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Inheritance of Dermatoglyphic Asymmetry in 500 Indian Pedigrees: Complex Segregation Analysis

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A B S T R A C T

The major aim of this study is to determine the mode of inheritance of asymmetry of quantitative dermatoglyphic traits based on principal factors through the application of complex segregation (genetic model fitting) analyses on a large ethnically homogeneous sample of 500 Indian pedigrees (2435 individuals) of two generations. By segregation analysis of the traits- PC1_FA both Mendelian and Environmental models were rejected (<0.001) with the General model, i.e. that despite presence of significant inheritance (rejection of Environmental model), the nature of inheritance is more complex, than Mendelian one. Although a little genetic effect was observed due to familial correlations on asymmetry traits, no evidence was found of major gene contribution to be involved, but this does not contradict the notion postulated by several earlier authors[1,2,17] that asymmetry (fluctuating) provides a measure of developmental instability in human.

Key words: *dermatoglyphic asymmetry, segregation analysis, Indian population*

Introduction

It is known fact that dermatoglyphic features are formed before the 19th week of gestation[3] and thereafter are not amenable to change due to age and/or environmental factors. Thus, dermatoglyphic characteristics permanently preserve an earlier stage of fetal development and thus phylogenetically more stable than other biological traits[4,5]. The development of dermatoglyphics is controlled by the genetic factors has long been demonstrated by Galton[6] through his pioneering work. But even after more than a century, little research has been directed toward assessing the genetic component in the development and expression of dermatoglyphic characters. Recently a number of scientists have become interested in studying of bilateral asymmetry of dermatoglyphic traits. Because dermatoglyphic asymmetry has different applied areas: neurology[7], visual search[8], hormone levels[9], disease incidence[10], congenital anomalies[11–14] and population variation[15,16]. However, the actual utility of asymmetry is also limited because of inadequate knowledge of its genetic nature. Therefore, a through understanding of the mode of inheritance of dermatoglyphic asymmetry is essential. The human body exhibits a variety of bilateral asymmetries (differences in the size and /or shape of supposedly identical right and left sided structures). Some of these asymmetries are inborn and others are acquired. In the literature, there are mainly two types of asymmetry namely (1) fluctuating asymmetry (FA), which is the random deviation i.e. irrespective of sign, from perfect bilateral symmetry[17] and (2) directional asymmetry (DA), which reflects a consistent bias of a character toward systematically greater development on one side i.e. considering sign[18]. Asymmetry is considered as a good indicator of overall developmental homeostasis[19]. It is generally believed that genetic information for both sides of an individual is the same[20]. However, an individual's inability to buffer against environmental (intrauterine) and genetic perturbations[21] are the causes of asymmetry (especially FA).

As far as the literature is concerned, few investigators have considered the genetics of asymmetry, on finger pattern types[22], finger ridge counts[23], on the main line index[24]. However, the results of these studies vary widely, some studies implicate an absence of any genetic component[17,25,26]. While on the contrary, slight hereditary components of asymmetry of finger and palmar ridge

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counts have been reported[27–32] and some recent studies[33–35] have found significant heritability values, varying between 20% and 45% respectively. Another source of evidence implying a genetic basis for dermatoglyphic FA comes from well-established differences among populations[36,37]. From the above review of literatures, it is clear that despite some efforts and approaches regarding the genetics of asymmetry of dermatoglyphic traits, this enigma is still unsolved, and thus remains to be explored, probably because of the lack of appropriate statistical applications. Most of the earlier studies are simply based on the application of correlation or regression between relatives. These analyses are very poor to detect the mode of inheritance of a trait. No adequate model of inheritance has yet been established, in spite of rapid progress in statistical analysis as well as widespread availability of computers. A number of program packages[38–46] are now available, which are very useful in performing complex segregation analysis to determine the effect of genes. Some inheritance studies have been published that include segregation analysis on anthropometric traits[44,47–51]; bone mineral density[52–54]; on serological and biochemical markers[55,56] as well as in some other biological areas. Very few studies[16,57–61] on dermatoglyphic traits (general) also include segregation analysis, but hardly available such study on dermatoglyphic asymmetry[2]. Thus, a model of inheritance has yet to be established to resolve the existing inconsistencies in the literature to understand the nature of genetic and environmental bases of dermatoglyphic asymmetry. The main goal of the present communication is not only to estimate the resemblance between relatives, but also to evaluate the mode of inheritance, which represents the causal factors presumed to be operating on the dermatoglyphic asymmetry by the use of maximum likelihood- based complex segregation analyses.

Materials and Methods

Study population

The 500 pedigrees including two generations, consisting of 2435 individuals were collected from five populations (Table 1), residing in the rural areas of Howrah and Midnapore districts of West Bengal (Figure 1).

The characteristics of all five populations are: (1) Bengali-speaking; (2) practices monogamy and strictly endogamous (without inter-caste marriage); (3) demographically stable family structure with traditional relations between family members; (4) living under similar environmental conditions for the last several and (5) maintain a common gene pool through endogamy. The five populations with the status in the society according to Indian caste hierarchy are: Brahmin (Rarhi), a group at the top in the society in whose traditional job was priesthood; Mahisya, a group having middle caste as a large cultivating group in West Bengal; Padmaraj, who belong to scheduled caste with very low status in the society, their main job is agriculture; Muslim (Sunni), who belong to religious communities, their main job is architectural work and agriculture; and Lodha is a small hunting-gathering tribal group. For further details see Karmakar et al.[60].

Fig. 1. Map showing studied area.

Furthermore, genetic variations among endogamous castes and tribes from West Bengal (including these populations) were studied[62,63] based on serological and biochemical markers. Chakraborty et al.[63] strongly suggested that the constituent genetic profile of any given population does not always correspond exactly to its present social ranking, since some low-caste groups are

TABLE 1
STUDY POPULATION

Population	Abbreviation	Caste hierarchy	No of family	Total individuals
Brahmin (Rarhi)	BR	High	100	449
Mahishya	MA	Middle	100	504
Padmaraj	PA	Low	100	525
Muslim (Sunni)	MU	Religious	100	555
Lodha	LO	Tribe	100	402
Total			500	2435

seen to have stronger genetic affiliation with high ranking groups, instead of being close to groups of their own rank. The present caste hierarchy, therefore, may not be the reflection of the genetic origin of the populations. In this context, the present chosen populations may provide a good opportunity for complex segregation analysis.

Pooled data

In our previous study[64], on the same sample, we observed the nature of transmission and trait variance, which revealed the existence of a common homogeneous dermatoglyphic trait in five populations irrespective of different ethnic and geographic backgrounds. Considering this remarkable similarity as well as the population characteristics with respect to dermatoglyphic trait variation from the Mantel test, we can assume that it is possible to combine pedigrees of five populations for further segregation analysis to obtain better results from this pooled data. Therefore, for the present study to reduce the number of statistical tests in the subsequent analyses (not shown), and to substantially increase the overall power of the study, the data were standardized in each population separately and then combined to form the joint data sample.

Data collection

The data were not collected by a random sampling method; rather each pedigree was specially chosen to have a pure caste descent with living parents and at least two children were included. Thus the data have a limitation; the selected families do not represent the five entire population groups of West Bengal. Since the major objective of the present study is to determine the mode of inheritance of dermatoglyphic traits, reliable family data on any pure endogamous (without inter-caste marriage) population is especially necessary. To obtain such genuine family data and to avoid inter-observer variations, the first author collected the entire data set by herself.

Print analysis and variable used

Dermatoglyphic prints were collected following widely used rolled print (inked) method[65] and dermatoglyphic traits were mostly evaluated by the methods[65,66]. In order to avoid any inter-observer error, the first author alone analyzed the whole dermatoglyphic prints of 2435 individuals. In the present report 25 asymmetry traits were used namely 12 directional asymmetry (DA) and 13 fluctuating asymmetry (FA). The dermatoglyphic variables are presented in Appendix 1 (22 quantitative traits and 11 indices of diversity traits were excluded for the present study) and the formulae for calculating various indices are in Appendix 2.

Statistical Analyses

Z-transformation

Each quantitative dermatoglyphic traits was converted to normalize the data. The formula is: $Z=(Xi–X)/SD$, where Xi, X, and SD are the individual's measurements, average and standard deviation for the trait respectively. The transformed score has a mean of zero and a standard deviation of one. All further calculations are based on these transformed Z-scores.

Principal component analysis (PCA)

PCA were performed using STATISTICA version 6 software (Stat Soft 2001). To avoid the problem of multiple comparisons, redundancy of information, and repetition of measurement error, we performed principal component analysis (PCA) using the original traits (FA, and DA) regardless of the sex and age of the individual to capture as much common variation as possible. The eigenvalue >1 criterion was used to extract factors for FA, DA trait groups (Varimax rotation).

Homogeneity test

Estimation of homogeneity of the total sample, constructed from the representatives of the five populations, was checked by Univariate analysis of ANOVA. Age, sex, and population are the independent variables, and effects of their interactions are reflected on dermatoglyphic variables (dependent variables). We used this analysis for the checking of the homogeneity of five variables between the five populations. In order the complex segregation analyses can be performed on the total sample, initial traits should be adjusted on significant covariates.

Familial correlations

To examine the potential familial aggregation of the factor scores, we have done two types of intra-familial correlations: (a) inter-class and (b) intra-class. Correlations between spouses and between parents and offspring as inter-class were computed by the Pearson product--moment correlations method. The correlations between siblings as intra-class were computed using the Package MAN-5 version[45].

Segregation analysis (genetic model tests)

Complex segregation analysis was carried out following Maximum Likelihood Methods by using the Package MAN-5 version[45] to evaluate the mode of inheritance. This program estimates the following parameters: p is the population frequency of the first of the two major alleles, A_1 and A_2, μ_g is the average trait value (genotype value) in all individuals having genotype g; g=1, 2, and 3 corresponds to genotypes A_1A_1, A_1A_2 and A_2A_2, respectively. The value σ^2_g is the trait variance in individuals having the same MG genotype *g*; it estimates the trait variability resulting from all possible environmental factors and minor genes influencing the trait value; ρ, β, and ε represent the partial correlation coefficients of non-MG residual of the trait between spouses, between parents and offspring, and between siblings, respectively. Correlation ρ is due to common environmental factors shared by spouses, whereas the two other correlations can be caused both by the corresponding environmental factors and by minor genes affecting the trait, which are unidentified in the model (for details, see Karmakar et

al.[64]). No ascertainment correction of likelihood was made because our method of the pedigree collection was not connected with the individual's dermatoglyphic characters. The following genetic models have been tested:

1. The General model (Free) 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. The Mendelian model (Mixed) assumes Mendelian transmission with the assumption of the Hardy-Weinberg equilibrium; the probabilities of three putative genotypes in the populations are p^2, 2p (1–p), and $(1–p)^2$. The transmission probabilities of allele A_1 by the above corresponding genotypes are $\tau_1=1.0$, $\tau_2=0.5$, $\tau_3=0.0$, respectively.

3. The Environmental model assumes independence of offspring genotypes from the parental genotypes. Since the selective effect of the three genotypes on the trait variation is not assumed, then $\tau_1=\tau_2=\tau_3$.

Hypotheses 2–3 are the sub-models of the general model and thus were compared with this model. The differences in the log-likelihood values (LH) were distributed as χ^2 and the degrees of freedom (df) depend on the number of constraints imposed by the model. Since the method of pedigree collection for this study was in no way connected with the individual's dermatoglyphic traits, no ascertainment correction of likelihood was made.

Results

Principal component analysis (PCA)

A clear separation of DA traits into 3 factors is easily interpretable in Table 2, which jointly accounted for more than 51% of the total variation.

Factor 1 alone accounted for about 27% of the total variation, whereas factor 2 and factor 3 explain approximately 12% each. Table 3 described the FA traits into 2 factors, which jointly accounted for more than 40% of the total variation. Factor 1 accounted for about 29% of the total variation, whereas factor 2 explains approximately 11%.

TABLE 2
UNROTATED LOADINGS OF 9 DA TRAITS FOR 3 PRINCIPAL COMPONENTS

Variables	Factor 1	Factor 2	Factor 3
DA 2	0.551	0.345	–0.361
DA 3	–0.029	–0.564	–0.481
DA 4	0.933	–0.107	0.122
DA 7	0.008	0.648	0.301
DA 10	0.440	–0.198	0.408
DA 11	0.441	–0.352	0.444
DA 12	0.552	–0.140	0.007
DA 13	0.529	0.137	–0.511
DA 14	0.509	0.266	–0.079
Eigen value	2.407	1.141	1.099
V.P.	0.267	0.127	0.122
C.V	26.75%	39.42%	51.64%

V.P, Variance explained by factor; C.V, cumulative proportion of the total variance

TABLE 3
UNROTATED LOADINGS OF 9 FA TRAITS FOR 2 PRINCIPAL COMPONENTS

Variables	Factor 1	Factor 2
FA 2	0.529	–0.219
FA 3	–0.022	–0.589
FA 4	0.808	0.054
FA 7	0.015	–0.731
FA 10	0.484	0.283
FA 11	0.610	0.057
FA 12	0.635	0.011
FA 13	0.639	–0.030
FA 14	0.514	–0.174
Eigen value	2.407	1.047
V.P.	0.291	0.116
C.V	29.06%	40.69%

V.P, Variance explained by factor; C.V, cumulative proportion of the total variance

Homogeneity test

The F-values revealed a non-significant interaction between sex and population covariates in PC1_DA, while all single covariates were significant for PC1_FA. For PC1_DA – age and sex covariates were non-significant, but population was significant (Table 4). The univariate test for adjustment is necessary, where the influence of age, sex, and population are significant.

Familial correlations

Table 5 provides correlations between spouses, between parents and offspring, and between siblings based on three factors and two individual traits.

The correlations between spouses are low values; some are even negative and non-significant ($p > 0.05$). All the other correlations are positive and significant at the 1% for PC1_FA and not significant for PC1_DA.

Segregation analysis

In the present study, segregation analysis was done for PC1_ FA trait, because no significant familial correlations were observed for the trait PC1_DA and thus was excluded from this analysis. Three genetic models were tested: General, Mendelian and Environmental and the results are presented in Table 6.

The table presented maximum likelihood estimates (LH), respective χ^2 values with their degrees of freedom, and the model parameters: p – frequency of A_1 allele; μ_1 – genotypic values for genotype A_1A_1; μ_2 – genotypic values

TABLE 4
HOMOGENEITY TEST OF PC1_DA AND PC1_FA VARIABLES IN 500 PEDIGREES

PC1_DA	SS	DF	MS	F	P
Intercept	0.16	1	0.16	0.16	0.686
Age	0.53	1	0.53	0.54	0.464
Sex	2.37	1	2.37	2.38	0.123
Population	15.11	4	3.78	3.80	0.004
Sex*Population	2.90	4	0.73	0.73	0.572
Error	2177.00	2188	0.99		
PC1_FA	SS	df	MS	F	p
Intercept	4.10	1	4.10	4.15	0.042
Age	4.82	1	4.82	4.89	0.027
Sex	15.02	1	15.02	15.22	0.000
Population	15.29	4	3.82	3.87	0.004
Sex*Population	2.21	4	0.55	0.56	0.691
Error	2159.43	2188	0.99		

TABLE 5
FAMILIAL CORRELATIONS OF 2 NORMALIZED VARIABLES PC1_DA AND PC1_FA

Relationship		PC1_DA	PC1_FA
Spouses	r	0.078	–0.034
	N	402	400
	p	0.122	0.509
Parent-Offspring	r	0.033	0.136
	N	2320	2302
	p	0.110	0.001
Sib Pairs	r	0.017	0.129
	N	1300	1284
	p	0.563	0.001

for genotype A_1A_2; μ_3 – genotypic values for genotype A_2A_2; σ^2 – variance of genotypic values; β,ε – partial residual correlations for parent-offspring and siblings; τ_1, τ_2, τ_3 – probabilities of transmitting allele A_1 to offspring from parents showing genotype A_1A_1, A_1A_2, A_2A_2, correspondingly.

As the first step of analysis, two models- Mendelian and Environmental were compared with the General model for four traits. The transmission probabilities of the General model differ significantly from the expected Mendelian probabilities (PC1_FA χ^2>61.30, df=3, p< 0.001) as well as from Environmental model (PC1_FA χ^2>25.60, df=3, p<0.001) i.e. both of these models were rejected. Thus, the present result of the population failed to provide evidence in support of major gene effect of FA traits. The fact that the model with equal τ (Environmental model) was rejected means that there exists some type of genetic factors, but the inheritance pattern is more complex, than the Mendelian one.

TABLE 6
SEGREGATION ANALYSIS OF TRAIT PC1_FA

Parameter	General	Mendelian	Environmental
p	0.597±0.050	0.430	0.605
μ_1	–0.699±0.042	–0.796	–0.698
μ_2	0.086±0.122	–0.289	0.038
μ_3	1.758±0.233	0.990	1.603
σ_1^2	0.124±0.016	0.095	0.122
σ_2^2	0.424±0.059	0.290	0.421
σ_3^2	0.973±0.154	1.251	1.087
β	0.100±0.031	–0.019	0.139
ε	0.167±0.015	0.066	0.144
τ_1	0.758±0.058	[1]	0.605!
τ_2	0.636±0.048	[0.5]	0.605!
τ_3	0.398±0.070	[0]	0.605!
LH	–2651.1	–2681.7	–2663.9
χ^2		61.3	25.6
d.f.		3	3
p		<0.001	<0.001

Parameter constraints: ! – Parameter is equal to the parameter above; [] – parameter was fixed to the specified value

Discussion

Unfortunately, the existing information regarding mode of inheritance by the genetic model-fitting test especially on asymmetry is very limited and thus we are unable to provide an accurate explanation compared with such studies in other populations. However, we will try to describe on the following headlines.

Principal component analysis

In order to reduce the number of interrelated variables to a few factors, we performed a principal compo-

nent analysis on the studied asymmetric traits. Froehlich [67] suggested that the factors give a clearer picture than the traditional variables do. Thus the application of factor analysis is not new in the study of dermatoglyphic asymmetry and diversity[2,15,68–72]. In the present study, the DA and FA of dermatoglyphic traits were clearly separated and these results are perfectly corroborated with earlier studies[2,68,69].

Familial correlations

Departures from random mating and consanguinity lead to changes in the correlation coefficient. The spouse correlation in the present study indicates the absence of any assortative matting for asymmetry traits. Our present results of asymmetric traits are similar with previous study Sengupta and Karmakar[2]. Absence of assortative matting on the asymmetry of qualitative dermatoglyphic traits has been reported in other studies[22,73]. Parent-offspring correlations in five investigated traits are low but positive and significantly different, which suggests the contribution of some genetic factors to bilateral asymmetry. This finding supports earlier works[2,17,31,35,74]. However, it is well known that in the absence of environment effects when additive genes with independent effects without dominance are present, the correlation is 0.5 for the parent-child and sib-sib pairs Fisher[75]. The strength of correlation of both DA and FA in the present investigation is much lower than the theoretical value, indicating that along with the genetic component, environmental (intrauterine) factors are considerable. The results do not contradict the previous hypothesis that, although there is a genetic component to dermatoglyphic asymmetry, a principal role can be attributed to exogenous factors[30,76]. Our results are also consistent with Martin et al.[31] suggested that there exits a genetic component in asymmetry variation between hands but environmental factors are more important. The correlation coefficients for three traits (DA and FA) varied from one trait to another, suggesting that the influence of genetic effect on asymmetry might differ with respect to examined traits. In a previous study Karev[77] suggested that any general buffering capacity is apparently absent not only for different traits but also in correlated traits, such as finger ridge counts. The present results are also consistent with the idea that genetic contribution is specific to different areas of the finger and palm Malhotra et al.[78]. This similarity between general dermatoglyphic traits and their bilateral asymmetry is again compatible with the suggestion of Jantz[36] that the genetic mechanisms responsible for ridge counts may also mediate their bilateral asymmetry. The sib-sib correlations in the present study are lower: PC1_DA (0.017) and PC1_FA (0.129) than parent-offspring PC1_DA (0.033) and PC1_FA (0.136), which strongly indicate that the dominance cannot be established. It may be due to environmental factors rather than dominance.

Segregation analysis

The family resemblance may be due to major gene (i.e. single gene having a large influence), polygenes (i.e. many genes, each with a small effect), and common (shared) familial environment, or interaction among these causes Feitosa et al.[79]. Maximum likelihood method in segregation analysis allows us to distinguish between the components of genetic and family environment. Therefore, segregation analyses give clearer picture of the pattern of inheritance than earlier traditional methods of correlation and heritability estimation. The goal of this present report was therefore, the use of family data to identify Mendelian mechanisms with respect to asymmetry and diversity of dermatoglyphic traits. Two traditional criteria are required to derive a major gene effect. (1) The environmental hypothesis must be rejected with a chi-square test $p<0.05$, indicating that the general model fits better than the environmental hypothesis. (2) The most general Mendelian hypothesis, must be accepted with a chi-square test $p>0.05$ indicating that the general model does not fit significantly better than Mendelian hypothesis. For the present population both of these Mendelian and Environmental models were strongly rejected for all the four traits. Or in other words, the transmission probabilities of general model differ significantly from the expected Mendelian probability i.e. that despite presence of significant inheritance (rejection of Environmental model), the nature of inheritance is more complex, than Mendelian one.

To our knowledge, there is no any single study (except only, Sengupta and Karmakar[2]) dealing with segregation analysis with genetic model fittings of dermatoglyphic asymmetry both in India as well as in abroad. Thus, due to lack of literature the present result could not be compared properly with the earlier studies. Still an attempt is made to view the present result in the light of some hypotheses postulated by different authors on dermatoglyphic asymmetry based on the traditional analyses. The present result does not fully contradict with the results based on dermatoglyphic asymmetry of the previous study (Sengupta and Karmakar[2]) on an Indian population. In this study, Mendelian model was rejected but Environmental model was accepted, which suggested that the inheritance pattern does not appear to be consistent with Mendelian transmission. However, the discrepancy of our present results with this study may be due to different sets of variables composed the factors of dermatoglyphic asymmetry. Our results are similar to Holt[80] on diversity of finger ridge counts (twin data), suggested as being under genetic control but with the absence of genetic causes of asymmetry based on correlation analysis. Thus we can point out that asymmetry of dermatoglyphics are influenced by considerable amount of intra-uterine environmental influence, which do not contradict the earlier ideas postulated by several authors[1,2,17] that asymmetry (fluctuating) provides a measure of developmental instability in human. Further, the present results strongly suggest that there is no variation between individual trait and its factor with respect to the mode of inheritance, which may be due to the involvement of the same genetic component Karmakar et al.[61]

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REFERENCES

1. ROSE RJ, REED T, BOGLE A, Behav Genet, 17 (1987) 125. — 2. SENGUPTA M, KARMAKAR B, Hum Biol, 78 (2006) 199. — 3. PENROSE LS, OHARA PT, J Med Genet, 10 (1973) 20. — 4. ROTHHAMMER F, CHAKRABORTY R, LIOP E, Am J Phys Anthrop, 46 (1977) 51. — 5. FROEHLICH JW, GILES E, Am J Phys Anthrop, 54 (1981) 93. — 6. GALTON F, Finger Prints (Macmillan and Co, London, 1892). — 7. VERENICH SV, Ontogenez, 27 (1996) 137. — 8. NICHOLLS ME, SEARLE DA, BRADSHAW JL, Psychol Sci, 15 (2004) 138. — 9. SORENSON JC, JAMISON C, MEIER RJ, CAMPBELL BC, Am J Phys Anthropol, 90 (1993) 185. — 10. TOTH C, RAJPUT M, RAJPUT AH, Mov Disord, 19 (2004) 151. — 11. KOBYLIANSKY E, BEJERANO M, VAINDER M, KATZNELSON MB, Anthropol Anz, 55 (1997) 315. — 12. KOBYLIANSKY E, BEJERANO M, YEAKOVENKO K, KATZNELSON MB, Coll Antropol, 23 (1999) 1. — 13. KATZNELSON MB, BEJERANO M, YAKOVENKO K, KOBYLIANSKY E, Anthropol Anz, 57 (1999) 193. — 14. BEJERANO M, YAKOVENKO K, KATZNELSON MB, KOBYLIANSKY E, Z Morphol Anthropol, 83 (2001) 75. — 15. KARMAKAR B, YAKOVENKO K, KOBILIANSKY E, Coll Anthropol, 25 (2001) 167. — 16. SENGUPTA M, KARMAKAR B, Ann Hum Biol, 31 (2004) 526. — 17. ARRIETA MI, CRIADO B, MARTINEZ B, LOBATO N, Ann Hum Biol, 20 (1993) 557. — 18. PALMER AR, STROBECK C, Ann Rev Ecol Syst, 17 (1986) 391. — 19. PALMER AR, STROBECK C, J Ecol Biol, 10 (1997) 39. — 20. POTTER RH, NANCE WE, Am J Phys Anthropol, 44 (1976) 391. — 21. POLAK M, STARMER WT, Int J Org Evol, 55 (2001) 498. — 22. BENER A, Hum Evol, 10 (1981) 387. — 23. LOESCH D, MARTIN NG, Act Anthropogenet, 6 (1982) 85. — 24. KARMAKAR B, Trends in Dermatoglyphic Research. In: DURHAM NM, PLATO CC (Eds) (Kluwer Acad Publ, Netherlands, 1990,). — 25. HOLT SB, Ann Eugen, 18 (1954) 211. — 26. BOGLE AC, REED T, Am J Med Genet, 72 (1997) 379. — 27. SINGH S, Hum Hered, 20 (1970) 403. — 28. TRIMBLE BK, GO RCP, MI MP, RASHAD MN, Hum Genet, (1971) 204. — 29. MI.MP, RASHED MN, Hum Hered, 27 (1977) 273. — 30. JANTZ RL, Birth Defects Orig Artic, 15 (1979) 53. — 31. MARTIN NG, EAVES LJ, LOESCH DZ, Ann Hum Biol, 9 (1982) 539. — 32. POLIUKHOV AM, Genetika, 20 (1984) 1894. — 33. LIVSHITS G, KOBYLIANSKY E, Ann Hum Biol, 16 (1989) 121. — 34. MOLLER AP, THRONHILL R, J Ecol Biol, 10 (1997) 1. — 35. PECHENKINA AE, ROBERT AB, GALINA GV, ANDREY IK, Am J Phys Anthropol, 111 (2000) 531. — 36. JANTZ RL, Am J Phys Anthropol, 42 (1975) 215. — 37. DITTMAR M, Am J Phys Anthropol, 105 (1998) 377. — 38. ELSTON RC, STEWART J, Hum Hered, 21 (1971) 523. — 39. MORTON NE, MACLEAN CJ, Am J Hum Genet, 26 (1974) 489. — 40. LALOUEL JM, Methods in genet Epidemiol. In: MORTON NE, RAO DC, LALOUEL JM (Eds). (Karger, Basel, 1983). — 41. KONIGSBERG LW, KAMMERER CM, MACCLUER JW, Genet Epidemiol, 6 (1989) 713. — 42. MORTON NE, Ann Rev Genet, 27 (1993) 523. — 43. HASSTEDT SJ, PAP: Pedigree analysis package (University of Utah, Salt Lake City, Department of Human Genetics, Medical Center, 1994). — 44. GINSBURG E, LIVSHITS G, Ann Hum Biol, 26 (1999) 103. — 45. MALKIN I, GINSBURG E, MAN-5: Program package for Mendelian analysis of pedigree data (Technical Report, Department of Anatomy and Anthropology, Sackler Faculty of Medicine, Tel Aviv University, Israel, 2002). — 46. MALKIN I, GINSBURG E, Program package for Mendelian analysis of pedigree data MAN, version 6 (Technical Report, Department of Anatomy and Anthropology, Sackler, Faculty of Medicine, Tel Aviv University, Israel, 2003). — 47. RICE T, SJOSTROM CD, PERUSSE L, RAO DC, SJOSTROM L, BOUCHARD CA, Obesity Research, 7 (1999) 246. — 48. LIVSHITS G, YAKOVENKO K, GINSBURG E, KOBYLIANSKY E, Ann Hum Biol, 25 (1998) 221. — 49. GINSBURG E, LIVSHITS G, YAKOVENKO K, KOBYLIANSKY E, Ann Hum Genet, 62 (1998) 307. — 50. ŠKARIĆ-JURIĆ T, GINSBURG E, KOBYLIANSKY E, MALKIN I, SMOLEJ-NARAN⊥IĆ N, RUDAN P, Coll Antropol, 1 (2003) 135. — 51. SENGUPTA M, KARMAKAR B, Am J Hum Biol, 19 (2007) 399. — 52. CARDON LR, GARNER C, BENNETT ST, MACKAY IJ, EDWARDS RM, CORNISH J, HEDGE M, MURRAY MAF, REID IR, CUNDY T, J Bone & Mineral Res, 15 (2000) 1132. — 53. LIVSHITS G, KARASIK D, PAVLOVSKY OM, KOBYLIANSKY E, Hum Biol, 71(1999) 155. — 54. LIVSHITS G, KARASIK D, KOBYLIANSKY E, J Bone & Mineral Res, 17 (2002) 152. — 55. LIVSHITS G, GERBER LM, Hypertension, 37 (2001) 928. — 56. FRIEDLANDER Y, KARK JD, SINNREICH R, BASSO F, HUMPHRIES SE, Ann Hum Genet, 67 (2003) 228. — 57. DARLU P, IAGOLNITZER ER, Ann Hum Biol, 11 (1984) 537. — 58. GILLIGAN SB, BORECKI LB, MATHEW S, MALHOTRA KC, RAO DC, Am J Phys Athrop, 68 (1985) 409. — 59. GILLIGAN SB, BORECKI LB, MATHEW S, VIJAYKUMAR M, MALHOTRA KC, RAO DC, Am J Phys Anthrop, 74 (1987) 117. — 60. KARMAKAR B, MALKIN L, KOBYLIANSKY E, Anthrop Anz, 63 (2005a) 365. — 61. KARMAKAR B, YAKOVENKO K, KOBYLIANSKY E, Am J Hum Biol, 18 (2006) 377. — 62. MUKHERJEE BN, WALTER H, MALHOTRA KC, CHAKRABORTY R, SAUBER P, BANERJEE S, ROY M, Anthropol Anz, 45 (1987) 239. — 63. CHAKRABORTY R, WALTER H, MUKHERJEE BN, MALHOTRA KC, SAUBER P, BANERJEE S, ROY M, Am J Phys Anthropol, 71 (1986) 295. — 64. KARMAKAR B, YAKOVENKO K, KOBYLIANSKY E, Ann Hum Biol, 32 (2005b) 445. — 65. CUMMINS H, MIDLO C, Fingerprints, palms, and soles (Research Publishers, South Berlin, MA, 1976). — 66. HOLT SB, Genetics of dermal ridges (Springfield Charls C, Thomas Publ, 1968). — 67. FROEHLICH JW, The Measures of Man. In: GILLES E, FRIEDLANDER J (Eds) (Peabody Musium Press, Cambridge, 1976). — 68. MICLE S, KOBYLIANSKY E, Hum Biol, 60 (1986) 123. — 69. MICLE S, KOBYLIANSKY E, Homo, 42 (1991) 21. — 70. KARMAKAR B, YAKOVENKO K, KOBYLIANSKY E, Coll Antropol, 32 (2008) 467. — 71. KARMAKAR B, MALKIN I, KOBYLIANSKY E, Anthrop Anz, 67 (2009a) 237. — 72. KARMAKAR B, KOBYLIANSKY E, Anthrop Anz, 67 (2009b) 253. — 73. BENER A, ERK FC, Ann Hum Biol, 6 (1979) 349. — 74. PARSONS PA, Hum Hered, 23 (1973) 226. — 75. FISHER RA, Trans Roy Soc Edinb, 52 (1918) 99. — 76. MALHOTRA KC, Coll Antropol, 11 (1987) 39. — 77. KAREV GB, Anthrop Anz, 46 (1988) 245. — 78. MALHOTRA KC, MAJUMDAR L, REDDY BM, In: REDDY BM, ROY SB, SARKAR BN (Eds) Dermatoglyphics Today (IBRAD, ASI & ISI, Calcutta, 1991). — 79. FEITOSA MF, RICE T, NIRMALA A, RAO DC, Hum Biol, 72 (2000) 781. — 80. HOLT SB, Ann Hum Genet, 24 (1960) 253.

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NASLJEĐIVANJE DERMATOGLIFSKIH ASIMETRIJA NA UZORKU OD 500 INDIJSKIH RODOSLOVLJA: KOMPLEKSNA SEGREGACIJSKA ANALIZA

S A Ž E T A K

Glavni cilj istraživanja bio je utvrditi način nasljeđivanja asimetrija kvantitativnih dermatoglifskih svojstava na temelju glavnih faktora kroz primjenu analize složene segregacije (genetic model fitting) na velikom, etnički homogenom, uzorku od 500 indijskih rodoslovlja (2435 osobe) dvije generacije. Segregacijskom analizom tragova – PC1_FA oba i Mendelov i Okolišni modeli odbačeni su (<0,001) Glavnim modelom, tj. unatoč značajnoj prisutnosti nasljeđa (odbacivanje Okolišnog modela), priroda nasljeđivanja puno je složenija od one mendelovske. Iako je posmatrano malo genetskih učinaka u odnosu na obiteljsku korelaciju asimetrijskih osobina, nema dokaza o uključenju velikog genetskog doprinosa, ali to ne proturječi postavkama ranijih autora[1,2,17] po kojima asimetrija (promjenjiva) pruža mjere razvojnih nestabilnosti kod čovjeka.

Appendix 1: List of the utilized traits and indices

22 quantitative traits	15 Directional Asymmetry (DA) traits
Finger RC, I r	DA I = Div II – Div I
Finger RC, II r	DA II = PII, rh – lh
Finger RC, III r	DA III = a-b RC, r – l
Finger RC, IV r	DA IV = hRC, rh – lh
Finger RC, V r	DA V = S^2, rh – lh
Finger RC, I l	DA VI = Div VIII – Div VII
Finger RC, II l	DA VII = atd angle, r – l
Finger RC, III l	DA VIII = a-b dist., r – l
Finger RC, IV 1	DA IX = ridge breadth, r – l
Finger RC, V 1	DA X = fRC, Vr – Vl
Total Finger RC (TFRC)	DA XI = fRC, IVr – IVl
Absolute Finger RC (AFRC)	DA XII = fRC, IIIr – IIIl
PII, lh	DA XIII = fRC, IIr – IIl
PII, rh	DA XIV = fRC, Ir – Il
PII, both h	DA XV = MLI, rh – lh
a-b RC, rh	16 Fluctuating Asymmetry (FA) traits
a-b RC, lh	FA I = [Div I – Div II]
A-line exit, l	FA II = PII, [rh – lh]
A-line exit, r	FA III = a-b, RC, [rh – lh]
D-line exit, l	FA IV = hRC, [rh – lh]
D-line exit, r	FA V = [Div V – Div IV]
MLI	FA VI = [Div VIII – Div VII]
42 traits (diversity and asymmetry):	FA VII = atd angle, [r – l]
11 Diversity traits (Div)	FA VIII = a-b dist, [r – l]
Div I = max – min fRC (lh)	FA IX = ridge breadth [r-l
Div II = max – min fRC (rh)	FA X = fRC, [Vr – Vl]
Div III = max – min fRC (both h)	FA XI = fRC, [IVr – IVl]
Div IV = S^2 for lh, (or S^2L)	FA XII = fRC, [IIIr – IIIl]
Div V = S^2 for rh, (or S^2R)	FA XIII = fRC, [IIr – IIl]
Div VI = S2 (both h)	FA XIV = fRC, [Ir – Il]
rDiv VII = IIDL (for lh)	FA XV = MLI, [rh – lh]
Div VIII = IIDR (for rh)	FA XVI = A1, asymmetry index
Div IX = S **FORM**, (both h)	22 quantitative traits and 11 indices of diversity traits were excluded in the present study.
Div X = S **FORM**, (both h)	
Div XI = Shannon's index	

Abbreviations: RC = ridge count; r = right; l = left; h = hand; PII – Pattern Intensity Index; MLI = main line index; Div I to Div XI = indices of intra-individual diversity of finger ridge counts; DA I to DA XV = indices of directional asymmetry; FA I to FA XVI = indices of fluctuating asymmetry.

Appendix 2: Formulae for some indices of dermatoglyphic diversity and asymmetry

The directional asymmetry (DA) was computed by the following equation: $DA_{ij}=X_{iR}–X_{iL}$. The fluctuating asymmetry (FA) was computed by using the absolute differences between the bilateral measurements. The distributions of the non-absolute differences for each individual were corrected (Livshits et al., 1988) to avoid addi- tional influences (scaling effects) such as size of the trait or directional asymmetry, yielding the following equation for computing FA:

$$FA_{ij} = (X_{iR} - X_{iL}) - 1 / n \sum_{i=1}^{n} [(X_{iR} - X_{iL})]$$

Where, xi = trait (x) of individual (i); R, L = right and left, n = size of the sample and FA_{ij} is the value of FA of trait (j) in the i-th individual.

Div I, Div II, Div III. Maximal minus minimal finger ridge counts in the five left (Div I), five right (Div II), or in the ten finger ridge counts (Div III). Div IV, Div V $= \sum_{i=1}^{5} q_i^2 - Q^2 / 5$, for the left (Div IV, S^2L), or right fingers (Div V, S^2R); Div VI, $S^2 = \sum_{i=1}^{10} q_i^2 - Q^2 / 10$ Div VII, Div VIII $= \sqrt{\sum_{i=1}^{5} q_i^2 - Q^2 / 5}$, for the left (Div VII, IIDL), or right finger (Div VIII, IIDR); Div IX, $S\sqrt{10} = \sqrt{\sum_{i=1}^{10} (q_i^2 - Q^2 / 10)/ 10}$; Div X, $S\sqrt{5} = \sqrt{\sum_{i=1}^{5} (k_i^2 - Q^2 / 5)/ 5}$.

In these formulae, q_i is the ridge count for the ith finger, Q is the sum of the five finger ridge counts of a hand (Div IV, V, VII, VIII) or of all the ten fingers (Div VI, IX, X), and k is the sum of ridge counts of the ith pairs of homologous right and left fingers.

Div XI. Shannon's index, $D = - \sum_{i=1}^{4} P_i \log P_i$ where P_i is the frequency of each of the four basic finger pattern types on the ten fingers; Abs XVI, $AI = \sqrt{\sum_{i=1}^{5} (R_i - L_i)^2}$, where R_i and L_i are the ridge counts for the ith finger of the right and left hand.