Finger patterns and age of onset for the determination of the parent-of-origin in the transmission of schizophrenia

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ABSTRACT

Summary: Dermatoglyphic traits which are reported to be largely determined by genes could be considered as phenotypic characteristics and if the same are expressed through generations in schizophrenic families it can be speculated to serve as genetic markers for schizophrenia. Another factor that might be influenced by genes is the age of onset of the illness in the offspring and the parent of origin.

Objective: This study was aimed to elucidate the occurrence of identical finger patterns in the schizophrenic patients and their affected parents. The other objective was to assess the age of onset of the illness in them.

Methods: Forty six schizophrenic patients in whom one of the parents was also affected with schizophrenia or related disorders were recruited. Of these pairs 29 were taken up for finger patterns analysis, with an equal number of control group pairs. 35 proband and parent pairs were investigated for the age of onset of the illness.

Results: The frequency of occurrence of identical patterns in the right thumbs of proband and their affected mother pairs was significantly more than between the proband and their affected father pairs. Additionally, the number of identical patterns was also more in the right thumbs of proband and their affected mother pairs compared with the control group. The difference between the mean age of onset of the illness in the probands and their affected fathers was more than between the probands and their affected mothers.

Conclusion: The genetic association of schizophrenic patients with the affected maternal side appear to be more stronger than with the paternal side.

Key words: Dermatoglyphics, genes, genetic marker, inheritance, phenotypes

INTRODUCTION

Ever since Kallman[1] reported on the empirical risks in the families of schizophrenics, several observers have reported on the prevalence of schizophrenia in the families of probands. Schizophrenia is aggravated in families because of genetic factors. Many genetic conditions segregate sharply within families, that is, the abnormal phenotype can be distinguished clearly from the normal one. In clinical experience, however, some disorders are not expressed at all in genetically predisposed persons, and others have extremely variable expression in terms of clinical severity or onset age or both. Furthermore, expression of an abnormal genotype may be modified by other genetic loci or environmental factors. Exploring the pathophysiology of schizophrenia through genetic research is a critical step toward improving prevention, detection and implementing early treatment of this devastating illness.

Genomic imprinting

Based on Mendelian principles, we expect that an autosomal
gene is equally likely to be transmitted from a parent of either sex and to an offspring of either sex, similarly, a female is equally likely to transmit either of her X chromosomes to a child of either sex. Little attention used to be paid to whether the sex of the transmitting parent had any effect on the expression of genes.

Assessment of the manifestation of the same phenotypic characteristics of the parent in origin and their offspring could be one way of understanding the transmission of genes and the genotypic influence of the affected parent on the concerned phenotypic expression in the affected offspring.

**Dermatoglyphics**

Dermatoglyphic characteristics that are unique for any individual is said to be determined by genes. Although the exact loci for these dermatoglyphic parameters is still unknown, if any of the characteristics occur in one of the parent as well as in their offspring, it could be inferred that the genes responsible for these characteristics might have been transmitted from that parent to the offspring.

**Age of onset of the illness**

In the genetic epidemiology of schizophrenia, the phenomenon of anticipation and the influence of parent-of-origin effect in the age of onset is a recent focus. However, this phenomenon in schizophrenia lacks adequate support. Furthermore, anticipation is probably a consequence of bias ascertainment rather than a true biological phenomenon. In fact, when the imprinted genes are transmitted, the age of onset in the offspring and parents might not manifest substantial variation.

**AIMS AND OBJECTIVES**

The objective of this study was to find out the frequency of occurrence of identical finger patterns between a proband affected with schizophrenia and their parents affected with schizophrenia or its related disorders.

Besides, it was aimed to elucidate the age of onset of the illness in the probands and in their respective affected parent that could indicate any specific pattern of imprinting occurring depending on the sex of the parent-of-origin.

**MATERIALS AND METHODS**

**Subjects**

All new cases of schizophrenia who reported to the psychiatric outpatient services of Government General Hospital, Chennai, from January 1981 to April 1982 were surveyed. Informed consent was obtained from the patients and their relatives for this study. Confidentiality of the information obtained from them was also assured. These patients and their parents had been inhabitants of Chennai city, an urban area in the state of Tamil Nadu of South India. Each case was evaluated independently by two senior consultant psychiatrists for ensuring diagnostic consensus before being entered into the project. Only those who had positive family history and fulfilled the Feighner criteria for schizophrenia were selected. Their interviews were performed using the schedule for affective disorders and schizophrenia by trained psychiatrists (RP and JJ). Fifty-seven schizophrenic patients were thus chosen. A retrospective analysis of case histories showed that they fulfilled the Diagnostic and Statistical Manual of Mental Disorders IV (DSM IV) criteria for schizophrenia.

As a source of diagnostic information about the relatives, at least two key informants who were personally acquainted with the family members, either through friendship or blood relationship, were interviewed in accordance with the interview guide for the Family History Research Diagnostic Criteria (FHRDC). Diagnosis in the relatives was made on the basis of FHRDC, and wherever possible information from personal interviews of the other family members, from hospital records and from various other sources were also pooled. The relatives who manifested schizophrenia, schizophreniform psychosis, atypical psychosis, were considered as affected persons. This sample was further strengthened by recruiting all consecutive family history positive schizophrenic patients in a similar manner, but who reported to the Institute of Mental Health, Chennai, from January 2003 to August 2003 and fulfilled the DSM IV criteria for schizophrenia. As a result, another 37 subjects were added to the original sample, which enabled to enlarge the sample to 94. Excluded were the subjects with inadequate family history, with consanguineous parents, whose parents were both affected and those who were unwilling to participate in this investigation. Of the 58 subjects thus selected only 46 subjects with either of their parental sides affected were taken up for this study. Both these centers from which the subjects were recruited were located in the city of Chennai within a radius of about 5 km. The clinical data, including of the screening schedule for affective disorder and schizophrenia, diagnostic criteria, and family history diagnostic criteria, pertaining to the first group of subjects were again screened by the senior consultant psychiatrists involved in the recruitment of the second group (RP, JJ). Consensus was reached on the diagnosis of the first group and also on their merger with the second group, in view of the diagnostic agreement between the two groups. Hence, intra-center and inter-center reliability of the diagnosis and ascertainment of the family history were ensured. In fact, a different study of the first group of subjects has already been reported elsewhere and the merged first and second groups were the subjects for another type of analysis.

Three cases of inexplicable suicides and one case of absconder, who were otherwise also suffering from...
schizophrenia as per FHRDC, were included, since no noteworthy co-morbid causes for these behaviors including depression or substances abuse or personality disorder were elicited. Other diagnostic categories such as mood disorders, neurotic disorders, mental retardation, dementias, substances abuse, and psychosis due to medical conditions were not considered. Any parent of the proband who remained unaffected, but if their sib was affected, such parent was considered as a possible carrier of the disease gene and counted as affected individuals for the purpose of this study. Inheritance of these disease genes by them could be from their common ancestor, and the same might have been transmitted to their next generation. This assumption has also been adopted earlier in a computational model by some,\textsuperscript{[13-15]} while utilizing the data of affected paternal and maternal side family members as ancestral secondary cases for assessing the mode of inheritance. Wherever >1 affected sib of the parent was encountered, to avoid the possible influence of sex and age related bias, the one belonging to the same sex of the parent with the lower age of onset was only considered.

**Finger print analysis**

After excluding those whose prints were unclear in all the fingers and whose parents were also not alive, only 29 Proband-Parent pairs could be recruited for finger pattern analysis. However, if some of the finger patterns were only unclear in a subject, those fingers alone were excluded in the offspring-parent pairs of the patient and control groups. An equal number of (N-29) sex- and age-wise matched control offspring-parent pairs who reported to the Government General Hospital for common general medical problems such as febrile illness, and bronchitis were recruited. Absence of any psychiatric illness or genetic disorders either in them or in their families was another criteria for including them in this study as a control group. Ultimately the total subjects for fingerprint analysis were 116.

The method used to take the rolled fingerprints was the ink and pad method. The classification and analysis of the finger patterns were done as per the guidelines of Cummins and Midlo\textsuperscript{[16]} and also wherever needed by following the pictorial descriptions provided by Schaumann and Alter.\textsuperscript{[17]} Of the three usual pattern types viz., arch/tented arch, loop (ulnar or radial), whorls of different configurations, and also of the unusual complex pattern types, whichever type is displayed on the distal phalange on a particular finger of the proband and if the same pattern type is noted in the distal phalange of the corresponding finger of the parent, the concerned finger patterns of the proband parent pair is considered identical. On the contrary, if a particular finger of the proband and the corresponding finger of the parent do not display the same patterns (e.g. displayed whorl and loop respectively) the concerned finger patterns of the proband parent pair is considered nonidentical for the purpose of this study. When all the finger patterns in the hand were together compared, even if one of the finger patterns in a hand was at variance with that of the corresponding finger of the other hand, the concerned pair of hands were considered as nonidentical.

The identification of the pattern type was made independently by the two authors (R.P and J.J) and there was complete agreement between the two authors on the interpretation of pattern types. Inter-rater reliability was thus ensured. Prior training was obtained in analyzing the dermatoglyphic data from the Director, Finger Prints Bureau, Government of Tamil Nadu, who is primarily involved in crime detection in the State of Tamil Nadu.

**Assessment of age of onset**

After excluding those in whom the age of onset of the illness could not be correctly obtained, 35 proband-parent pairs could only be recruited for this investigation. For establishing the age of onset of schizophrenia, the time of occurrence of the first characteristic symptoms\textsuperscript{[18,19]}

| Table 1: Frequency distribution of identical and nonidentical finger patterns between probands and affected parents |
|---------------------------------------------------------------------------------------------------------------|
| Finger patterns | Proband versus affected fathers | Proband versus affected mothers | Chi-square | Pearson uncorrected | Yates corrected |
|-----------------|---------------------------------|-------------------------------|------------|-------------------|---------------|
| Thumb (right)   | Nonidentical (9 (60))          | 12 (85.71)                      | 6.428      | P=0.011*          | P=0.031*      |
|                 | Nonidentical (6 (40))          | 2 (14.28)                       | 2.392      | P=0.122           | P=0.243       |
| Thumb (left)    | Nonidentical (6 (40))          | 9 (69.23)                       | 1.362      |                   |               |
| Index (right)   | Nonidentical (9 (60))          | 4 (30.77)                       | 0.291      |                   |               |
| Index (left)    | Nonidentical (9 (64.29))       | 12 (85.71)                      | 1.714      | P=0.190           | P=0.383       |
|                 | Nonidentical (5 (35.71))       | 2 (14.28)                       | 0.303      |                   |               |
| Middle (right)  | Nonidentical (11 (73.33))      | 11 (78.57)                      | 0.109      | P=0.534           | P=1.000       |
|                 | Nonidentical (4 (26.67))       | 3 (21.43)                       | 0.762      |                   |               |
| Middle (left)   | Nonidentical (9 (64.29))       | 8 (61.53)                       | 0.000      |                   |               |
|                 | Nonidentical (5 (35.71))       | 5 (38.46)                       | 0.303      |                   |               |
| Ring (right)    | Nonidentical (12 (80))         | 10 (71.43)                      | 0.291      | P=0.590           | P=0.917       |
|                 | Nonidentical (3 (20))          | 4 (28.57)                       | 0.011      |                   |               |
| Ring (left)     | Nonidentical (9 (64.29))       | 8 (57.14)                       | 0.150      | P=0.699           | P=1.000       |
|                 | Nonidentical (5 (35.71))       | 6 (42.86)                       | 0.000      |                   |               |
| Little(right)   | Nonidentical (13 (92.86))      | 10 (76.92)                      | 1.356      | P=0.244           | P=0.534       |
|                 | Nonidentical (1 (7.4))         | 3 (23.08)                       | 0.387      |                   |               |
| Little(left)    | Nonidentical (13 (92.86))      | 11 (84.61)                      | 0.464      | P=0.496           | P=0.946       |
|                 | Nonidentical (1 (7.4))         | 2 (15.38)                       | 0.005      |                   |               |

*Significant. Figures in parenthesis indicate percentage
RESULTS

Finger patterns

The frequency of occurrence of identical patterns in the right thumbs of the proband and their affected mother pairs was significantly more in comparison to the frequency between the proband and their affected father pairs. Interestingly, the number of identical patterns was also notably more in the right thumbs of proband and their affected mother pairs compared with the control offspring and their mother pairs [Tables 1-3]. However, when all the finger patterns together in the hand as a whole were compared with the corresponding hands of the proband and their affected either side parents no noteworthy difference emerged [Table 4].

Age of onset

The noted differences between the mean age of onset of the illness in the probands and their affected fathers was significantly more (17.58) than the observed differences of 6.68 in the age of onset between the probands and their affected mothers [Table 5]. Perhaps this finding reflects more similarity in the age of onset between the probands and their mothers in comparison to their fathers.

DISCUSSION

Dermatoglyphics

Dermal ridge differentiation takes place early in fetal development. The resulting ridge configurations are genetically determined and influenced (or modified) by environmental forces. Furthermore, the configuration types are individually variable, but, their determination by genes indicate that blood relatives may however depend on the sharing of the genes responsible for their formation.

Finger patterns

If one of the parent serves as the parent of origin for pattern formation, similarity of the finger patterns could be noted between the offspring and either of the concerned parent of origin when they both share the same genes responsible for this dermatoglyphic trait.

In the present investigation, the significantly more occurrence of identical patterns in the right thumbs of the proband and their affected mother pairs in comparison to the proband and their affected father pairs is noteworthy. In addition, the significantly more frequency of identical patterns in the right thumbs of proband and affected mother pairs in comparison to the control offspring and their mother pairs, might perhaps indicate more sharing of the genes that is responsible for right thumb pattern of the affected mothers and their offspring than between the

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was considered as per the furnished history from reliable informants.

Data analysis

The difference in the frequency of occurrence of the identical finger patterns in the proband and affected father pairs in comparison to the proband and affected mother pairs was analyzed by using the Chi-square test. Similar analysis was performed of the distribution of identical finger patterns in proband and the affected parent of either sex in comparison to the control offspring and their either sex parent.

Apart from individual digits, each side finger patterns together in a hand as a group were compared with those of the corresponding hands in the sample subjects and the hands were grouped as identical and nonidentical for comparison using Chi-square test.

The difference between the mean age of onset of the illness in the proband and father pairs and in the proband and mother pairs was compared using unpaired t-test with equal variance.
| Table 3: Frequency distribution of identical and nonidentical finger patterns between proband and affected mother pairs, and controls |
|---------------------------------------------------------------|
| Finger patterns | Proband and affected mothers | Control off spring and mothers | Chi-square |
|                 | | | Pearson uncorrected | Yates corrected |
| Thumb (right)   | | | | |
| Identical       | 12 (85.71) | 6 (42.86) | 5.600 | 3.889 |
| Nonidentical    | 2 (14.28)  | 8 (57.14)  | 0.018* | 0.049* |
| Thumb (left)    | | | | |
| Identical       | 9 (69.23)  | 7 (53.85)  | 0.650 | 0.163 |
| Nonidentical    | 4 (30.77)  | 6 (46.15)  | 0.420 | 0.687 |
| Index (right)   | | | | |
| Identical       | 12 (85.71) | 7 (50) | 4.094 | 2.620 |
| Nonidentical    | 2 (14.28)  | 7 (50) | 0.043 | 0.106 |
| Index (left)    | | | | |
| Identical       | 3 (23.08)  | 4 (30.77)  | 0.195 | 0.000 |
| Nonidentical    | 10 (76.92) | 9 (69.23)  | 0.658 | 1.000 |
| Middle (right)  | | | | |
| Identical       | 11 (78.57) | 10 (71.43) | 0.190 | 0.000 |
| Nonidentical    | 3 (21.43)  | 4 (28.57)  | 0.663 | 1.000 |
| Middle (left)   | | | | |
| Identical       | 8 (61.53)  | 8 (61.53)  | 0.000 | 0.000 |
| Nonidentical    | 5 (38.46)  | 5 (38.46)  | 1.000 | 1.000 |
| Ring (right)    | | | | |
| Identical       | 10 (71.43) | 10 (71.43) | 0.000 | 0.000 |
| Nonidentical    | 4 (28.57)  | 4 (28.57)  | 1.000 | 1.000 |
| Ring (left)     | | | | |
| Identical       | 8 (57.14)  | 8 (57.14)  | 0.571 | 0.143 |
| Nonidentical    | 6 (42.86)  | 8 (57.14)  | 0.450 | 0.705 |
| Little (right)  | | | | |
| Identical       | 10 (76.92) | 10 (76.92) | 0.000 | 0.000 |
| Nonidentical    | 3 (23.08)  | 3 (23.08)  | 1.000 | 1.000 |
| Little (left)   | | | | |
| Identical       | 11 (84.61) | 7 (53.85)  | 2.889 | 1.625 |
| Nonidentical    | 2 (15.38)  | 4 (26.15)  | 0.089 | 0.203 |

*Significant. Figures in parenthesis indicate percentage.

| Table 4: Frequency distribution of identical and nonidentical finger patterns of either hands in proband and affected parents |
|---------------------------------------------------------------|
| Finger patterns | Proband and affected fathers pairs | Proband and affected mother pairs | Chi-square |
|                 | | | Pearson uncorrected | Yates corrected |
| Right hand      | | | | |
| Identical       | 2 (15.38)  | 6 (46.15)  | 2.889 | 1.625 |
| Nonidentical    | 11 (84.61) | 7 (53.85)  | 0.089 | 0.202 |
| Left hand       | | | | |
| Identical       | 4 (28.57)  | 3 (25)      | 0.042 | 0.000 |
| Nonidentical    | 10 (71.43) | 9 (75)      | 0.838 | 1.000 |

| Table 5: Differences in the age of onset between the probands and their parents |
|-----------------------------------------------|
| Difference in the age of onset between the probands and their affected fathers | Difference in the age of onset between the probands and their affected mothers |
| Mean | 17.579 | 6.625 |
| SD   | 9.856 | 11.430 |
| n    | 19    | 16    |

df=33, T=3.0454, P=0.005. SD - Standard deviation.

The emergence of the significant difference only in the thumb patterns might subscribe to the theory that different genetic or other biological mechanisms could operate for the formation of each of the finger patterns as well as for various other dermatoglyphic parameters. Furthermore, absence of noteworthy difference when all the finger patterns in the hand as a whole were compared could strengthen the argument for the need to analyze the various dermatoglyphic parameters separately.

In a study on the sequential development of dermatoglyphs in schizophrenia,[20] greater difference between ridge counts of right thumbs and right big toes of female schizophrenic patients compared to controls was noted. Interestingly, notwithstanding a different design and objective in this study, it was pointed out that it was the right thumb ridge counts of the female patients only that had contributed to this exaggerated difference.

It may be argued that the comparison of the finger ridge counts might have been more informative in addition to finger patterns. It is pointed out that although the finger patterns may be identical between two homologous fingers, there may be substantial difference in their ridge counts and conversely when the finger patterns are nonidentical, still there may not be any appreciable variation between their ridge counts. Furthermore, sex differences in ridge counts are another factor that prompted the authors to avoid finger ridge count comparison. Ideally speaking, comparison of the nature of individual ridges of identical patterns, as is done in criminal investigations, might have been more valid. However, this approach will inevitably fail to yield any pair of ideal identical patterns as the ridge configurations are unique for individuals.

**Age of onset and parent of origin**

A long series of family, twin, and adoption studies has clearly demonstrated that inheritability is the strongest determinant of schizophrenia. The closeness of the age of onset of the schizophrenic illness in the probands and their affected mothers perhaps reveal a genetic association between them, in comparison to the probands and their affected fathers. This might occur probably even through the same at-risk haplotypes; thus a shared genetic vulnerability between them becomes an emerging scenario. This finding might predict the preponderance of the susceptible genes from the maternal side to the affected offspring rather than from the paternal side, but, the underlying genetic mechanism might not be clearly Mendelian.

It is also important to note that there are no known schizophrenia-related copy number variants for which a
severe psychiatric phenotype is inevitable, and although they may occur de novo they are also frequently transmitted from an apparently healthy parent. Moreover, when a phenotype is present, expressivity is highly variable, with phenotypes ranging from mild cognitive or physical anomalies through to schizophrenia, intellectual disability, attention deficit hyperactivity disorder, epilepsy and autism even within the same family.\textsuperscript{[21]}

However, DeLisi \textit{et al.},\textsuperscript{[22]} and Imamura \textit{et al.},\textsuperscript{[23]} found no evidence for a parent of origin effect in the pattern of inheritance in schizophrenia. Yet, noteworthy also the hypothesis that a specific pattern of imprinting occurs due to the differential expression of genes depending on the parental origin of the gene contributing to the pattern of inheritance of schizophrenia.\textsuperscript{[24]}

\textbf{Assessment of other clinical variables}

Apart from the age of onset of the illness, assessment of some of the other evident clinical variables could not be undertaken in our study for determining the parent of origin. The severity and the clinical signs in the illness that could help to draw comparison between the proband and parent could not be undertaken in this study, since substantial number of our patients owing to their ignorance and illiteracy, had undergone various interventions such as magico-religious treatments, management by general practitioners or treatments with alternative medicines including Indian Medicine, Sidha, Unani, and Homeopathy, prior to reporting to our facility which could mask the clinical findings. Equally, hospitalization too may be a biased variable, since some subjects needing hospitalization might decline the same due to the stigma attached to mental illness in our country and also partly because of financial and other domestic reasons. On the contrary, those who could have been on domiciliary care preferred hospitalization due to factors such as poor housing facility, inability to commute frequently to our center and lack of adequate caregivers at home. Furthermore, even the assessment of the dosage and nature of the drugs given were considered unwise, since our first group of subjects were mostly on older generation of antipsychotics, whereas substantial proportion of the second group were on newer generation drugs. In addition, our centers are run costless for poor patients by the government, and many of our patients could not afford costly drugs. At any rate, noteworthy is also the observation by Imamura \textit{et al.},\textsuperscript{[21]} that no appropriate criterion has been developed to estimate the disease’s lifetime severity.

\textbf{LIMITATIONS OF THE STUDY}

The small sample size might raise concerns in this study. Since the selection had to be restricted to only informative families and also consanguineous families, which are in fact more prevalent in our culture, were not included, substantial proportion of participants from our original sample had to be excluded. Hence, considering the sample size, caution is needed in the interpretation of the results. Recruiting the subjects from two different clinical settings might appear to be another weakness. But, it should be noted that the two centers are in the same city with their patients from the same background. The possibility of omission of a few patients from the original sample who could otherwise have been included if the present DSM-IV criteria were applied at that time itself might be another criticism. However, the application of only the Feighner’s criteria for recruiting the original sample could be considered appropriate at that time. At any rate, this study could prompt further studies involving not only larger sample size, but also incorporating more number of dermatoglyphic parameters and other variables.

\textbf{CONCLUSION}

It seems reasonable to argue that the sharing of the susceptible genes between the schizophrenic patients and their maternal side might be more than their paternal side. It is speculated that thumb patterns, and perhaps some of the other dermatoglyphic parameters too, might be promising tools as genetic markers for schizophrenia.

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