Original Research Article

Mode of Inheritance of Dermatoglyphic Pattern Intensity Index on Fingers in Five Indian Populations: A Comparative Study between Individual Trait and Its Factor

B. KARMAKAR,[1]* K. YAKOVENKO,[2] AND E. KOBYLIANSKY[2]
[1]*Biological Anthropology Unit, Indian Statistical Institute, 203 B.T. Road, Kolkata 700 108, India*
[2]*Department of Anatomy and Anthropology, Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv 69978, Tel Aviv, Israel*

ABSTRACT Our previous study (Karmakar et al. [2005] Ann. Hum. Biol. *32:*445–468) was on 500 pedigrees of five different populations, with factor 1 comprising quantitative finger dermatoglyphics (including pattern intensity index, PII) and factor 1 controlled by major genes. The present results of a complex segregation analysis of the individual trait PII of the same five populations were compared with previous results to ascertain the extent of variation between individual trait PII and its factor (factor 1) with respect to mode of inheritance. The comparative findings are very similar in five populations, irrespective of different ethnic groups. This result suggests that the variability of their biological relevance is influenced by the same genetic component, thus representing a similar mode of inheritance with major gene involvement in all populations. Am. J. Hum. Biol. 18:377–386, 2006.

In a previous study (Karmakar et al., 2005), we used three factors in 500 families extracted from principal components analysis (PCA) of 18 quantitative dermatoglyphic traits. Segregation analysis was applied on these three factors, with a view toward examining the genetic nature/inheritance pattern and extent of variation in five different ethnic groups. The pattern intensity index (PII) for left (PII-L) and right (PII-R) on finger was included in factor 1, and in accordance with their construction, factor 1 covered 12 out of 18 variables, accounting for about 40% of the total variation. The results revealed a significant major gene (MG) effect on factor 1, with two codominant alleles. The trait variance H^2 strongly supports the existence of a common nature of dermatoglyphic trait inheritance in five populations, irrespective of different ethnic and geographic areas.

In terms of the importance of the application of factors from PCA in genetic analysis, we know that "factors may be more general and meaningful anatomically and more specific genetically" (Howells, 1953). Thus a factor pattern may be a more direct representation of underlying gene structure than the original variables. Therefore, the genetic analysis of factors may help us to understand the hereditary aspect of those characters where genes and environment are involved in a complex manner. Several studies are available with such an approach by Howells (1953) for anthropometric characters, by Potter et al. (1968) for measurements of permanent dentition, and by Nakata et al. (1974) for craniofacial measurements. The application of factor analysis is not new in dermatoglyphic variables (Knussmann, 1967, 1969; Roberts and Coope, 1975; Froehlich, 1976; Jantz and Owsley, 1977; Reed et al., 1978; Reed and Young, 1979; Chopra, 1979; Das Chaudhuri and Chopra, 1983). However, based on individual trait PII (right (R) + left (L)) of finger dermatoglyphics on 200 pedigrees from the Vaidya population, Sengupta and Karmakar (2004) obtained a similar result to that of Karmakar et al. (2005), i.e., Mendelian transmission with a dominant major gene effect on dermatoglyphic trait. Probably this study (Sengupta and Karmakar, 2004) represents the first application of a genetic model test on PII, and thus the emphasis was on further application

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*Correspondence to: Dr. Bibha Karmakar, Biological Anthropology Unit, Indian Statistical Institute, 203 B.T. Road, Kolkata 700 108, India. E-mail: bibha@isical.ac.in

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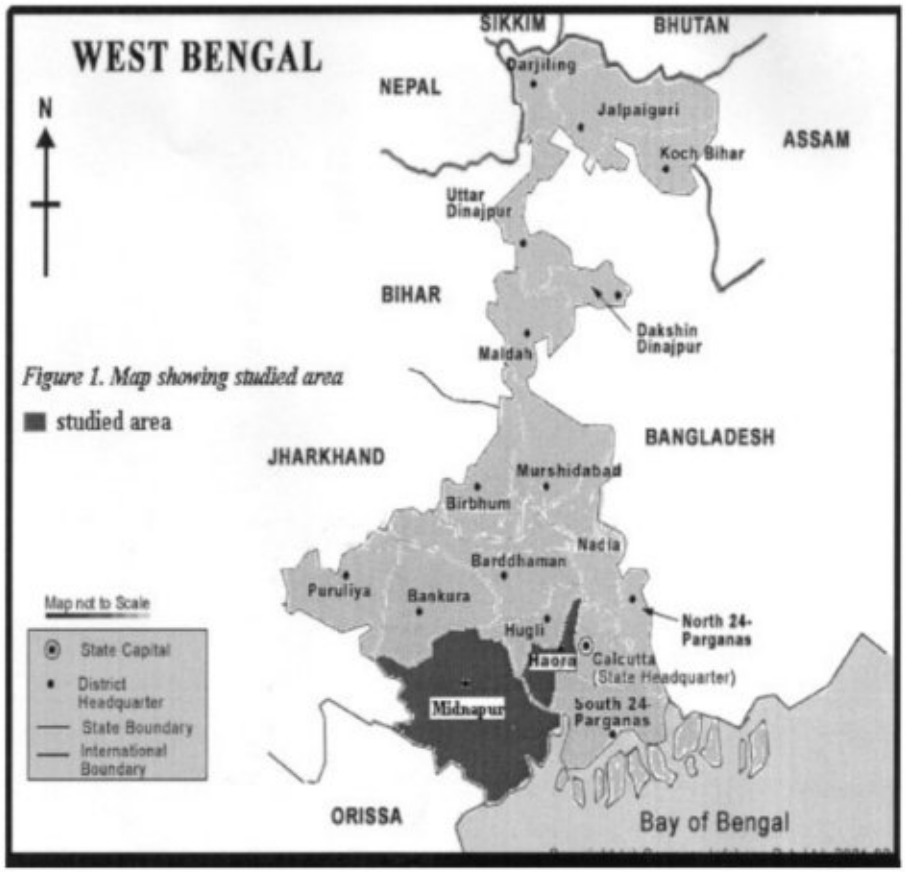

Fig. 1. Map of study area.

of segregation analysis in a large number of different ethnic populations, to confirm the validity and consistency of this finding. Karlin et al. (1983) used structured exploratory data analysis (SEDA) on PII in 125 South Indian Brahmin families, and suggested a multifactorial mode of transmission. From a review of previous studies, it appears that there is a need for further work on this aspect of elucidating the dynamic interaction of genes and environment in shaping human phenotypes, especially in finger dermatoglyphics, such as PII as an individual trait.

Therefore, the present article is an extension of our previous work (Sengupta and Karmakar, 2004; Karmakar et al., 2005) in a large number of population samples to understand the mode of inheritance and to evaluate its relative importance between factor and individual traits.

MATERIALS AND METHODS

Subjects

The populations selected for the present study, i.e., Brahmin (Rarhi), Mahisya, Padmaraj, Muslim (Sunni), and Lodha, are five Bengali-speaking groups and were collected from rural areas of the Howrah and Midnapore districts of West Bengal (Fig. 1). Each of these populations practices monogamy and is strictly endogamous. According to Indian caste hierarchy, the Brahmins stand at the top and are traditionally recognized as priests, although nowadays they are engaged in white-collar jobs. They are divided into five main subcastes: Rarhi, Barendra, Vaidiki, Saptasati, and Madhyasreni. The Rarhi Brahmins make up the principal concentration in West Bengal. The Mahisya are a large cultivating group of middle-caste status. The Padmaraj are mainly a

TABLE 1. Sample size

Population	Abbreviation	Caste hierarchy	No. of families	No. of individuals
Brahmin (Rarhi)	BR	High	100	449
Mahisya	MA	Middle	100	504
Padmaraj	PA	Low	100	525
Muslim (Sunni)	MU	Religious	100	555
Lodha	LO	Tribe	100	402
Total			500	2,435

fishing and cultivating group; their social status is very low, belonging to the scheduled caste. The Muslims belong to the religious community. There are two sectarian groups in West Bengal, i.e., Shia and Sunni Muslims. The Sunni group is one of the largest sects in West Bengal. The Lodha are a small tribal group mostly found in the western part of Midnapore district. Both males and females participate in different economic activities, mainly in cultivation and as agricultural laborers.

All populations are characterized by a demographically stable family structure with traditional relations between family members and living under the same environmental conditions for the last several generations. Historically, they have not been exposed to outside influences such as gene flow, and thus maintain a common gene pool through endogamy. Further, genetic variations among endogamous castes and tribes from West Bengal (including these populations) were studied based on serological and biochemical markers (Mukherjee et al., 1987; Chakraborty et al., 1986). Following Chakraborty et al. (1986), "the constituent genetic profile of any given population does not always correspond exactly to its present social ranking, since some low-caste groups are 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 these populations." Thus the five present populations provide a good opportunity for segregation analysis.

Data collection

As 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 intercaste marriage) population are especially necessary. To obtain such genuine family data and to avoid interobserver variation, the first author alone collected the entire data set. The data were not collected by a random sampling method; rather, each pedigree was specially chosen to have pure caste descent with living parents, and at least two children were included. Thus the data have a limitation, in that the selected families (500 pedigrees) are not representative of all five population groups of West Bengal. The sample sizes are given in Table 1.

Print analysis and variable used

Dermatoglyphic prints for the total number of subjects (2,435 individuals) from 500 pedigrees (100 each), including two generations from five populations, were considered for the present study. The pattern intensity index (PII) on finger of left (PII-L) and right (PII-R) sides was used for correlation analysis. However, for the homogeneity test and segregation analysis, PII (R + L) was used, because parental (FM) correlations are close to zero, while father-offspring (FO), mother-offspring (MO), and sibling (Sib-Sib) correlations are approximately equal for both hands. For this reason, segregation analyses were performed on the combined PII of both hands.

Statistical analyses

Z-transformation. Each value of dermatoglyphic traits was converted to Fisher's Z-transformation to normalize the data. The formula is $Z = (Xi - \bar{X})/SD$, where Xi, $\bar{X}$, and SD are the individual 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.

MANCOVA test. Estimation of homogeneity of the total sample, constructed from the representatives of the five populations, was checked by multivariate analysis of covariance (MANCOVA). MANCOVA is a more sensitive test for

assessing differences between groups than MANOVA (multivariate analysis of variance), because it uses an independent variable to test the similarity of groups being compared. Age, sex, and population are the independent variables, and effects of their interactions are reflected on dermatoglyphic variables (dependent variables). We used MANCOVA (the covariates of dermatoglyphic variables were used) only for the checking of the homogeneity of PII values between the five populations.

Familial correlation (r). To examine the potential familial aggregation of the factor scores, we carried out two types of intrafamilial correlation: 1) Pearson correlation (Pearson, 1893) for resemblance between the *interclass pairs* of relatives in the nuclear families (parent-child and mid parent-child), and 2) *intraclass* correlation (Fisher, 1958) to estimate the degree of resemblance for sib-sib, with a *t*-test of significance.

Segregation analysis. In this study, maximum likelihood (represented as LH) was tested through a number of genetic models to evaluate the potential genetic sources that were shared within familial environmental components contributing to the inheritance of dermatoglyphic traits. The following genetic models were used with the program package MAN-5 (Malkin and Ginsburg, 2002): 1) The *general model* does not assume a particular mode of transmission, and therefore estimates with free probabilities of genotype transmission. 2) The *Mendelian model* assumes Mendelian transmission, with the assumption of Hardy-Weinberg equilibrium. The probabilities of three putative genotypes in the populations are p2, 2pq, and q2. The transmission probabilities of allele A_1 by the corresponding above genotypes are constrained to $\tau_1 = 1.0$, $\tau_2 = 0.5$, $\tau_3 = 0.0$, respectively. 3) The *environmental model* assumes independence of the offspring's major gene genotypes from the parental genotypes of his or her parents, but with possible heterogeneity between generations, i.e., estimated with equal probabilities of genotype transmission, $\tau_1 = \tau_2 = \tau_3$, but τ does not equal p (allele frequency). 4) The *most parsimonious Mendelian model* (MP) was tested if the Mendelian model was accepted, and then three submodels were tested and were used to construct the most parsimonious one: dominant ($\mu A_1A_1 = \mu A_1A_2$), additive ($\mu A_1A_2 = 0.5(\mu A_1A_1 + \mu A_2A_2)$, and recessive ($\mu A_2A_2 = \mu A_1A_2$). The *maximum log likelihood ratio* test was used to justify the restriction of the selected parameters, and finally to accept the MP model obtained. 5) The *arbitrary model* was tested by estimating transmission probabilities and with other model parameters. 6) τ's are equal to p; the hypothesis of nontransmission of the major gene effect was tested by constraining the τ parameters equal to the first allele frequency, i.e., $p = \tau_1 = \tau_2 = \tau_3$. For a detailed description of the models, see Ginsburg and Livshits (1999).

Segregation analysis was performed with the program package MAN-5 (Malkin and Ginsburg, 2002). Here a list of parameters is given describing the major gene model (MG) of the quantitative trait and some characteristics of the selected genetic models: p is the population frequency of the first of the two major alleles A_1 and A_2. μg is the average trait value (genotype value) in all individuals having genotype g, with g = 1, 2, and 3 corresponding to genotypes A_1A_1, A_1A_2, and A_2A_2, respectively. $\sigma^2 g$ is the trait variance in individuals having the same MG genotype g. It estimates the trait variability due to all possible environmental factors and minor genes influencing the trait value; ρ, β, and ε are the correlations of non-MG residuals of the trait between spouses, between parents and offspring, and between siblings, respectively. Correlation ρ is due to common environmental factors shared by spouses, while the two other correlations can be caused both by the corresponding environmental factors and by minor genes affecting the trait that are unidentified in the model.

The MG hypothesis was tested using two maximum likelihood ratio tests (Elston and Stewart, 1971): $\gamma^2_A = 2\,[\mathrm{LH}\,(\tau) - \tau 0]$ and $\gamma^2_E = 2\,[\mathrm{LH}\,(\tau) - \tau]$, where LH ($\tau$) is the maximal likelihood value (natural logarithm) obtained with transmission probabilities $\tau = \Pr(A1 \mid g)$; $\tau 0$ denotes Mendelian transmission probabilities 1.0, 0.5, and 0.0 for the parent's genotype g = 1, 2, and 3, respectively. τ denotes the maximal likelihood estimates of these probabilities, and τ denotes that all three transmission probabilities were assumed to be equal (a so-called "nontransmissible" model). The MG model of trait inheritance is accepted if γ^2_E exceeds the critical value corresponding to df = 2 and the a priori given type 1 error $\alpha = 0.01$ (the hypothetical independence of the offspring's genotype from genotypes of his/her parents is rejected), while concurrently, γ^2_A does not exceed the critical value corresponding to df = 3 and type I error $\alpha = 0.05$ (the Mendelian hypothesis of transmission is accepted). There are additional

TABLE 2. Homogeneity test of PII among five populations by MANCOVA

Parameter	SS	df	MS	F	P
Intercept	120,017.16	1	120,017.16	8,977.97	0.000
Age	32.05	1	32.05	2.40	0.122
Populations	417.00	4	104.25	7.80	0.000
Sex	110.24	1	110.24	8.25	0.004
Population* sex	21.55	4	5.39	0.40	0.807
Error	32,297.01	2,416	13.37		

SS, sum of square; df, degree of freedom; MS, mean square; F, F value.

characteristics of the tested model that help to evaluate its fitting to the pedigree data. These are as follows: 1) $H^2 = \sigma^2\mu/\sigma^2_X$ is the trait variance evaluating the proportion of phenotypic variance attributable to the hypothetical MG effect. 2) $D^2 = H^2 + d^2$ is the proportion of the trait variability attributable to the MG effect and to the non-MG effects described by the correlations ρ, β, and ε included in the model. 3) $0 \leq d^2 \leq 1 - H^2$ and $H^2 \leq D^2 \leq 1$, because $d^2 = 0$ only when the three parameters ρ, β, and ε are equal to zero. The method of pedigree collection for this study was in no way connected with an individual's dermatoglyphic and anthropometric characteristics, and therefore no ascertainment corrections of likelihood were made.

RESULTS

Homogeneity test

Heterogeneity was established by statistical analysis, i.e., MANCOVA between five populations. The results are presented in Table 2, and F-values illustrated very significant differences between populations and also revealed significant differences of the interaction between sexes among the five populations.

Familial correlation

It appears from Table 3 that the correlation between spouses is negligible (nonsignificant), which indicates that no assortative mating or substantial inbreeding had taken place in the studied populations, both for left PII (0.050) and right PII (−0.011). The father-offspring (FO), mother-offspring (MO), and sibling (Sib) correlations for left (0.377, 0.370, and 0.369) and right (0.356, 0.358, and 0.369) are significantly high and approximately equal for both hands.

Segregation analysis

Complex segregation analyses (Tables 4–9) were performed for each of the five populations separately, because significant differences were observed between populations in homogeneity tests by the MANCOVA method. According to the frequency of gene "*p*" in the Muslim population, there is a wide distance between them and others. Among Muslims, the frequency of gene "*p*" (first genotype) is maximal, and these frequencies (0.754, 0.791, 0.755, 0.778, and 0.916) are almost similar in models 1–6, except for model 3, the environmental model (0.131). The average values (not shown in tables) of the trait for different genotypes are equal between males (μm1, μm2, and μm3) and females (μf1, μf2, and μf3) in Muslims and the Lodha, while these are not similar for the remaining groups in all models.

TABLE 3. Familial correlation of pooled sample in five populations

Variable	Parameter	FM	FO	MO	Sib
PII-L	r	0.050	0.377	0.370	0.369
	N	495	1,409.0	1,411.0	1,542.0
	P	0.265	0.000	0.000	0.000
PII-R	r	−0.011	0.356	0.358	0.369
	N	495.0	1,408.0	1,410.0	1,539.0
	P	0.811	0.000	0.000	0.000

FM, father-mother; FO, father-offspring; MO, mother-offspring; Sib, sib pairs; r, correlation; N, sample size.

For the characteristics of the tested models (Table 10) at the first step of the analysis, we compared the general model with other models. γ^2 values show that the environmental and τs-equal-to-p models were significantly different ($P \ll 0.001$) at a 0.1% level in all five populations. Therefore, in all populations, the Mendelian model was accepted, while environmental and τs-equal-to-p models were rejected at a very high statistical level.

The final stage of segregation analysis for choosing the best-fitting model is the most parsimonious Mendelian model (MP) to understand the mode of inheritance. We compared MP and τs-equal-to-p models with the arbitrary model, and the results of γ^2 values strongly rejected the τs-equal-to-p model,

TABLE 4. Segregation analysis of PII in five populations: general model (1)[1]

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.461 ± 0.046	0.581 ± 0.048	0.429 ± 0.040	0.754 ± 0.056	0.521 ± 0.105
μm1	−0.605 ± 0.073	−0.738 ± 0.134	−0.576 ± 0.051	−0.598 ± 0.140	−0.867 ± 0.205
μm2	−0.071 ± 0.148	0.162 ± 0.156	−0.146 ± 0.123	0.803 ± 0.174	−0.008 ± 0.265
μm3	1.056 ± 0.129	1.408 ± 0.069	0.913 ± 0.100	1.403 ± 0.097	1.034 ± 0.249
μf1	−0.709 ± 0.084	−0.977 ± 0.131	−0.722 ± 0.065	−0.663 ± 0.147	−0.753 ± 0.122
μf2	−0.324 ± 0.145	0.066 ± 0.142	−0.537 ± 0.139	0.871 ± 0.126	−0.155 ± 0.251
μf3	0.925 ± 0.140	1.195 ± 0.076	0.841 ± 0.100	0.837 ± 0.128	1.004 ± 0.271
σ_1^2	0.122 ± 0.038	0.520 ± 0.097	0.075+	0.580 ± 0.084	0.212 ± 0.100
σ_2^2	0.980 ± 0.134	0.489 ± 0.068	0.896 ± 0.125	0.255 ± 0.076	0.777 ± 0.159
σ_3^2	0.352 ± 0.083	0.090 ± 0.022	0.315 ± 0.057	0.075+	0.423 ± 0.156
β	0.198 ± 0.057	0.193 ± 0.053	0.129 ± 0.050	0.200 ± 0.042	0.226 ± 0.075
ε	0.020 ± 0.105	0.052 ± 0.058	0.126 ± 0.031	0.074 ± 0.057	0.012 ± 0.134
τ_1	1.000+	0.999 ± 0.052	1.000+	1.000+	1.000+
τ_2	0.631 ± 0.069	0.519 ± 0.059	0.546 ± 0.057	0.514 ± 0.059	0.493 ± 0.070
τ_3	0.000+	0.000+	0.000+	0.226 ± 0.137	0.000+
LH	**−579.67**	**−624.45**	**−638.19**	**−674.05**	**−511.75**

[1]ρ, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_1A_1, A_1A_2, and A_2A_2; σ^2g, trait variance in individuals of genotype g; β and ε, partial correlations in parents and offspring, and sibs; τg, probability of transmission of allele A_1 to offspring generation by each genotype; +, parameter estimate achieved its limit; m, male; f, female.

TABLE 5. Segregation analysis of PII in five populations: Mendelian model (2)[1]

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.482	0.587	0.440	0.791	0.519
μm1	−0.598	−0.744	−0.575	−0.543	−0.867
μm2	−0.087	0.173	−0.147	0.892	−0.011
μm3	1.030	1.411	0.920	1.382	1.034
μf1	−0.706	−0.973	−0.719	−0.606	−0.754
μf2	−0.334	0.076	−0.532	0.906	−0.157
μf3	0.909	1.200	0.851	0.863	1.002
σ_1^2	0.113	0.517	0.075+	0.610	0.212
σ_2^2	0.985	0.488	0.892	0.232	0.776
σ_3^2	0.366	0.089	0.309	0.075+	0.424
β	0.198	0.194	0.128	0.185	0.226
ε	0.036	0.051	0.127	0.085	0.012
τ_1	[1.000]	[1.000]	[1.000]	[1.000]	[1.000]
τ_2	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]
τ_3	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]
LH	**−581.48**	**−624.51**	**−638.52**	**−676.87**	**−511.76**

[1]ρ, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_1A_1, A_1A_2, and A_2A_2; σ^2g, trait variance in individuals of genotype g; β and ε, partial correlations in parents and offspring, and sibs; τg, probability of transmission of allele A_1 to offspring generation by each genotype; brackets indicate parameter fixed to shown value; +, parameter estimate achieved its limit; m, male; f, female.

which clearly indicates that the Mendelian model with a major gene (MG) effect is present in all populations for the PII dermatoglyphic trait.

DISCUSSION

The goal of this report was to compare the present results with our earlier study (Karmakar et al., 2005). Unfortunately, the existing information regarding mode of inheritance by a genetic model-fitting test is very limited (Sengupta and Karmakar, 2004; Karmakar et al., 2005), and thus we are unable to provide an accurate explanation compared with such studies in other populations. Segregation analyses confirmed major gene involvement with a Mendelian expectation on PII of the present study, which is exactly similar to our previous findings based on factor 1 including PII (Karmakar et al., 2005) of the same populations. Our earlier study (Sengupta and Karmakar, 2004), based on individual trait PII in the Vaidya population, demonstrated that the existence of a major gene on PII and the transmission of this effect are consistent with a Mendelian expectation; the present findings fully agree with this result. The involvement of a major gene on PII was also suggested by correlation analysis (Mukherjee, 1966;

TABLE 6. Segregation analysis of PII in five populations: environmental model (3)[1]

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.091	0.656	0.429	0.131	0.307
μm1	−3.026	−0.207	−0.479	−1.774	−1.184
μm2	−0.665	0.014	−0.044	1.096	0.258
μm3	0.252	1.344	0.681	−0.201	0.005
μf_1	−2.790	−0.533	−0.654	−2.553	−0.955
μf_2	−0.671	0.067	−0.488	0.997	0.057
μf_3	0.099	1.164	0.688	−0.207	0.033
σ_1^2	0.075	0.871	0.075+	0.455	0.073
σ_2^2	0.101	0.641	1.028	0.134	0.946
σ_3^2	0.908	0.075	0.396	0.713	0.848
β	0.222	0.222	0.211	0.212	0.282
ε	0.112	0.047	0.062	0.078	0.001
τ_1	0.117	0.664	0.447	0.100	0.337
τ_2	0.117!	0.664!	0.447!	0.100!	0.337!
τ_3	0.117!	0.664!	0.447!	0.100!	0.337!
LH	**−592.17**	**−652.62**	**−670.39**	**−704.06**	**−521.43**

[1] p, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_1A_1, A_1A_2, and A_2A_2; $\sigma^2 g$, trait variance in individuals of genotype g; β and ε, partial correlations in parents and offspring, and sibs; τg, probability of transmission of allele A_1 to offspring generation by each genotype; !, parameter is constrained to equal parameter above in table; +, parameter estimate achieved its limit; m, male; f, female.

TABLE 7. Segregation analysis of PII in five populations: arbitrary model (4)

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.470	0.596	0.431	0.755	0.591
μm1	−0.645	−0.872	−0.578	−0.628	−0.714
μm2	−0.020	0.169	−0.130	0.839	0.040
μm3	1.004	1.411	0.874	1.394	1.225
μf1	−0.645$	−0.872$	−0.723	−0.628$	−0.714$
μf2	−0.333	0.169$	−0.525	0.839$	0.040$
μf3	1.004$	1.211	0.874$	0.839!	1.225$
σ_1^2	0.137	0.500	0.075+	0.578	0.296
σ_2^2	0.989	0.500!	0.902	0.257	0.793
σ_3^2	0.343	0.087	0.317	0.075+	0.296!
β	0.205	0.232	0.125	0.242	0.226
ε	[0.000]	[0.000]	0.128	[0.000]	[0.000]
τ_1	1.000+	1.000+	1.000+	1.000	1.000+
τ_2	0.634	0.501	0.546	0.507	0.498
τ_3	0.000+	0.000+	0.000+	0.247	0.000+
LH	**−580.40**	**−626.77**	**−638.42**	**−675.31**	**−512.34**

[1] p, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_1A_1, A_1A_2, and A_2A_2; $\sigma^2 g$, trait variance in individuals of genotype g; β and ε, partial correlation in parents and offspring, and sibs; τg, probability of transmission of allele A_1 to offspring generation by each genotype; brackets indicate parameter fixed to shown value; !, parameter is constrained to equal parameter above in table; $, parameter constrained to equal corresponding parameter in males; +, parameter estimate achieved its limit; m, male; f, female.

Hreczko and Ray, 1985) in Indian populations and in Polish families (Loesch, 1971). The evidence of the above similarities indicates that there is a common variation in dermatoglyphic variables represented by the factor and the individual trait may be due to the involvement of the same genetic component. This was proved by the existence of the same mode of inheritance in all five populations. Therefore, we did not find any variation between the present and previous results of hereditary aspects in cases of finger dermatoglyphics. Especially concerning factor 1 (i.e., general size of the finger pattern), Chopra (1979) stated that no separate complexes are responsible for individual fingers. This also supports the field theory (Butler, 1963) that each finger is a discrete part of a digital complex comprising 10 fingers, and not a separate unit acted upon independently by the genes involved. Knussmann (1969), Roberts and Coope (1975), Jantz and Owsley (1977), and Das Chaudhuri and Chopra (1983) supported this theory. Our present results also agree with this theory. In cases of familial relationship, Reed and Young (1979) demonstrated that the factors might have even stronger genetic components than the individual varia-

TABLE 8. Segregation analysis of PII in five populations: most parsimonious model (5)[1]

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.491 ± 0.046	0.596 ± 0.042	0.443 ± 0.037	0.778 ± 0.037	0.591 ± 0.050
μm1	−0.643 ± 0.059	−0.872 ± 0.101	−0.577 ± 0.049	−0.605 ± 0.097	−0.714 ± 0.102
μm2	−0.044 ± 0.148	0.170 ± 0.113	−0.132 ± 0.122	0.859 ± 0.089	0.040 ± 0.168
μm3	0.985 ± 0.114	1.411 ± 0.063	0.884 ± 0.084	1.363 ± 0.110	1.225 ± 0.123
μf1	−0.643$	−0.872$	−0.720 ± 0.064	−0.605$	−0.714$
μf2	−0.351 ± 0.145	0.170$	−0.520 ± 0.133	0.859$	0.040$
μf3	0.985$	1.211 ± 0.076	0.884$	0.859!	1.225$
σ_1^2	0.128 ± 0.037	0.500 ± 0.061	0.075+	0.593 ± 0.067	0.296 ± 0.045
σ_2^2	0.988 ± 0.130	0.500!	0.899 ± 0.121	0.252 ± 0.053	0.793 ± 0.149
σ_3^2	0.354 ± 0.083	0.087 ± 0.021	0.310 ± 0.056	0.075+	0.296!
β	0.206 ± 0.047	0.232 ± 0.027	0.124 ± 0.038	0.232 ± 0.020	0.226 ± 0.048
ε	[0.000]	[0.000]	0.129 ± 0.023	[0.000]	[0.000]
τ_1	[1.000]	[1.000]	[1.000]	[1.000]	[1.000]
τ_2	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]
τ_3	[0.000]	[0.000]	[0.000]	[0.000]	[0.000]
LH	**−582.26**	**−626.77**	**−638.74**	**−678.20**	**−512.34**

[1]p, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_2A_2, A_1A_2, and A_2A_2; σ^2g, trait variance in individuals of genotype g; β and ε, partial correlations in parents and offspring, and sibs; τg, probability of transmission of allele A_2 to offspring generation by each genotype; brackets indicate parameter fixed to shown value; !, parameter is constrained to equal parameter above in table; $, parameter constrained to equal corresponding parameter in males; +, parameter estimate achieved its limit; m, male; f, female.

TABLE 9. Segregation analysis of PII in 5 populations: τs-equal-to-p model (6)[1]

Parameter	Brahmin	Mahisya	Padmaraj	Muslim	Lodha
ρ	0.421	0.664	0.428	0.916	0.581
μm1	−0.625	−0.156	−0.479	−0.186	−0.535
μm2	−0.035	−0.156	−0.053	1.078	−0.067
μm3	0.540	1.328	0.686	1.389	1.117
μf1	−0.625$	−0.156$	−0.654	−0.186$	−0.535$
μf2	−0.373	−0.156$	−0.488	1.078$	−0.067$
μf3	0.540$	1.128	0.686$	1.078!	1.117$
σ_1^2	0.089	0.835	0.075+	0.845	0.398
σ_2^2	1.173	0.835!	1.030	0.128	0.844
σ_3^2	0.606	0.079	0.397	0.075+	0.398!
β	0.275	0.253	0.216	0.253	0.312
ε	[0.000]	[0.000]	0.057	[0.000]	[0.000]
τ_1	0.421	0.664	0.428	0.916	0.581
τ_2	0.421!	0.664!	0.428!	0.916!	0.581!
τ_3	0.421!	0.664!	0.428!	0.916!	0.581!
LH	**−597.84**	**−655.68**	**−670.57**	**−709.33**	**−524.42**

[1]p, population frequency of first allele; μg, mean genotypic value of trait, where g = 1, 2, and 3 for genotypes A_2A_2, A_1A_2, and A_2A_2; σ^2g, trait variance in individuals of genotype g; β and ε, partial correlations in parents and offspring, and sibs; τg, probability of transmission of allele A_2 to offspring generation by each genotype; brackets indicate parameter fixed to shown value; !, parameter is constrained to equal parameter above in table; $, parameter constrained to equal corresponding parameter in males; +, parameter estimate achieved its limit; m, male; f, female.

bles (based on twin data). These results were compared with those results reported for the individual variables comprising each factor (Reed et al., 1978). On the basis of their comparison, Reed and Young (1979) concluded that "Multivariate pattern factors display the same findings as those individual variables comprising the factors and may provide additional genetic information over considering each variable singly." The present and previous results of correlation are very similar and support these results. Based on family data in a German population, Chopra (1979) stated that the factors (for example, factor 1 for finger dermatoglyphic variables) provide more information due to its underlying structure. Because factor 1 means that the finger patterns of all 10 fingers belong to one complex, factors for other areas do not seem to add any additional genetic information. Chopra (1979) stated that the use of individual characters for population studies is justified.

However, our present results in all five populations strongly suggest that there is no variation between individual trait and its factor with respect to the mode of inheritance which

TABLE 10. Characteristics of tested models of PII in five populations

Populations		Model	2, Mendelian	3, Environmental	4, Arbitrary	5, MP-Mendelian	6, ts equal to p
Brahmin	γ^2 vs. general	1	3.62	25.68	5.18	1.46	36.64
	df		3	3	6	3	6
	P		0.306	<<0.001	0.521	0.692	<<0.001
	γ^2 vs. arbitrary	4				3.72	34.88
	df					3	3
	P					0.293	<<0.001
Mahisya	γ^2 vs. general	1	0.12	56.36	4.64	4.64	62.46
	df		3	3	7	4	7
	P		0.989	<<0.001	0.704	0.326	<<0.001
	γ^2 vs. arbitrary	4				0.00	57.82
	df					3	3
	P					1.000	<<0.001
Padmaraj	γ^2 vs. general	1	0.66	64.76	1.10	0.46	64.76
	df		3	3	4	1	4
	P		0.883	<<0.001	0.894	0.498	<<0.001
	γ^2 vs. arbitrary	4				0.64	64.30
	df					3	3
	P					0.887	<<0.001
Muslim	γ^2 vs. general	1	5.64	61.10	8.30	2.52	70.56
	df		3	3	7	4	7
	P		0.131	<<0.001	0.307	0.641	<<0.001
	γ^2 vs. arbitrary	4				5.78	67.98
	df					3	3
	P					0.122	<<0.001
Lodha	γ^2 vs. general	1	0.02	19.62	1.18	1.18	25.34
	df		3	3	8	5	8
	P		0.999	<<0.001	0.997	0.947	0.001
	γ^2 vs. arbitrary	4				0.00	24.16
	df					3	3
	P					1.000	<<0.001

may be due to the involvement of the same genetic component.

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