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Mode of inheritance of finger dermatoglyphic traits among Vaidyas of West Bengal, India

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Summary. *Background*: It is well established that dermatoglyphics are genetically determined. But, to date, few studies have given attention to the inheritance pattern of dermatoglyphics. Furthermore, despite the existence of different advanced statistical packages, none of these previous studies implemented a model-fitting technique to reveal the mode of inheritance. Thus, the genetic nature of dermatoglyphics is still not clear.
Aim: In the present communication, an attempt has been made to provide some information regarding the genetics of finger dermatoglyphics by estimating the magnitude and mode of inheritance of these traits.
Subjects and methods: The fingerprints of 824 individuals from 200 families including two generations were collected from Barasat in North 24-Parganas, West Bengal. The study includes familial correlations between first-degree relatives and corresponding heritabilities. In the final stage, segregation analyses by the Pedigree Analysis Package (PAP) were conducted on these data to understand the mode of inheritance.
Results: The major findings indicated the following: (a) Familial correlations in all possible relationships (except spouse correlation) were statistically significant and of comparable magnitude. (b) The corresponding heritabilities were in the range between 59% for Pattern Intensity Index (PII) and 77% for Total Finger Ridge Count (TFRC). These estimates were in agreement with previously published data on this subject. (c) By segregation analysis, the 'Sporadic', 'Environmental', 'No major gene effect' as well as 'No polygenic component' models were strongly rejected ($p < 0.05$) and the hypothesis of a major gene's (MG) influence on all studied traits was accepted, though the proportion of MG variance was low. (d) The Most Parsimonious Mendelian model clearly indicated the contribution of a major gene with dominant (for PII) and additive (for two ridge counts) effects.
Conclusion: The present report supports the evidence of the existence of a major gene on these dermatoglyphic traits and the transmission of this effect is consistent with Mendelian expectation.

1. Introduction

Galton (1892) was the first to demonstrate that the development of dermatoglyphics is controlled by genetic factors. Numerous other studies of dermatoglyphics have established that it has a genetic basis and there is no alteration in the structure of ridge pattern after birth. But even after more than a century little research has been directed toward assessing the genetic component in the development and expression of dermatoglyphics and few studies have focused on their inheritance pattern (Loesch 1971, Kleopfer 1979, Chakraborty *et al.* 1982, Martin *et al.* 1982, Das Choudhuri and Chopra 1983, Singh 1995, Devi 2000). Although most of these studies have assumed a polygenic mode of inheritance (Holt 1968, Slatis *et al.* 1976, Pons 1979), others do not agree with the assumption of additive polygeny (Pons 1970, Loesch 1971, Roberts and Coope 1975, Roberts 1979).

In the reviewed literature it has been found that most of the previous studies are simply based on the application of correlation or regression between relatives though these analyses are not sufficient to detect the mode of inheritance of a trait. The information on the genetics of dermatoglyphics is, therefore, fragmentary and the results are contradictory. No adequate model of inheritance has yet been established, in spite of rapid progress in statistical analysis as well as widespread availability of computers. Some inheritance studies have been published that include segregation analysis on anthropometric traits (Rice *et al.* 1993, 1999, Livshits *et al.* 1995, 1998, Ginsburg *et al.* 1998, 1999, Ginsburg and Livshits 1999, Skaric-Juric *et al.* 2003), body fat measurement (Bouchard *et al.* 1993, Mitchell *et al.* 1993, Comuzzie *et al.* 1995, Olson *et al.* 2001), blood pressure (Nirmala *et al.* 1992, Weissbecker 1993, Cheng *et al.* 1998), bone mineral density (Gueguen *et al.* 1995, Cardon *et al.* 2000, Livshits *et al.* 1999a, 2002) as well as on serological and biochemical markers (Iselius *et al.* 1989, Livshits *et al.* 1999b, Friedlander *et al.* 2003). Very few studies on palmar dermatoglyphics also include segregation analysis (Gilligan *et al.* 1985, 1987). To our knowledge, hardly any available studies use a genetic model test on finger dermatoglyphics to understand the pattern of inheritance. We, therefore, still have a vague and obscure idea about the nature of genetic and environmental bases of these traits.

It is well known that dermatoglyphics has manifold applications (Cummins and Middlo 1976), such as in personal identification, genetics, population variation, twin diagnosis, dispute paternity, primatology as well as in medicine. But its actual utility is limited due to the lack of adequate knowledge of the genetic nature of dermatoglyphics. Therefore, a thorough understanding of the mode of inheritance of these quantitative traits is essential and in this respect we should adopt some complicated models to resolve the existing inconsistencies in the literature. Thus the goal of the present communication is not only to estimate the resemblance between relatives and the magnitude of heritability of these quantitative traits, but also to evaluate the mode of inheritance or the type of characters represented by ridge patterns by the use of complex segregation analyses.

2. Data

For the present study, data on 200 families consisting of 824 individuals (table 1) from a particular caste (Vaidyas) were considered. Data were collected from the area Barasat in the district of North 24-Parganas, West Bengal. The Vaidya families, which have living parents and at least two children, were included in the present study. The nomenclature of the possible pairs of relatives within a family is presented in table 2.

This population provides a good opportunity for an inheritance study, as it still maintains a gene pool through endogamy. The exact time and origin of this caste as a separate caste group is under dispute among different scholars. Some consider the Vaidyas to be an offshoot of the Brahmins who have intermarried with the other

Table 1. The sample size of the present study.

	Male	Female	Total
Parental generation	200	200	400
Offspring generation	229	195	424
Total	429	395	824

Table 2. The nomenclature of the possible relatives.

Serial no.	Relationships	Abbreviation
	inter-class	
1	Husband–wife	HW
2	Father–son	FS
3	Father–daughter	FD
4	Mother–son	MS
5	Mother–daughter	MD
6	Father–child	FC
7	Mother–child	MC
8	Parent–child	PC
9	Mid-parent–son	MidS
10	Mid-parent–daughter	MidD
11	Mid-parent–child	MidC
	intra-class	
12	Brother–brother	BB
13	Brother–sister	BS
14	Sister–sister	SS
15	sib–sib	Sib

castes like Vaishya, Sudra, etc., but there are also claims that they are direct descendants of Aryans who immigrated to Bengal (Dutta 1969). They generally occupy a high rank in society and are traditionally recognized as physicians although today, most of them are engaged in white-collar jobs.

Few authors have studied this particular caste, and most of these studies are based on serological and biochemical markers (Choudhury *et al.* 1969, Chakraborty *et al.* 1986). Scanty information is available about their dermatoglyphic characters (Banerjee and Banerjee 1975). In particular, the study of their inheritance has been very rare. For this reason this particular caste was chosen for the present study.

3. Methodology

3.1. Print analysis

The prints were collected by the widely used traditional ink method proposed by Cummins and Middlo (1976). Following Galton's classification, on the basis of the number of triradii present on finger, patterns were classified into four types: arches, radial loops, ulnar loops and whorls. The prints were analysed quantitatively by ridge counting following the method of Holt (1968). The whole data collection and print analysis were done by a single investigator (first author) to avoid inter-observer error. For the present study, Pattern Intensity Index (PII), Total Finger Ridge Count (TFRC) and Absolute Finger Ridge Count (AFRC) were considered.

3.2. Statistical analysis

The following statistical analyses were carried out:

(a) Z-transformation. Each value for dermatoglyphic traits was converted by Fisher's Z-transformation to normalize the data. $Z = (X_i - \bar{X})/\mathrm{SD}$ where X_i, $\bar{X}$ and SD are the individual measurement, average and standard deviation of the trait, respectively.

*(b) Familial Correlation (*r*).* The analyses was carried out with the help of two methods: (1) *Interclass correlation*—the degree of resemblance between parent–child and midparent–child was computed by usual Pearson's correlation, and (2) *Intraclass*

correlation—Fisher's formula (1958) was used to estimate the degree of resemblance between sib–sib, with a *t*-test of significance carried out.

(c) Heritability Estimate (h^2). The heritability of morphological characteristics has been studied by employing parent–offspring regression and intra-familial correlation following Falconer's method (1960). The analysis was carried out by means of the computer program Excel 97, while the first two were carried out using SPSS (version 7.5).

(d) Genetic Model tests. To evaluate the mode of inheritance, complex segregation analyses of the data were performed by Maximum Likelihood methods using the computer program Pedigree Analysis Package (PAP) (Hasstedt 1994). The program estimates the allele frequency (p), transmission probabilities (τ_{A1A1}, τ_{A1A2} and τ_{A2A2}), three genotypic means (μ_{A1A1}, μ_{A1A2}, μ_{A2A2}) with their standard deviations (σ_{A1A1}, σ_{A1A2}, σ_{A2A2}) and heritability of the traits (h^2), which represents the proportion of the within-genotype variance attributed to polygenes. However, the following genetic models have been tested (for the detail description of the models see Ginsburg and Livshits 1999):

1. *General Model (Free).* The General Model assumes the existence of two alleles (A_1 and A_2) at a single autosomal locus affecting the studied traits. In this model all the parameters are free from any restriction.
2. *Sporadic Model.* The hypothesis of no inter-generational transmission of studied traits was tested by constraining h^2 to zero ($h^2 = 0$) and the transmission probabilities of three genotypes (A_1A_1, A_1A_2, A_2A_2) to equal values ($\tau_1 = \tau_2 = \tau_3$).
3. *Environmental Model (Fixed).* The model assumes independence of offspring genotypes from the parental genotypes. As the selective effect of the three genotypes on the trait variation is not assumed, $\tau_1 = \tau_2 = \tau_3$.
4. *τ's equal to p.* the hypothesis of non-transmission of the major effect was tested by constraining the transmission probabilities to equal the first allele frequency ($p = \tau_1 = \tau_2 = \tau_3$).
5. *Mendelian Model (Mixed).* Under this hypothesis with the assumption of Hardy–Weinberg equilibrium, the probabilities of three putative genotypes in the population are p^2, $2pq$ and q^2. The transmission probabilities of allele A_1 by the corresponding above genotypes are: 1.0, 0.5, 0.0.
6. *No polygenic component.* Though this model assumes the inter-generational transmission (major gene (MG) effect) it does not assume the involvement of the additional polygenes (minor genes) into a trait transmission ($h^2 = 0$ and the other parameters are as in model 1).
7. *Most Parsimonious (MP) Mendelian.* If the Mendelian model was accepted, then the following sub-models were tested—*dominant*: $\mu_{A1A1} = \mu_{A1A2}$, *additive*: $\mu_{A1A2} = 0.5(\mu_{A1A1} + \mu_{A2A2})$ and *recessive*: $\mu_{A2A2} = \mu_{A1A2}$ major gene effects to understand the mode of inheritance of major genes.

Hypotheses 2–6 are sub-models of the general model and thus were compared with this model. Hypothesis 7 is the sub-model of the Mendelian model (model 5) and, therefore, was tested against it. The differences in the log-likelihood values ($2\ln L$) were distributed as χ^2. The degrees of freedom depend on the number of constraints imposed by the model. As the method of pedigree collection was not

Table 3. Correlation coefficients (*r*-values) of TFRC.

	TFRC									
	Holt (1952)		Loesch (1971)		Matsuda (1973)		Mathew (1980)		Present study	
Combinations	*n*	*r*	*n*	*r*	*n*	*r*	*n*	*r*	*n*	*r*
HW	149	0.05	186	0.04	195	0.02	100	0.03	200	-0.10^{NS}
FS	159	0.55	267	0.35	268	0.51	151	0.38	229	0.56*
FD	145	0.45	287	0.43	247	0.50	135	0.52	195	0.47*
MS	165	0.41	270	0.39	276	0.41	151	0.57	229	0.42*
MD	155	0.60	290	0.46	257	0.38	135	0.40	195	0.58*
FC	–	–	–	–	515	0.48	286	0.42	424	0.46*
MC	–	–	–	–	533	0.39	286	0.50	424	0.47*
PC	602	0.50	1114	0.41	1048	0.44	572	0.45	848	0.46*
MidS	–	–	–	–	243	0.62	151	0.62	229	0.65*
MidD	–	–	–	–	227	0.58	135	0.61	195	0.66*
MidC	301	0.67	552	0.58	470	0.60	286	0.61	424	0.65*
BB	–	–	–	–	–	–	–	–	152	0.44*
BS	–	–	–	–	–	–	–	–	240	0.45*
SS	–	–	–	–	–	–	–	–	110	0.46*
Sib	642	0.49	1342	0.38	–	–	371	0.45	502	0.45*

n, number of pairs.
$^{NS}p > 0.05$. $*p < 0.01$.
–, not available in the literature.

connected with the individual's dermatoglyphic traits, no ascertainment correction of likelihood was made.

The major gene variance and total variance were calculated from the MP model using following formulae—major gene variance $= (p^2\mu_1^2 + 2pq\mu_2^2 + q^2\mu_3^2) - (p^2\mu_1 + 2pq\mu_2 + q^2\mu_3)^2$, and total variance = major gene variance $+ \sigma^2$ (when σ^2 is the estimate of common variance).

4. Results

The estimated correlation coefficients of dermatoglyphic traits of the present study along with the results of some previous studies are presented in tables 3 and 4. Husband–wife correlations of the present study exhibit low values ($p > 0.05$) indicating the absence of assortative mating for these traits. All the other correlations are positive and significant at the 1% level. There is no striking difference in the correlation values of the four kinds of parent–child relationships, although the father–child correlation is slightly less than mother–child correlation in case of TFRC (MC, $r = 0.47$; FC, $r = 0.46$; see table 2 for explanation of abbreviations). TFRC shows the highest value for both parent–child ($r = 0.46$; $p < 0.01$) as well as midparent–child correlation ($r = 0.65$; $p < 0.01$). Sib–sib correlation is higher than parent–child correlation for both PII (Sib, $r = 0.31$; PC, $r = 0.30$) and AFRC (Sib, $r = 0.39$; PC, $r = 0.36$).

If we compare the correlation values with other previous studies we can see that the values of TFRC are close to the value of Holt (1952) for the British population. The correlation coefficients of present TFRC are slightly higher than those of Loesch (1971), Matsuda (1973) and Mathew (1980), while the present results of PII are slightly lower than those of Loesch (1971). On the other hand for parent–offspring and midparent–offspring combinations, the result of AFRC of the present study does not show higher values than that of Mathew (1980).

Table 4. Correlation coefficients (*r*-values) of PII and AFRC.

	PII				AFRC			
	Loesch (1971)		Present study		Mathew (1980)		Present study	
	n	*r*	*n*	*r*	*n*	*r*	*n*	*r*
HW	186	−0.07	200	0.03^{NS}	100	−0.03	200	-0.04^{NS}
FS	267	0.26	229	0.33*	151	0.35	229	0.41*
FD	287	0.38	195	0.30*	135	0.56	195	0.30*
MS	270	0.37	229	0.26*	151	0.51	229	0.32*
MD	290	0.31	195	0.32*	135	0.41	195	0.40*
FC	–	–	424	0.31*	286	0.42	424	0.36*
MC	–	–	424	0.29*	286	0.47	424	0.36*
PC	1114	0.33	848	0.30*	572	0.44	848	0.36*
MidS	–	–	229	0.41*	151	0.59	229	0.63*
MidD	–	–	195	0.43*	135	0.65	195	0.67*
MidC	552	0.47	424	0.42*	286	0.60	424	0.66*
BB	–	–	152	0.28*	–	–	152	0.36*
BS	–	–	240	0.30*	–	–	240	0.39*
SS	–	–	110	0.32*	–	–	110	0.42*
Sib	1342	0.33	502	0.31*	371	0.40	502	0.39*

n, number of pair.
$^{NS}p > 0.05$. $*p < 0.01$.
–, not available in the literature.

The results of the heritability estimates of these traits have been presented in table 5. Parent–offspring regression gives heritabilities ranging between 0.61 and 0.74, whereas those from sibling vary between 0.62 and 0.90. All traits show that the heritability coefficients estimated from sibling are higher than the heritability from midparent–child as well as parent–offspring regression; on the other hand, heritability estimates from midparent–offspring regressions are lower in magnitude than from single-parent offspring. The highest heritability is observed in TFRC (mean $h^2 = 0.77$) followed by AFRC (mean $h^2 = 0.71$), while PII has the lowest heritability (mean $h^2 = 0.59$).

The heritabilities of TFRC and AFRC of the present study are slightly lower than that of Hreczko and Ray (1985) on another Hindu population of West Bengal, India. The heritability of PII of the present sample is higher for parent–child and sib–sib but lower for midparent–child than the heritability calculated by Das Chaudhuri and Chopra (1983) (PC, $h^2 = 0.52$; Sib, $h^2 = 0.56$; MidC, $h^2 = 0.65$).

The segregation analysis for the studied traits is presented in table 6. For each trait, maximum likelihood estimates of the model parameters, corresponding $2\ln L$ values and respective χ^2 values with their degrees of freedom are given in the table. The transmission probabilities of the general model did not differ significantly from the expected Mendelian probabilities. For all traits, first the hypotheses of 'no intergenerational transmission' (model 2) was tested, which was rejected in 1% level ($\chi^2 > 11.3$). The environmental model (model 3) is also much less likely than the general model, since all χ^2 values exceeded the critical value of 9.21 for 2 degrees of freedom ($p = 0.01$). The rejection of model 4 (at 5% level) does not support the 'non-transmission of MG' on these traits. 'No polygenic component' or model 6 ($\chi^2 > 6.63$) was also rejected at the 1% level. According to this result, the hypothesis of the MG effect of the analysed dermatoglyphic traits can be accepted, as for the

Table 5. Heritability coefficients (h^2) of finger dermatoglyphics.

	PII				TFRC				AFRC			
	Das Choudhuri and Chopra (1983)		Present study		Hreczko and Ray (1985)		Present study		Hreczko and Ray (1985)		Present study	
Combination	n	h^2	n	h^2	n	h^2	n	h^2	n	h^2	n	h^2
FS	–	–	229	0.66	124	0.81	229	0.82	124	1.00	229	0.80
FD	–	–	195	0.61	115	0.90	195	0.75	115	0.91	195	0.62
MS	–	–	229	0.54	144	0.90	229	0.69	144	0.75	229	0.67
MD	–	–	195	0.62	133	0.89	195	0.75	133	0.85	195	0.77
FC	–	–	424	0.64	148	0.94	424	0.76	148	1.02	424	0.72
MC	–	–	424	0.57	176	0.94	424	0.75	176	0.85	424	0.72
PC	–	0.52	848	0.61	–	–	848	0.74	–	–	848	0.72
MidS	–	–	229	0.52	–	–	229	0.66	–	–	229	0.63
MidD	–	–	195	0.52	–	–	195	0.60	–	–	195	0.60
MidC	–	0.65	424	0.53	140	0.72	424	0.64	140	0.70	424	0.61
BB	–	–	152	0.56	123	0.80	152	0.88	123	0.83	152	0.72
BS	–	–	240	0.60	86	1.00	240	0.90	86	0.87	240	0.78
SS	–	–	110	0.64	40	0.96	110	0.92	40	1.01	110	0.84
Sib	–	0.56	502	0.62	249	0.92	502	0.90	249	0.88	502	0.78
Mean h^2	–	–	–	0.59	–	–	–	0.77	–	–	–	0.71

n, number of pairs.
–, not available in the literature.

Table 6. Segregation analysis of PII, TFRC and AFRC.

Parameter	General 1	Sporadic 2	Environmental 3	τ's equal to p 4	Mendelian 5	No polygenic component 6	MP Mendelian 7
PII							
p	0.339	0.645	0.498	0.648	0.158	0.442	0.262 ± 0.012
τ_1	0.924	0.428	0.526	0.648†	[1.00]	0.819	[1.00]
τ_2	0.531	0.428†	0.526†	0.648†	[0.50]	0.398	[0.50]
τ_3	0.027	0.428†	0.526†	0.648†	[0.00]	0.120	[0.00]
μ_1	−0.829	0.672	−0.206	0.423	−0.299	0.147	-0.287 ± 0.018
μ_2	−0.619	−0.669	0.552	0.629	0.564	0.852	−0.287†#
μ_3	0.744	0.537	−0.902	0.531	0.392	−0.931	0.346 ± 0.026
σ_1	0.706	0.889	0.608	0.634	0.694	0.585	0.452 ± 0.009
σ_2	0.683	0.591	0.977	0.527	0.504	0.566	0.531 ± 0.031
σ_3	0.892	0.899	0.582	0.802	0.101	0.485	0.210 ± 0.023
h^2	0.891	[0.00]	0.880	0.693	0.731	[0.00]	0.742 ± 0.034
−2 ln L	2157.042	2174.185	2171.889	2220.041	2160.912	2214.817	2161.325
χ^2(model 1)	–	17.143 (3)**	14.847 (2)**	11.932(3)**	3.870 (3)^{NS}	57.775(1)**	4.283 (4) ^{NS}
χ^2(model 5)	–	–	–		–	–	0.413 (1) ^{NS}
TFRC							
p	0.632	0.574	0.369	0.431	0.787	0.584	0.528 ± 0.21
τ_1	0.878	0.533	0.468	0.431†	[1.00]	0.615	[1.00]
τ_2	0.465	0.533†	0.468†	0.431†	[0.50]	0.521	[0.50]
τ_3	0.135	0.533†	0.468†	0.431†	[0.00]	0.563	[0.00]
μ_1	0.645	0.513	0.561	0.422	0.354	−0.354	0.787 ± 0.02
μ_2	−0.574	−0.348	−0.523	0.349	−0.756	−0.206	0.560‡

μ_3	0.289	−0.229	0.423	0.210	0.256	0.350	0.332±0.08
σ_1	0.862	0.765	0.935	0.613	0.568	0.429	0.521±0.03
σ_2	0.632	0.418	0.622	0.335	0.589	0.542	0.533±0.04
σ_3	0.815	0.437	0.957	0.326	0.612	0.765	0.592±0.07
h^2	0.823	[0.00]	0.838	0.627	0.732	[0.00]	0.715±0.13
−2 ln L	2135.251	2168.835	2146.615	2145.875	2139.572	2163.605	2140.231
χ^2(model 1)	–	33.584 (3)**	11.364 (2)**	10.624 (3)*	4.321 (3)^{NS}	28.354 (1)**	4.980 (4)^{NS}
χ^2(model 5)	–	–	–		–	–	0.659 (1)^{NS}
AFRC							
p	0.622	0.523	0.329	0.648	0.506	0.520	0.521±0.09
τ_1	0.901	0.518	0.496	0.648†	[1.00]	0.849	[1.00]
τ_2	0.589	0.518†	0.496†	0.648†	[0.50]	0.521	[0.50]
τ_3	0.156	0.518†	0.496†	0.648†	[0.00]	0.263	[0.00]
μ_1	0.682	0.518	0.529	0.423	0.362	0.350	0.232±0.06
μ_2	−0.581	−0.341	−0.511	0.629	−0.722	−0.211	0.447‡
μ_3	0.237	0.583	0.467	0.531	0.260	0.315	0.661±0.08
σ_1	0.850	0.718	0.826	0.632	0.538	0.409	0.621±0.09
σ_2	0.619	0.629	0.762	0.527	0.519	0.532	0.710±0.11
σ_3	0.882	0.492	0.918	0.802	0.629	0.717	0.532±0.08
h^2	0.702	[0.00]	0.597	0.693	0.533	[0.00]	0.719±0.13
−2 ln L	2208.109	2267.316	2221.323	2220.041	2215.079	2284.100	2216.002
χ^2(model 1)	–	59.207(3)**	13.214(2)**	11.932(3)*	6.970(3)^{NS}	75.991(1)**	7.893(4)^{NS}
χ^2(model 5)	–	–	–			–	0.923(1) ^{NS}

MP, Most parsimonious; n, number of degrees of freedom; [ ], parameter is fixed at given value.
†Parameter constrained to equal parameter value listed above it.
‡Model is additive.
#Model is dominant.
$^{NS}p>0.05$, $*p<0.05$, $**p<0.01$.

Mendelian model, $\chi^2 < 7.81$ (d.f. $= 3$, $p > 0.05$) and for the environmental model, $\chi^2 > 9.21$ (d.f. $= 2$, $p < 0.01$).

As the result indicates that the dermatoglyphic phenotypes are under the possible control of MG, we tested additional hypotheses by constructing the most parsimonious Mendelian model to understand the mode of inheritance of the MG effect (only most parsimonious one is given in the table). Constraining model 5, with dominant, additive and recessive sub-models, the trait PII demonstrated that only dominant was compatible with the general and Mendelian models, and both additive and recessive sub-models were rejected at 1% level. But the most parsimonious Mendelian model for two ridge counts showed additive interaction between the major gene alleles ($p > 0.05$).

The MP model explains about 25%, 29% and 44% of the total variation in PII, TFRC and AFRC, respectively. The contribution of MG effect estimated from these models are 10% for PII, 3% for TFRC and 2% for AFRC. The models also show that the frequency of the alleles determining PII, TFRC and AFRC to be 26.2%, 52.8% and 52.1%, respectively.

5. Discussion

The examination of correlation coefficients among relatives suggests that dermatoglyphic traits differ in the extent of the genetic determination. The values are least in PII for most of the pair of relationships and highest in TFRC for all combinations. Using a number of finger and palmar traits, Loesch (1971) also observed that TFRC has the highest correlation figure. The theoretical correlations for an additive metrical character are: sib–sib, $r = 0.5$; parent–child, $r = 0.5$ and midparent–child, $r = 0.71$ (Fischer 1918). In case of TFRC, the observed correlation coefficients do not significantly deviate from the expected values for all combinations suggesting additive inheritance without dominance, which has been taken for granted by many authors (Holt 1955, 1956). The present values of TFRC are slightly higher than those of Loesch (1971), Matsuda (1973) and Mathew (1980), which may be attributed to the lower homogeneity of the present sample.

The values of PII exhibit much lower correlation coefficients than expected, and were also lower than the values estimated by Loesch (1971). The result of correlation values of different combinations of relatives may suggest the involvement of dominance in the expression of alleles, as dominance depresses the coefficient from the theoretical expectation (Hreczko and Ray 1985) and reduces the parent–child correlation to a much greater extent than it does the sib–sib correlation (Matsuda 1973). Another previous investigation (Mukherjee 1966) on a Bengali sample obtained from Calcutta, India also revealed the evidence of dominance of genes responsible for the expression of PII. The additive character of TFRC and dominant nature of PII, as indicated by correlation values of the present study, do not contradict with the result of segregation analysis.

Mather and Jinks (1963) studied the influence of one X-linked locus on the familial correlations and established the hypothesis: $r_{SS} > r_{BB} > r_{BS}$ and $r_{FD} = r_{MS} > r_{MD} > r_{FS}$. The present study does not support the hypothesis of the presence of X-linked loci as most are in contradiction with it. For instance, mother–daughter correlation is higher than father-daughter and brother–brother correlation is lower than brother–sister. Negative or very low husband–wife correlations for different dermatoglyphic traits suggest that assortative mating for these traits were either absent or too few to affect the intra-familial correlations.

However, both the ridge counts have a heritability of more than 70%, which suggests that these traits are greatly influenced by genetic factors with slight influence of environmental factors. But the heritability coefficients of TFRC of the present study is slightly higher than the study of Loesch (1971) ($h^2 = 0.70$). On the other hand, heritability coefficients of TFRC obtained by Mathew (1980) on Telaganya Brahmans of Andhra Pradesh, India are higher (90%) than the values estimated in the present study. These differences may be due to the use of different methodologies in these studies, besides genetic and environmental factors. Using the same method on 100 Andhra families of India, Das Coudhuri and Chopra (1983) calculated the magnitude of heritability of TFRC, which is slightly higher for midparent–child combination ($h^2 = 0.69$) but lower for parent–offspring ($h^2 = 0.72$) and sib–sib combination ($h^2 = 0.74$).

With regard to the problem of mode of inheritance, the results from different studies using familial resemblance are not satisfactory. Using the Polish family material, Loesch (1971) mentioned that PII and other dermatoglyphic traits might be determined by single genes. Holt (1968) explained the inheritance of TFRC by polygenic hypothesis—a series of independent additive genes of equal effect, without dominance and without environmental influence. But contrary to this result, the influence of sex chromosomes on TFRC was referred by other authors (Alter 1965, Penrose 1967). In a study, de Wilde (1967) also disagreed with the additive polygenic inheritance of TFRC. Hreczko and Ray (1985) also suggested the theory of a series of additive genes with small independent effect involving the expression of TFRC and AFRC.

In the present study, the hypothesis denying transmissibility was strongly rejected in segregation analysis, which indicates that the effect is transmitted in the families. Then the other models were tested, and the analyses indicate that the nature of transmission of these traits is not consistent with the hypothesis assuming the proportion of the within-genotype variance attributed to polygenes is equal to zero. The hypothesis containing 'non-transmissibility of MG effect' was also rejected, suggesting the possibility of the influence of MG. When the Mendelian hypothesis was tested, the mode of inheritance was found to be compatible with MG influence, though the proportion of the variance attributable to MG effect is very low. In the present study, heritability of these traits ranges between 0.59 and 0.77, while in the MP model total variance ranges only 25–44%. This is because of the fact that h^2 expresses the proportion of the total variance attributable to average effect of genes which determines the degree of resemblance between relatives, whereas total variance of the MP model represents the variance attributable to the genetic factors incorporated only into this particular model.

However, the present study probably represents the first application of genetic model test on finger ridge counts that employs family data. For this reason, we could not properly compare the present result with earlier studies. But the influence of MG effects on other quantitative traits like anthropometry has already been reported (Ginsburg *et al.* 1999, Olson *et al.* 2001), though the MG effect on anthropometric traits obtained in these studies is higher than the present study.

Our results suggest that the best fitting model for the dermatoglyphic traits as judges by likelihood ratio test includes Mendelian transmission with either additive (TFRC and AFRC) or dominant (PII) major gene effect. But it is true that statistical analysis cannot give any positive proof of a hypothesis, it can only state that the hypothesis cannot be rejected. Thus, though the model of major gene was accepted

in the present study, the acceptance of this model on different samples may provide the evidence of its existence in the studied traits. Such evidence has come through the studies of Jantz (1977), in which the relationships between the mean of TFRC and its variability was examined to detect the nature of gene. Later on it was supported by Reddy and Malhotra (1985) who employed 100 populations from the Indian sub-continent. Spence *et al.* (1973) also reported the evidence of major gene effect on AFRC. The application of segregation analysis on other ethnic population is still required to confirm the validity and consistency of the present findings.

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Zusammenfassung. *Hintergrund:* Es ist wohlbekannt, dass Dermatoglyphen genetisch determiniert sind. Aber wenige Studien haben sich bisher mit dem Erbgang von Dermatoglyphen beschäftigt. Darüber hinaus hat keine der vorangegangenen Untersuchungen, obgleich verschiedene moderne Statistikprogramme existieren, eine Technik zur Kurvenanpassung (model-fitting technique) verwendet, um den Vererbungsmodus zu klären. Insofern ist die Genetik von Dermatoglyphen noch unklar.
Ziel: In der vorliegenden Mitteilung ist der Versuch unternommen worden, einige Informationen hinsichtlich Genetik der Fingerdermatoglyphen durch Bestimmung der Ausprägung und hinsichtlich des Erbganges dieser Eigenschaften zusammenzustellen.
Probanden und Methoden: Fingerabdrücke von 824 Personen aus 200 Familien über zwei Generationen wurden in Barasat, Nord 24-Parganas, West Bengalen, gesammelt. Die Untersuchung schloss familiäre Beziehungen zwischen Verwandten ersten Grades und ihre entsprechende Heritabilität ein. Im letzten Schritt erfolgte eine Segregationsanalyse mittels Pedigree Analysis Package (Nachkommen-Analyse-Programmpaket, PAP), um den Vererbungsmodus zu verstehen.
Ergebnisse: Die Hauptbefunde sprachen für folgendes: (a) Familiäre Beziehungen in allen möglichen Verwandtschaftsgraden (außer Beziehungen zwischen Ehepartnern) waren statistisch signifikant und von vergleichbarer Ausprägung. (b) Die entsprechenden Heritabilitäten waren im Bereich zwischen 59% für Pattern Intensity Index (Musterintensitätsindex, PII) und 77% für die absolute Anzahl von Fingerfurchen (total finger rigde count). Diese Werte stimmten mit früher publizierten, denselben Sachverhalt betreffenden Daten überein. (c) Durch die Segregationsanalyse konnten die Modelle: 'Sporadisch', 'Umfeldbedingt', 'Kein wesentlicher genetischer Effekt', und 'Keine polygene Komponente' ausgeschlossen werden ($p < 0{,}05$), und es wurde die Hypothese eines Hauptgeneinflusses (major gene's influence) auf alle in der Untersuchung befindlichen Merkmale akzeptiert, obgleich der Anteil der Varianz eines Hauptgens insgesamt niedrig war. (d) Das einfachste Mendel'sche Modell sprach klar für den Einfluss eines Hauptgens mit dominanten (für PII) und additiven (für zwei Fingerfurchenzählungen) Effekten.

Zusammenfassung: Der vorliegende Bericht unterstützt Beobachtungen, dass ein Hauptgen für dermatoglyphische Merkmale existiert, und dass die Weitergabe dieses Effektes mit einem Mendel'sche Erbgang erklärt werden kann.

Résumé. *Arrière-plan:* Si la détermination génétique des dernatoglyphes est bien établie, peu d'études ont jusqu'à présent porté sur leur modalité héréditaire et de surcroît malgré l'existence de programmes informatiques permettant des analyses statistiques sophistiquées, celles-ci n'ont pas étudié le mode de transmission au moyen de techniques d'ajustement de courbe. La nature génétique des dermatoglyphes n'est donc pas encore claire.
But: Apporter plus d'information concernant la génétique des dermatoglyphes digitaux en estimant la magnitude et le mode de leur transmission.
Sujets et méthodes: Les dermatoglyphes de 824 individus appartenant à 200 familles incluant deux générations, ont été collectés à Barasat dans le Nord 24-Parganas (ouest du Bengale). Cette étude comprend les corrélations familiales entre parents et enfants et les héritabilités correspondantes ainsi que des analyses de ségrégation au moyen du Pedigree Analysis Package (PAP) afin de comprendre le mode de transmission.
Résultats: (a) Les corrélations familiales de toute nature (à l'exception des corrélations entre époux), sont statistiquement significatives et de magnitude comparable. (b) Les héritabilités correspondantes varient de 59% pour l'indice d'Intensité du dessin (IID) à 77% pour le Compte Total de Crêtes Digitales (CTCD). Ces estimations concordent avec les résultats publiés antérieurement sur ce sujet. (c) Les modèles "sporadique", "environnemental", "absence de gène majeur" et "absence de composant polygénique" établis par analyse de ségrégation sont nettement exclus ($p < 0{,}05$) et l'hypothèse d'une influence d'un gène majeur (GM) sur tous les traits étudiés est validée en dépit d'une faible proportion de la variance GM. (d) Le modèle mendélien le plus parcimonieux indique clairement la contribution d'un gène majeur avec des effets dominants pour IID et additifs pour deux comptes de crêtes.
Conclusion: Ce travail indique l'existence d'un gène majeur dans la transmission de ces caractères dermatoglyphiques et montre que la transmission de ses effets est conforme aux règles mendéliennes.

Resumen. *Antecedentes*: Es un hecho bien establecido que los dermatoglifos están determinados genéticamente. Sin embargo, hasta la fecha, son pocos los estudios que han prestado atención al patrón de herencia de los dermatoglifos. Además, a pesar de la existencia de diferentes paquetes estadísticos avanzados, ninguno de los estudios previos ha puesto a punto una técnica de modelos de ajuste para demostrar el modo de herencia. Por tanto, la naturaleza genética de los dermatoglifos aún no está clara.
Objetivo: En esta comunicación se ha intentado proporcionar informacion sobre la genetica de los dermatoglifos digitales, mediante la estimación de la magnitud y modo de herencia de estos rasgos.
Sujetos y Métodos: Se recogieron las huellas digitales de 824 individuos pertenecientes a 200 familias que incluían dos generaciones, en el distrito Norte 24- Parganas de Barasat (Bengala occidental). El estudio incluye correlaciones familiares entre parientes de primer grado y las correspondientes heredabilidades. En la etapa final, se realizaron análisis de segregación con estos datos por medio del Pedigree Análisis Package (PAP), para comprender el modo de herencia.
Resultados: Los principales resultados indicaban lo siguiente: (a) Las correlaciones familiares entre todos los parientes posibles (excepto la correlación entre esposos) eran estadísticamente significativas y de magnitud comparable. (b) Las correspondientes heredabilidades oscilaban entre el 59% para el indice del patrón de intensidad (PII) y el 77% para el número total de crestas digitales (TFRC). Estas estimaciones concordaban con los datos previamente publicados sobre este tema. (c) El análisis de segregación permitió rechazar contundentemente ($p < 0{,}05$) los modelos "esporádico", "ambiental", "sin efecto de un gen mayor" y "sin componente poligénico", y se aceptó la hipótesis de la influencia de un gen mayor (MG) sobre todos los rasgos estudiados, aunque la proporción de varianza del MG era baja. (d) El modelo mendeliano más parsimonioso indicaba claramente la contribución de un gen mayor con efectos dominantes (para PII) y aditivos (para dos crestas).
Conclusión: El presente informe apoya la evidencia de la existencia de un gen mayor sobre estos rasgos dermatoglificos y la transmisión de este efecto es consistente con las leyes mendelianas.