detail in the earlier section.

It is well known that characteristic dermatoglyphic deviation can occur in patients with specific chromosomal aberrations involving both the autosomes and sex chromosomes.

The association between particular dermatoglyphic features and chromosomal abnormalities has long been recognized (Morris, 1991). The studies regarding chromosomal abnormalities and dermatoglyphics are discussed including conditions like trisomy, aneuploidy, structural changes like additions and deletions.

**Dermatoglyphic studies in chromosomal aberrations**

The dermatoglyphic study of trisomic condition, 21 (Down syndrome) has already been discussed. Another anomaly involving an additional chromosome is Trisomy 18. Patients with trisomy 18 show developmental and mental retardation. The most typical dermatoglyphic finding was a strikingly high frequency of arches on fingertips (Schaumann and Alter, 1976) Arches account for
over 90% of all fingertip patterns of these patients; more than 40 percent has ten digital arches. Fewer than 6 arches is very unusual in full trisomy 18, although it is sometimes observed in mosaic, translocation or partial trisomy 18. Ulnar loops and whorls were found markedly diminished in frequency. Radial loops were only slightly reduced in frequency but tended to be displaced from their usual site in the finger tips so that 62% were found on the thumbs, the remaining ones were usually in the third, fourth and fifth digits. Only very rarely was the radial loop observed on the second digit. A distal axial tri-radius was present in 53 percent of palm with an increase in atd angle. Flexion creases in the palm are often abnormal. About 75% of the individuals show a simian crease, usually bilaterally. A single flexion crease on the fingers, usually the fifth digit, is also characteristic of trisomy 18. Some of these dermatoglyphic characters are similar to that of trisomy 21. Reed (1981) has reported absent C-line more frequently in trisomy 18.

Trisomy 13

Characteristic features of trisomy 13 include microcephaly, microphthalmos, cleft lip and cleft palate, low set malformed ears, polydactyly, mental retardation etc. Arches and radial loops have increased and ulnar loops and whorls have decreased. Reed
(1981) reported radial loops in digit IV and V. The axial tri-radius was usually displaced distally to the centre of the palm which resulted in very wide atd angles. The mean atd angle was higher than that of trisomy 18 and trisomy 21. A marked increase of pattern was observed in the thenar area. Approximately 60% of the palm showed a single transverse flexion crease and a single crease on the fifth digit. In some cases ridge dissociation was also observed (Schaumann & Alter, 1976).

**Trisomy 8 Mosaicism**

Individuals with trisomy 8 sometimes show psychomotor retardation, a large and sometimes deformed head with a prominent forehead, low set malformed ears and bone dysplasia with extra ribs and vertebrae. The dermatoglyphic peculiarities include an increased frequency of arch patterns on finger tips, low TFRC, high palmar pattern intensity and a simian crease. Arches were found to be the most frequent fingertip patterns (Schaumann & Alter, 1976). Reed (1981) has reported deep vertical plantar furrows and deep palmar creases in trisomy & mosaicism.
According to Recd (1981) in trisomy 21, 18 and 13 syndromes, diagnosis can be made frequently on the basis of a print alone.

**Dermatoglyphic studies in sex chromosome aberrations**

Abnormalities of the sex chromosomes do not have as much influence on ridge formation as do autosomal chromosomal aberrations according to Schaumann and Alter (1976). Nevertheless, there are some noteworthy dermatoglyphic features associated with chromosomes defects.

**Turner syndrome**

Turner syndrome is caused by full or partial monosomy of an X chromosome, with or without mosaicism. Persons with Turner syndrome are phenotypic females of short stature with multiple anomalies. The corners of the mouth are depressed, hard palate is narrow, ears are prominent and low set, there is webbing of neck with low posterior hairline. There is a general lack of secondary sexual characters with gonadal dysgenesis.

The TFRC was increased in patients with Turner syndrome. Finger patterns are generally similar to those of the control group. The a-b ridge count showed an increase. A hypothenar pattern,
often large, was found in about 50 percent on the palm. Either a full or transitional single crease was found in 28 percent of the palm investigated according to Schaumann and Alter (1976). A missing C-line tri-radius and distally displaced axial tri-radius with high atd angle were other observed dermatoglyphic features.

Klienfelter syndrome

The clinical features of the XXY phenotype become apparent only after puberty and include frequent gynecomastia, sparse facial hair and disturbances of sexual function.

Dermatoglyphic characteristics of individuals with a 47, XXY chromosomal constitution are not very remarkable. The most distinctive feature was a lower TFRC. A reduced a–b ridge count was also reported. Other differences were not observed in this syndrome.

Dermatoglyphic studies in structural chromosomal aberrations

Structural chromosomal aberrations occur due to deletion or duplication of chromosome arm. A deletion of the short arm of chromosome 5 causes the Cri-du-chat syndrome. Typically, persons show profound mental retardation, microcephaly, oblique palpebral fissures, epicanthal folds, low-set ears and micrognathia. According to Schaumann and Alter (1976) apart from increased frequency of
whorls and a decrease of ulnar loops, no striking pattern abnormalities were found on the fingertip. The mean TFRC decreased in male patients, while it increased among the females. A single transverse flexion palmar crease was found in 82% of all the palm analyzed. 74% of all the simian creases were listed as true simian lines.

Dermatoglyphic analysis in the case of persons with deletion of the short arm of chromosome 4 also revealed several unusual traits. An increase in arch configuration, low TFRC, distally placed axial tri-radius with high 'atd' angle, an increase in thenar pattern, a single transverse palmar flexion crease, all these were present in this category. (Schaumann and Alter, 1976).

Similar dermatoglyphic studies were conducted in persons with deletion of the short arm of the chromosome 18, long arm of chromosome 18, ring chromosome 18 etc. Dermatoglyphic variations were observed in these studies also. Here prints alone are not diagnostic. If the dermatoglyphic findings are in agreement with the clinical impression, the diagnosis is highly probable.

Dermatoglyphic studies have been reported in single gene disorders like de Lang syndrome. Dermatoglyphics were studied in cleft lip patients, sibs and parents in relation to dermatoglyphic
asymmetry (Woolf and Gianas, 1976). They observed significant
differences in dermatoglyphic asymmetry. Abnormal dermatoglyphics
have been reported in children with proved rubella embryopathy
(Schaumann and Alter, 1976). This suggests that dermatoglyphics
may be a sensitive indicator of even subtle intra uterine rubella
damage. Alter and Schulenberg (1966) noticed an increased frequency
of transitional and full simian creases among patients with rubella
embryopathy. They also observed several additional unusual
dermatoglyphic traits, such as an increased frequency of whorls, a
decreased a-b ridge count, an increased ‘atd’ angle and more patterns
in the inter-digital area. This observation has been confirmed in other
studies by Purvis-Smith and Menser (1973). One cannot diagnose any
of these conditions by prints alone, but knowledge of dermatoglyphics
may help.

Nair (1996) made a study of dermatoglyphic patterns,
handedness and hand clasping of 108 epileptic persons. The study
found an association of dermatoglyphics with epilepsy. Another group
with several dermatoglyphic alterations reported is in schizophrenia.
Earlier studies by several groups reported certain deviations of
dermatoglyphic profile of persons with schizophrenia when compared
to control group.
Fananas et al (1990) reported significantly lower TFRC and a–b ridge count in a sample of schizophrenia patients as compared to control group.

Structural brain abnormalities and congenital malformations are more common in persons with schizophrenia than in controls. This suggests that schizophrenia is partially due to impaired neurodevelopment, that might be accounted for by both environmental and genetic factors (van Oel et al, 2001). Studies using dermatoglyphic indices, which strongly reflect intra-uterine environmental influences, reported higher levels in schizophrenia patients as compared to controls. Davis and Bracha, (1996); Markow and Wandler, (1986) and van Oel et al (2001) investigated the differences in both digital and palmar dermatoglyphic indices between twins discordant for schizophrenia and control twins. Non-genetic intra-uterine circumstances early in pregnancy (10 to 13 weeks of gestation) are associated with a susceptibility to schizophrenia, since both the twins with schizophrenia and the unaffected co-twins showed more fluctuating asymmetry of the finger ridges and marginally higher finger ridge counts than control twin pairs. Fluctuating asymmetry was as important as whole brain and left hippocampal volumes in
differentiating twins with a high susceptibility to schizophrenia from those with a low susceptibility.

Dermatoglyphic studies related to schizophrenia became more important when fluctuating asymmetry was used to identify persons with increased FA. Markow and Wandler (1986) examined dermatoglyphics to test the specific hypothesis that genetic liability to schizophrenia is associated with elevated FA. Using a–b ridge count they found that schizophrenia patients showed greater FA than normal controls. Furthermore, patients with earlier onset of schizophrenia showed greater asymmetry than later onset patients providing additional support for increased polygenic liability in early onset patients. The study suggested that the genetic predisposition for schizophrenia, in the presence of various environmental stressors may develop schizophrenia.

Two congenital dermatoglyphic malformations - ridge dissociation and abnormal features and two metric dermatoglyphic traits – TFRC and a–b RC – were assessed in a sample of patients with chronic DSM – III bipolar illness by Gutierrez et al. (1998). Bipolar cases showed a significant excess of ridge dissociation and abnormal features when compared with controls. No difference was found for
TFRC and a-b RC count. The study suggested that early developmental disruption as suggested by ridge dissociation and abnormal features is a risk factor for later bipolar disorder.

Dementia of the Alzheimer type is a degenerative neurological disorder. Weinreb (1986) made a study of dermatoglyphic patterns in Alzheimer disease. Selected dermatoglyphic variables were analyzed in 50 patients with Alzheimer disease with 100 control subjects. Patients had a significantly increased frequency of ulnar loops on the finger tips, Simian creases on the palms and palmar hypothenar patterns. A trend involving an increased frequency of radial loops on the fourth and fifth digits, and Sydney lines on the palms was also observed. The study proved that the presence of eight or more ulnar loops or bilateral hypothenar patterns separates Alzheimer patients from controls with 84% sensitivity and 63% specificity. This supports the discriminant value of dermatoglyphics in the potential identification of asymptomatic persons at increased risk for Alzheimer disease by dermatoglyphic criteria.

The association of dermatoglyphics with different types of congenital heart defects (CHD) has been studied by Nair (1986).
Congenital heart defects are a heterogeneous group of diseases in whose etiology importance of genetic as well as environmental factors have been recognized. A heterogeneity was seen in the distribution of finger tip patterns in the different categories of CHD. From the pattern of association and the estimate of genetic distance it was inferred that some common genes may be involved in the etiology of CHD and that, genetic differences may be responsible for determining the type of malformation. According to Nair (1986) the less frequent malformation could perhaps be due to greater stability against environmental fluctuation and therefore require a higher genetic threshold for clinical manifestation.

Endomyocardial fibrosis is a disease with restricted geographic distribution. It is prevalent in Kerala and the disease has a multifactorial origin. Dermatoglyphics of 63 persons with endomyocardial fibrosis was studied by Nair & Balakrishnan (1987). This gave clear proof of the association of some dermatoglyphic traits with endomyocardial fibrosis. The associations were found to be more significant in males as compared to females. The study suggested the possibility of the disease to occur by the addition effect of a number of genes in combination with the effect of certain environmental factors.
Several anomalies of dermatoglyphics were noticed in chromosomal disorders, and several disorders with multi-factorial origin. So the focus changed from finger and palmar patterns to asymmetry of dermatoglyphic traits. It is possible to use fluctuating asymmetry to predict the appearance of or predisposition to congenital disorders according to Livshits and Kobyliansky (1987).

The relationship between gestational age at birth and FA has been reported for 216 new born infants and their parents using 3 methods of assessing FA on 8 morphometric traits. The highest value for FA was found in extremely preterm infants and the lowest was found in term infants according to Livshits and Kobyliansky (1987). In persons having congenital anomalies, increased FA of various morphological traits has been reported by many investigators. Pertz et al (1988) compared FA for the meriodistal crown diameters of permanent teeth among persons suffering from Fragile X-syndrome and Down syndrome and from clinically healthy individuals. Healthy individuals showed lower dental asymmetry in both the mandible and the maxilla than do affected individuals. The increased FA with respect to a–b ridge count and finger pattern symmetry has already been discussed. Rose et al. (1987) studied dermatoglyphic asymmetry and behavioral discordance of monozygotic twins. They found that
twin pairs asymmetric for palmar a-b ridge count were significantly less similar in performance on many psychological tests, such as the Minnesota Multiphasic Personality Inventory scales. The total variance of these scales tended to be greater among individual twins with extreme left–right asymmetries.

Fluctuating asymmetry is commonly used to evaluate developmental homeostasis. Developmental homeostasis is the capacity of an individual genotype to produce a proper well formed adaptive phenotype in the face of perturbations and insults that occur in the course of development. Disruptions of developmental processes are likely to increase congenital disorders and susceptibility to various diseases. Deleterious genes and chromosomal aberrations thus disrupt the developmental processes.

Recent reports from Kerala are not very promising with respect to health care. 25 to 30 percent infants are born with low birth weight (Indian Express 8.4.05). Even though the infant mortality rate in Kerala is the lowest in the country, the scenario of child and adolescent health is dipping lower.

The low birth weight leads to a series of complications in later life. It is found that many of the adult diseases have their basis on
childhood health condition. Such persons are more prone to cardiac problems, anaemia and hypertension among others. The report also said that the rate of disability among the children is also shooting up.

The dermatoglyphic studies reported from Kerala are few. The present study of dermatoglyphics of different disability groups can throw light upon their peculiarities, it can also help in identifying children with disabilities at an earlier age.