INTRODUCTION

Fingerprints are found in humans and some animals. They are unique to all individuals and remain unchanged over the lifetime. For centuries the features of the hands have fascinated scholars, sages, theologians, doctors and layman alike. Rather through decades of scientific research, the hands have come to be recognized as a powerful tool in the diagnosis of psychological, medical and genetic conditions.

It was in the 1926 that Cummins introduced the term ‘dermatoglyphics’. It is the term applied to the study of the naturally occurring patterns of the surface of the hands and feet. The dermal pattern once formed remains constant throughout life. Dermatoglyphics is considered as the window of congenital abnormalities and is a sensitive indicator of intrauterine anomalies.

The epidermal ridges first appear in the form of localized cell proliferations around the 10th to 11th week of gestation. These ridges form shallow corrugations that project into the superficial layer of the dermis. The number of ridges continue to increase, being formed either between or adjacent to existing ridges. It is during this period of primary ridge formation, that the characteristic patterns are formed. At about 14 weeks, the primary ridge formation ceases and secondary ridges begin to form as sweat gland, and develop along the apices of the primary ridges at uniform intervals. At this time, the epidermal ridges first begin to appear on the volar surfaces. The dermal papillae are reported to develop in the valleys between the ridges on the deep surface of the epidermis around the 24th week. Till then, the morphology of primary and secondary ridges appears as a smooth ridge of tissue and thereafter
AIMS AND OBJECTIVES

1. To record and evaluate the finger print patterns of patients diagnosed with dental caries (study group) and caries free individuals (control group). Total numbers of 1250 individuals were considered for the study and the age group considered was between 5 and 12 years.

2. To observe a prevalent and specific dermatoglyphic patterns in study and control group.

3. To determine a degree of divergence of specific dermatoglyphic patterns among study and control group.

4. To predict the efficacy of dermatoglyphic patterns/imprints in assessing the risk of susceptibility to dental caries in study group.

MATERIALS AND METHODS

Source of data: A case-control study comprised a total number of 1250 cases was obtained from Chennai Corporation School, Vadapalani, Chennai. Data was collected from these 1250 children between the ages of 5 and 12 years with no difference between the sexes. Out of 1250 subjects, 625 subjects were grouped into study group and the remaining 625 subjects were considered as the control group. The study group included children with dental caries in 5 or more teeth based on the DMFT index and control group consisted of normal, healthy children without any dental caries. A4 size plain paper, cotton, stamp pad, soap, gloves, magnifying lens, scale, protractor, micro tip pencil and eraser, oil, case sheets were used as armamentarium (materials used).

Method of collection of data: Considering the ethical issue and confidentiality of fingerprints of patients, the procedure was explained to the parents of the subjects and permission was obtained through written consent forms before recording the fingerprints. Brief case history with clinical examination and DMFT index was recorded. Subject’s hand were cleaned and dried before imprinting. The finger and palmar prints of the subjects were taken using a stamp pad; a thin layer of stamp pad ink was applied to the fingers and palms. An imprint of five fingertips and palm was recorded on an A4 size bond sheet. The same procedure was repeated in relation to the other hand. Prints were dried and studied using a magnifying lens to identify the finger and palm patterns. After taking the imprints of all fingers and palm, ink was removed by using oil, soap and water. The fingertip patterns were analyzed according to the classical method and configurational types were classified according to the topological method.

Evaluation of patterns: The various patterns of fingerprints were analyzed according to the standard guidelines for classification of patterns. The data recorded was entered in Microsoft Excel sheet and applied for statistical analysis. Statistical analysis was performed using nonparametric tests and t-test to compare the dermatoglyphic pattern changes between the study group and the control group and was applied for each variable, to compare the proportions and p-value.

Limitations: The use of stamp pad ink in dermatoglyphic study has got certain disadvantages. The imprint is affected by the amount of pressure exerted while the palm is
recorded. Care must be taken while recording the prints to apply the stamp ink material in adequate amounts. A thin or thick application results in light or dark improper prints.

Results and observations: The data obtained by analyzing the fingerprints of study group and control group were entered in a primary data sheet. The two independent quantitative variables were dermatoglyphic variable (Figs 1A to C) (which included plain loop (PL), double loop (DL), arch with loop (AWL), plain whorl (PW), double whorl (DW), arch with whorl (AWW), plain arch (PA), tented arch (TA), central pocket loop (CPL) and accidental (A). Total number of independent quantitative variables = 10) and teeth with dental caries (criteria: 5 or more teeth in an individual were considered under study group; maximum value was 10 and minimum value was 5).

Descriptive statistics and correlation test was performed to determine the p-value for each variable. This included the analysis of mean, median, standard deviation, minimum and maximum values. N = total number of individuals, study group N = 625, control group N = 625. The mean and the SD of whorl pattern (PW + DW + AWW) in study group is (X ± SD) = 7.55 ± 2.03. The mean and the SD of whorl pattern in control group is (X ± SD) = 0.69 ± 1.22. The mean and the SD of loop pattern (PL + DL + AWL) in study group is (X ± SD) = 2.04 ± 0.76. The mean and the SD of loop pattern in control group is (X ± SD) = 8.45 ± 1.80.

In order to describe the characteristics of the large sample size, we had to record the long series of observations appropriately and systematically organize the results. So tabulation, frequency distribution and percentage of individual dermatoglyphic patterns were performed. Frequencies, percentage, valid percentage and cumulative percentage of dental caries were also done. From descriptive statistical analysis and its comparative study we can conclude that SD of whorl and loop pattern are very low in study group and control group respectively. This suggests that our data collected follows the normal distribution curve.

Prevalent and specific dermatoglyphic patterns in study and control group was assessed with a scatter plot diagram and correlation tests. The analysis of the relationship of two characteristics (bivariables) namely, dental caries and whorl pattern, are represented by a point on a graph. This graph is called scatter plot diagram (Graph 1). The configuration of the points on the graph indicates the nature of relationship. Since these points lie clustered, it suggests a correlation or relationship between variables (dental caries and whorl pattern).

To detect whether these variables (whorl pattern, loop pattern and dental caries) are interdependent or co-vary, that is, whether they vary together, correlation test was performed. Since, our variables were quantitative and continuous variables, coefficient of linear correlation or also called as Pearson correlation—two-tailed test was performed. It was performed in both study and control group between whorl vs dental caries and loop vs dental caries. Table p-value was considered as < 0.05.
Dermatoglyphics in Patients with Dental Caries: A Study on 1250 Individuals

Whorl vs dental caries when N = 1250 (Graph 2); with independent variable: Whorl; and dependent variable: Dental caries, Table 1 showed that 85% correlation existed between whorl and dental caries (p-value of 0.000). Thus, our result shows that there is a significant relationship between whorl pattern and dental caries. Thus, the two variables whorl and dental caries was positively correlated (r = 0.85). Table 1 shows the same result when permutation and combination was done.

Table 1: Correlation between whorl vs dental caries when N = 1250

<table>
<thead>
<tr>
<th>Whorl</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
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<tbody>
<tr>
<td>Whorl</td>
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<td>Dental caries (Total no. teeth)</td>
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Table 2: Correlation between whorl vs dental caries in study group when N = 625

<table>
<thead>
<tr>
<th>Groups</th>
<th>Whorl</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
</tr>
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<tbody>
<tr>
<td>Study</td>
<td>Whorl</td>
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<td>Dental caries (Total no. teeth)</td>
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<tr>
<td>Control</td>
<td>Whorl</td>
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<td>Dental caries (Total no. teeth)</td>
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*Correlation is significant at the 0.01 level (2-tailed)

*Cannot be computed because at least one of the variable (dental caries) is constant in control group
**Correlation is significant at the 0.01 level (two-tailed)
Thus, with an increase in the whorl pattern, the patient has an increased susceptibility to dental caries.

Loop vs dental caries when N = 1250 (Graph 2) with independent variable: Loop and dependent variable: Dental caries. Table 3 showed that –83% correlation existed between loop and dental caries (p-value of 0.000). Thus, the two variables loop and dental caries were negatively correlated (r = –0.60). Thus, the two variables loop and dental caries were negatively correlated (r = –0.83). Table 3 shows the same result when permutation and combination was done.

Loop vs dental caries (Graphs 2 and 3) in study group (Table 4) when N = 625 with independent variable: Loop and dependent variable: Dental caries. Table 4 showed that –60% correlation existed between loop and dental caries (p-value of 0.013). Thus, the two variables loop and dental caries were negatively correlated (r = –0.60). Table 4 shows the same result when permutation and combination was done.

Thus, with an increase in the loop pattern, the patient has a decreased susceptibility to dental caries (Graphs 3 to 6).

To determine a degree of divergence of specific dermatoglyphic patterns among study and control group, i.e. to find any significant difference exists between study and control group for both whorl and loop variable we used independent t-test to test the hypothesis.

**Table 3: Correlation between loop vs dental caries when N = 1250**

<table>
<thead>
<tr>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Loop Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
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<tbody>
<tr>
<td>Dental caries</td>
<td>1.000</td>
<td>*</td>
<td>1250</td>
<td>–0.826</td>
<td>0</td>
<td>1250</td>
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<td>Sig. (2-tailed)</td>
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<td>N</td>
<td>1250</td>
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<td>Loop</td>
<td>–0.826</td>
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**Correlation is significant at the 0.01 level (2-tailed)**

**Table 4: Correlation between loop vs dental caries in study group when N = 625**

<table>
<thead>
<tr>
<th>Group</th>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Loop Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
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<tr>
<td>Study</td>
<td>Dental caries</td>
<td>1.000</td>
<td>*</td>
<td>625</td>
<td>– 0.60</td>
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<td>Sig. (2-tailed)</td>
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<tr>
<td>Loop</td>
<td>Dental caries</td>
<td>– 0.60</td>
<td>0.013</td>
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<td>1.000</td>
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<td>625</td>
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<td>Sig. (2-tailed)</td>
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<td>N</td>
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<tr>
<td>Control</td>
<td>Dental caries</td>
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<tr>
<td>Loop</td>
<td>Dental caries</td>
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<td>Sig. (2-tailed)</td>
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*Cannot be computed because at least one of the variable (dental caries) is constant in control group
**Correlation is significant at the 0.01 level (two-tailed)
Whorl in study vs control group (Table 5): Showed calculated t-value was 72.333, with a mean difference of 6.85 and p-value was 0.000 for whorl pattern in study vs control group. This signifies that there exists a significant difference between study and control group. 95% confidence interval also supports that there is significant difference between study and control group. {Lower limit, upper limit} = {6.67, 7.04}, standard error difference = 9.48E-02, null value = 0, confidence interval (CI) = 95%.

W = S/C = at 95% CI = {6.67, 7.04}. Whorl (W) in study (S) vs control (C) group at 95% confidence interval was between 6.67 and 7.04 which do not include our null value. Hence, our result was statistically significant.

Loop in study vs control group: (Table 5) (Graphs 4 and 6): Table 5 showed calculated t-value was – 63.654, with a mean difference of – 6.40 and p-value was 0.000 for Loop pattern in study vs control group. This signifies that there exists a significant difference between study and control group. 95% confidence interval also supports that there is significant difference between study and control group. {Lower limit, upper limit} = {– 6.60, – 6.21}, standard error difference = 0.10, null value = 0, Confidence interval (CI) = 95%.

L = S/C = at 95% CI = {– 6.60, – 6.21}. Loop (L) in study (S) vs control (C) group at 95% confidence interval was between – 6.60 and – 6.21 which do not include our null value. So our result was statistically significant.

To summarize our results, dental caries susceptibility of an individual increased with incidence of whorl pattern and it decreased with incidence of loop pattern. (The analysis of the data was done using SPSS software version 13).

**DISCUSSION**

Dental caries is a microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth. Dental caries is the most common chronic disease of childhood and is unequally distributed in the population with most of the disease occurring in 20% of children. Dental caries is a chronic, complex, multifactorial disease for which a multitude of etiologies like host and environmental factors have been proposed. The relative roles of heredity and environmental (nature vs nurture) in the pathogenesis of dental caries has intrigued clinical and basic researchers for decades. There are numerous host resistance and risk factors for dental caries that are genetically determined. It is critical to realize that genes and environment do not act independently of each other and the appearance or magnitude of heritability may differ with various environments.

The pattern of dental caries is similar in members of the same family over several generations and hence, inheritance of this susceptibility is suspected. There are inherited traits that alter the susceptibility to dental caries in humans. Genetic variations in the host factors may contribute to
increased risks for dental caries. Environmental factors, such as diet, oral hygiene habits also play a large role in causing dental caries.

The type of fingerprints is unique and is based on the genetical characteristics of each individual. These dermal patterns once formed remain constant throughout life. Till now, only one study has been conducted in a very small group comprising only 24 patients by Metin Atasu (1992) to analyze the dermatoglyphic patterns in dental caries. We designed and undertook this study to evaluate and analyze the dermatoglyphic patterns in patients with dental caries. From our results we can conclude that the dermatoglyphic patterns varied significantly among the patients with dental caries and healthy individuals. Our study results were similar to other studies like Cummins et al on Down’s syndrome and Bierman et al on breast cancer, who noted significant variations in whorl and loop patterns.

Our results also showed that with an increase in the whorl pattern, the patient had an increased susceptibility to dental caries. This result could be compared to Engler et al (1982), who had analyzed dermatoglyphic patterns in breast cancer patients, and concluded that the presence of six or more whorls on the fingertips of a person could indicate a high risk of obtaining breast cancer.

There is a statistically significant difference between study and control group in loop and whorl pattern similar to Metin Atasu (1992). Thus, we found a definite correlation between the dermatoglyphic patterns and patients with dental caries.

In comparison with the control group, 83% positive correlation was found between whorl and dental caries at a p-value = 0.000. This is highly significant so, we analyze the possible reason for this significance. Dermal ridge differentiation takes place early in the fetal development. It is known that finger and palm prints are formed during the first 6 to 7 weeks of the embryonic period and are completed after 10 to 20 weeks of gestation. Abnormalities in these areas are influenced by combination of hereditary and environmental factors. These abnormalities are expected to appear only when the combined factors exceed a certain level. This threshold theory is now generally accepted and has been extrapolated by the studies of Carter (1969) and Mastunga (1977). Consequently, dermatoglyphics could indicate a genetic susceptibility to dental caries. In the recent decades, a considerable improvement has been achieved in the concept of correlation between the types of pattern of lines on the fingers and some individual disorders. The pattern of lines on the hand finger has been documented in medicine as a method of diagnosis.

Numerous studies have described a potential genetic contribution to the risk for dental caries. There are numerous familial, pedigree and twin studies on dental caries. Studies on twins have provided strong evidence for the role of inheritance. So, the most convincing data on the role of genetics in the pathogenesis of dental caries have been developed by analyzing the caries incidence in monozygotic and dizygotic twins. It was also suggested by different studies that the children showed a remarkable similarity in dental caries to the susceptibility of the parents.

The pathogenesis of the caries process is rather well understood today, and caries attack rate in humans is a consequence of various attributes. Genetically, regulated processes identified as contributing to caries incidence include tooth eruption, tooth morphology, density or structural integrity of the enamel, composition of the secretions of the salivary glands and salivary flow, the immune response and reduction in the clearance of the bacteria. Bordoni concluded from his study that there is a ‘strong genetic component in primary teeth which affects the incidence of caries’.

Individuals with high resistance to dental caries had a specific immunoglobulin within saliva conveying immunity by lysing the cariogenic bacterial cells. It was suggested that this phenotype was inherited and transmitted as an autosomal dominant trait. Several reports and studies have also shown significant heritability for several microorganisms, including streptococci. Thus, genes and genetic abnormalities that leads to abnormal structural organization of teeth and its environment leads to increased susceptibility to dental caries.

Hence, we can also conclude susceptibility to dental caries has genetic control and this control could be multifactorial in nature.
Studies reveal that HLA DR6-1, 2, 3 had a significant relationship to dental caries, with increased susceptibility to dental caries, enamel defect, as well as to low dose response to *Streptococcus mutans* antigens. HLA DR 5, 7 with decreased enamel defect and dental caries.2,25-27

Two different lines of investigation have proved that genes in the HLA complex are associated with altered enamel development and increased susceptibility to dental caries. Specific allelic variants of these genes could be used as a potential marker to assess the increased dental caries risk.2,28-32

Although conclusions could be drawn based on this study, digital dermatoglyphics may have a future role in identifying people either with or at increased risk for dental caries so that either risk reduction measures or earlier therapy may be instituted. We also have some evidence from this study to suggest that specific fingerprint patterns may be used as a potential noninvasive anatomical tool which could be used for screening for dental caries and for guiding future research. This relatively noninvasive technique can reasonably be used in selective nonsymptomatic patients (those with positive family history) as a part of definite risk assessment strategy with an ability to detect the earliest changes associated with cariogenesis, many years before the appearance of clinical lesion. This may allow the introduction of more preventive, early diagnosis and effective treatment strategies in patients with dental caries.33-51

SUMMARY
Thus from the our observations and study, it can be summarized that:

1. Dental caries susceptibility of an individual increases with an increase in the incidence of whorl pattern (83% correlation).
2. All the variables show statistically significant value, with a degree of divergence of specific dermatoglyphic patterns among study and control group.
3. The dermatoglyphic patterns are efficient and can predict in assessing the risk of susceptibility to dental caries in study group.

CONCLUSION
The dermatoglyphic patterns may be utilized effectively to study the genetic basis of dental caries. In a developing country like India, it might prove to be a noninvasive, inexpensive and effective tool for screening. These patterns may represent the genetic make up of an individual and therefore his/her predisposition to certain diseases.

Given the expenses involved in conducting the analysis of the chromosomes themselves, dermatoglyphics can prove to be an extremely useful tool for preliminary investigations. The pattern seems to be appearing wherein a definite approach in the form of ‘dermatoglyphics’ might play a significant role in the near future not only for the purpose of screening but also for studying the behavior of dental caries.

Since, dermatoglyphics is still an inexact science at the present time, further extensive research and studies in this field have to be done in order to determine, ascertain and to evaluate the significance of these variations in the dermatoglyphic features of patients with dental caries.

REFERENCES
24. Fogle T. Using dermatoglyphics from down syndrome and class populations to study the genetics of a complex trait. Association for Biology Laboratory Education 1990;11:129-50.
27. Balgir RS. Dermatoglyphics in clef lip and clef palate anomalies. PMID: 8365784.

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