Genetic Determinants of 22 Quantitative Dermatoglyphic Traits in the Chuvashian Population of Russia: Complex Segregation Analysis

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Abstract: *Background*: Dermatoglyphics is widely used as a genetically determined trait in Anthropology. However, little is known about their pattern of inheritance due to lack of advanced statistical genetic model-fitting techniques despite the existence of advanced statistical packages.

Objectives: The aim of the present study is to determine the mode of inheritance of dermatoglyphic traits through complex segregation analysis.

Subjects and Methods: Finger and palmar prints of 325 individuals belonging to 104 families from the Chuvashian population of Russia were used for principal component analysis, familial correlation, and segregation analysis (package MAN-5).

Results: The results suggest a common internal structure of three factors when compared with other populations. (b) Significant familial correlations (except spouse) indicate the involvement of familial components to the variation of dermatoglyphic traits. (c) Segregation analysis reveals the transmission of genetic effects in the families, which follows the Mendelian model and confirm major gene effect on factors 1 and 2, whereas there is no evidence of major gene effect or an environmental effect on factor 3.

Conclusion: Major gene involvement with Mendelian expectation regarding finger dermatoglyphics is confirmed for all analyzed traits. However there is no evidence of significant support for major gene effect or environmental effect on palmar a-b ridge counts.

Keywords: Segregation analysis, Chuvashian population, Russia.

INTRODUCTION

Dermal ridge patterns are permanently laid down during early fetal life in the third to fourth month of the gestation period. There is no postnatal change thereafter by environment or age factors throughout the life. Dermatoglyphic traits were found to be genetically determined and conservative in their evolution [1]. The family studies, in which segregation analysis is used, can provide the correct relationship between the genotype and phenotype. Several researchers who have used traditional methods of genetic analysis concluded that different genes are probably responsible for dermatoglyphic traits [2-4]. However, the conclusions from various studies are still contradictory [5-16]. All the earlier studies were based only on the statistical application of familial correlation or regression between relatives; however, these methods are insufficient to detect the mode of inheritance of a trait. We know that genetic model-bound analyses of microevolutionary processes have contributed significantly to our understanding of the mechanism underlying human phenotypic traits, i.e., trait heritability, which represents the genetic variability associated with the phenotype [17-19]. Recently, several advanced statistical program packages with model fitting techniques have become available, which are very useful in complex segregation analysis to determine the effect of genes [20-29]. However, we still do not know the exact source of genetic regulation for dermatoglyphic traits due to the paucity of such studies. A few studies on palmar dermatoglyphics include segregation analysis [30-32]. Surprisingly, an inheritance model of dermatoglyphic traits has yet to be established [33-35]. It is well established that the relative contribution of genetic and environmental factors to phenotypic variation of dermatoglyphics may differ from population to population [1,7,36-42]. In view of the above ethnic diversity, it would be interesting to determine whether any similarity exists between the results of our previous studies on Indian populations and the present Chuvashian population from Russia. Furthermore, family-based studies on dermatoglyphics in the Chuvashian population are hardly available. Here, we report the results of modern techniques of segregation analyses (using various genetic models) of nuclear pedigrees from a rural Chuvashian population of Russia. Our main goal is to elucidate whether there

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exists any major gene effects on dermatoglyphic traits. In addition, we examine if there any variation / similarity when compared with other populations with respect to dermatoglyphic trait inheritance patterns.

MATERIALS AND METHODOLOGY

Subjects and Historical Background

The studied individuals within the Chuvashian population reside in several small villages along the Volga River in the Chuvasha area of the Autonomy of the Russian Federation. This population migrated to these regions during the 7^{th} and 8^{th} centuries. Ethnically, the Chuvashian population is of a mixed Caucasian origin and came into existence during the last quarter of the first millennium AD in the forested or hilly portions of the Volga riverside [43]. Their ancestors were most likely Bulgars from the Volga and Kama riversides, who intermarried with the local Finno-Ugric tribes [44]. This population is characterized by a demographically stable familial structure with traditional relations between family members. Their principal source of livelihood is agriculture and they share similar biotic and economic conditions, as well as professions, as is usual for rural communities. The Chuvashian families have lived under the same environmental conditions for several generations and thus were not exposed to any outside gene pool [45,46]. The sample consists of 325 individuals from 104 families (92 parents and 233 offspring). All studied individuals were randomly selected through direct contact with all households who agreed to participate in the study. The data were collected by the joint expedition of the Department of Anatomy and Anthropology, Sackler Faculty of Medicine, Tel Aviv University, Israel and the Institute and Museum of Anthropology, Moscow State University, Russia for details, see [47].

Print Analysis and Variables Used

Dermatoglyphic prints were collected according to the rolled print (inked) method of Cummins and Midlo [1]. The variables included 22 quantitative traits (12 finger ridge counts, 2 palmar a-b ridge counts, 3 pattern intensity indices (PII), 4 palmar main line (A and D) endings, and main line index (MLI)). However, for principal component analysis only 18 variables were used, because 4 traits, namely, MLI, PII (both hands), TFRC and AFRC were excluded since these traits are the sum of ridge counts, sum of palmar main lines, and sum of PII- left and right. Dermatoglyphic traits were evaluated, for the most part, by using the methods of Cummins and Midlo[1], Holt [7] and Penrose [8]. The first author alone analyzed the whole dermatoglyphic prints to avoid any inter-observer errors.

Statistical Analysis

Z-Transformation

Each value of the dermatoglyphic traits was converted to Fisher's Z-transformation to normalize the data following Fisher [48]. 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 other calculations are based on these transformed Z-scores. *Principal component analysis*: By exploiting patterns in the genetic correlation matrix between the studied traits, principal factors were extracted following BMDP statistical software after Dixon [49] with varimax rotation of principal components. Factor scores were then computed for each individual of each pedigree sample. *Familial correlations*: To examine the potential familial aggregation, we carried out two types of 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 SPSS statistical package of Norusis [50]. *Genetic model tests*: Complex segregation analysis was carried out following Maximum Likelihood Methods by using the Package of MAN-5 version Malkin and Ginsburg [29] 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. H^2 represents the proportion of the within-genotype variance attributed to polygenes for details; see Karmakar *et al*. [34]. 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, 2pq and q^2. The transmission probabilities of allele A_1 by the above corresponding genotypes are τ_1=1.0, τ_2=0.5, τ_3=0.0, respectively.

3. τ values equal to p regarding the hypothesis of non-transmission of the major gene effect; this was tested by constraining the τ parameters equal to the first allele frequency, $p=\tau_1=\tau_2=\tau_3$.

4. The Most Parsimonious Mendelian model (MP) was tested if the Mendelian model was accepted; then the following three sub-models were tested: dominant: $\mu_{A1A1}= \mu_{A1A2}$, additive: $\mu_{A1A2} = 0.5 (\mu_{A1A1} + \mu_{A2A2})$, and recessive: $\mu_{A2A2} = \mu_{A1A2}$).

5. The Arbitrary model was tested by estimating transmission probabilities with other model parameters.

6. 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 2-6 are the sub-models of the general model and thus were compared with this model. Model 4 is the sub-model of the Mendelian model and therefore was tested against it. 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 corrections of likelihood was made (for a detailed description of the models, see Ginsburg and Livshits [28].

RESULTS

Principal Component Analysis (PCA)

Eighteen quantitative dermatoglyphic traits were selected for PCA; the results are presented in Table **1**. Clear separations of dermatoglyphic variables into three factors are easily interpretable. Factor 1 alone accounted for about 30% of the total variation, whereas Factor 2 and Factor 3 explained approximately 10% each. We retained the factor loading and the respective scores for the first three factors, which jointly accounted for more than 50% of the total variation. Factor 1 explained the finger ridge counts and the pattern intensity index (PII); it can serve as an indicator of the finger pattern intensity correlated with the size of the finger ridge count of the individuals. Factor 2 described the variance of the palmar main line terminations, which represent the palmar pattern intensity of the individual.

Factor 3 is a clear a-b interdigital ridge count factor and determines the ridge count size in the corresponding areas of the individual palm.

Familial Correlations Based on Individual Traits

Table **2** provides correlations between spouses, between parent-offspring, and between siblings.

The parental correlations are low values nearly zero; some are even negative and non-significant ($p > 0.05$) for 22 dermatoglyphic traits. All the other correlations are positive and significant at the 1% level with few exceptions: Finger RC, IIr; D-line exit, l between F-Off; Finger RC, IIIl; IVL and D-line exit, l between M-Off; Finger RC, II, Finger RC, III between siblings, respectively. There is no striking difference in the correlation values of the two kinds of parent-child correlations. However, 22 dermatoglyphic traits differ a little in the extent of correlation coefficient values: among the fingers are shown the highest r in 4 and 5 digits (0.361

Table 1. Principal Component Analysis of 18 Dermatoglyphic Traits

Variables	Factor 1	Factor 2	Factor 3
ZFRC_1R	0.528		
ZFRC_2R	0.585		
ZFRC_3R	0.742		
ZFRC_4R	0.767		
ZFRC_5R	0.754		
ZFRC_1L	0.307		
ZFRC_2L	0.715		
ZFRC_3L	0.702		
ZFRC_4L	0.593		
ZFRC_5L	0.504		
ZPII_L	0.722		
ZPII_R	0.788		
ZAB_RC_R			0.788
ZAB_RC_L			0.776
ZLINEAL		0.418	
ZLINEAR		0.750	
ZLINEDL		0.698	
ZLINEDR		0.787	
V.P.	5.196	1.956	1.848
Cum.Var.	28.9	39.8	50.1

Loading values below 0.25 are omitted. The V.P. is the variance explained by each factor. Cum.Var. is the cumulative proportion of explained variance.

Table 2. Familial Correlation Based on 22 Finger and Palmar Dermatoglyphic Traits

Variables		F-M	F-Off	M-Off	Sib- Sib
Finger RC, Ir	r	-0.038	0.302	0.255	0.187
	N	95	194	219	172
	P	0.712	0.000	0.000	0.014
Finger RC, IIr	r	0.272	0.144	0.261	0.156
	N	75	170	175	150
	P	0.018	0.062	0.000	0.056
Finger RC, IIIr	r	-0.040	0.258	0.224	0.244
	N	84	175	203	159
	P	0.718	0.001	0.001	0.002
Finger RC, IVr	r	-0.034	0.268	0.361	0.219
	N	101	197	225	168
	P	0.735	0.000	0.000	0.004
Finger RC, Vr	r	-0.160	0.322	0.359	0.261
	N	102	200	224	173
	P	0.107	0.000	0.000	0.001
Finger RC, II	r	0.101	0.249	0.268	0.088
	N	93	195	217	163
	P	0.338	0.000	0.000	0.265
Finger RC, Ill	r	0.045	0.297	0.149	0.145
	N	76	165	181	143
	P	0.700	0.000	0.044	0.084
Finger RC, IIIl	r	0.225	0.229	0.122	0.259
	N	88	181	199	154
	P	0.035	0.002	0.086	0.001
Finger RC, IVl	r	-0.084	0.269	0.126	0.169
	N	97	192	228	170
	P	0.412	0.000	0.057	0.028
Finger RC, Vl	r	-0.061	0.151	0.208	0.117
	N	98	198	221	173
	P	0.549	0.034	0.002	0.126
Total Finger RC	r	-0.077	0.406	0.333	0.291
	N	112	214	239	178
	P	0.421	0.000	0.000	0.000

(Table 2). Contd…..

Variables		F-M	F-Off	M-Off	Sib- Sib
Absolute FRC	r	-0.044	0.415	0.396	0.298
	N	111	210	233	174
	P	0.645	0.000	0.000	0.000
PII, lh	r	0.005	0.346	0.352	0.374
	N	101	197	232	175
	P	0.996	0.000	0.000	0.000
PII, rh	r	-0.113	0.326	0.255	0.301
	N	101	199	230	175
	P	0.261	0.000	0.000	0.000
PII, both h	r	-0.039	0.345	0.338	0.392
	N	93	183	225	172
	P	0.704	0.000	0.000	0.000
a-b RC, rh	r	0.148	0.280	0.222	0.162
	N	106	200	232	170
	P	0.129	0.000	0.001	0.034
a-b, RC, lh	r	-0.057	0.254	0.169	0.177
	N	106	202	232	175
	P	0.561	0.000	0.010	0.019
A-line exit, l	r	0.150	0.206	0.176	0.304
	N	107	207	236	176
	P	0.124	0.003	0.007	0.000
A-line exit, r	r	0.130	0.233	0.297	0.292
	N	106	204	234	178
	P	0.185	0.001	0.000	0.000
D-line exit, l	r	-0.209	0.085	0.113	0.349
	N	107	207	236	176
	P	0.030	0.221	0.083	0.000
D-line exit, r	r	-0.108	0.157	0.155	0.151
	N	106	204	234	178
	P	0.271	0.025	0.018	0.044
MLI	r	-0.032	0.223	0.253	0.358
	N	100	197	230	176
	P	0.754	0.002	0.000	0.000

and 0.359); higher r-values are in AFRC in 3 combinations: F-Off, M-Off, and Sib-Sib (0.415, 0.396, and 0.298).

Familial Correlations Based on PCA

Table **3** presents the results of familial correlations of three factors: between spouses, between parent-offspring, and between sib-pairs. A similar trend was observed as found correlations based on each dermatoglyphic trait separately (Table **1**).

The correlation between spouses is negligible, indicating no assortative mating in the studied population. There is no striking difference in the correlation values between other parent-offspring and sib-pair correlations except Factor 3 for

Table 3. Familial Correlation Based on Three Factors

Relatives	F-M	Parent-Offspring	Sib-Sib
Factor 1			
Correlation	0.096	0.334	0.261
Pair	46	228	98
p-value	0.544	0.001	0.010
Factor 2			
Correlation	-0.106	0.149	0.313
Pair	46	228	98
p-value	0.497	0.027	0.003
Factor 3			
Correlation	0.139	0.332	0.179
Pair	46	228	98
p-value	0.363	0.001	0.082

sib-pair correlations, which differed non-significantly (p> 0.05).

Segregation Analysis Based on Factors

Based on the three factors, we carried out segregation analysis. We applied six genetic models and made comparisons in order to choose the best fitting model between (a) the General model with the Mendelian, Environmental, MP, and Arbitrary models, and τ values equal to P and between (b) Arbitrary with MP and τ values equal to P. The results of segregation analyses are presented in Tables **4** to **6**. These tables 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_{m1}, μ_{f1}- genotypic values for genotype A_1A_1 for males (m) and females (f); μ_{m2}, μ_{f3}- genotypic values for genotype A_1A_2; μ_{m3}, μ_{f3}- 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. In the first step of analysis five models were compared with the General model (Model 1). The major gene effect was observed only for Factor 1 (Table **4**). The Mendelian model was not rejected when compared with the General model (p=0.18). The model with transmission probabilities (τ) equal to the allele frequency (P) was strongly rejected (p=0.0005). The best fitting most parsimonious (MP) Mendelian model was sex sensitive, but genotypic values for homozygous genotypes (μ_1, μ_2) were constrained to be equal between sexes. Partial correlations for parent-offspring (β) and siblings (ε) were both constrained to zero. The MP Mendelian model was not rejected by the likelihood ratio test in comparison with the constrained model with arbitrary τ values (p=0.51)**,** whereas the Environmental model was rejected ($p<10^{-6}$). For Factor 2 (Table **5**) the major gene effect was not accepted, because the Mendelian model was rejected (p=0.044).

Nevertheless, the model with equal τ values and the Environmental model were also rejected (p=0.001); this shows the presence of some type of inheritance in this trait. In analyzing Factor 3 (Table **6**), we were not able to reject either the Mendelian model (p=0.066), or the Equal τ value or the Environmental model (p=0.086). Thus, the major gene assumption was not accepted for this trait.

DISCUSSION

Principal Component Analysis

The structure of the factor depends upon the variables used in the analysis. Therefore, the results of only a few studies are in agreement with the present findings. Comparison of the present results with earlier studies was not possible because the different analyses were based on different sets of variables: Froehlich [51], in the Melanesian population sample, and Chopra [52], in the German family material, obtained three factors, as found in our present study. Roberts [10] reported Factor 1 for finger patterns in the English sample; Das Chaudhuri and Chopra [53], in comparing 100 Andhra families, also reported factors 1, 2, and 3, as we also observed in the present study. However, a general factor (Factor 1) was missing in the analysis of Knussman [5] and in Jantz and Owsley's [3] study; perhaps this discrepancy was due to the different sets of variables included in the analysis. The present findings support earlier suggestions [5,51-53] that finger and palmar variables are controlled by independent factors because in palmar variables a general factor has not been identified. Palmar variables are composed of different factors, unlike finger ridge counts (Factor 1), and these are for main line termination (Factor 2) and for the a-b ridge count (Factor 3). Factor 1 supports the hypothesis of Butler's field theory [54] that each finger is a discrete part of a digital complex comprising ten fingers and not a separate unit acted on independently by the genes involved. Roberts and Coope [55] as well as Jantz and Owsley [13] also support this field theory for their results of factor analysis on dermatoglyphic data. Our present findings fully

Table 4. Segregation Analysis of Factor 1

Parameter	General Model (1)	Mendelian τ-s (2)	τ-s equal to p (3)	Mendelian Most Parsimonious (4)	Arbitrary (5)	Environmental (6)
P	0.506 ± 0.047	0.513	0.360	0.515 ± 0.0485	0.521	0.514
μ_{m1}	-1.061 ± 0.107	-0.985	-1.067	-1.034 ± 0.105	-1.083	-1.057
μ_{m2}	0.294 ± 0.103	0.301788	0.050	0.303 ± 0.127	0.286	0.343
μ_{m3}	1.223 ± 0.114	1.196	0.714	1.139 ± 0.113	1.195	1.124
μ_{f1}	-1.231 ± 0.117	-1.127	-0.594	-1.033!(=μ_{m1})	-1.083!(=μ_{m1})	-1.057!(=μ_{m1})
μ_{f2}	-0.153 ± 0.132	-0.171	-0.594	-0.165 ± 0.154	-0.092	-0.136
μ_{f3}	1.114 ± 0.132	1.073	0.572	1.1394!(=μ_{m3})	1.195!(=μ_{m3})	1.124!(=μ_{m3})
σ^2	0.256 ± 0.0419	0.322	0.596	0.334 ± 0.049	0.272	0.328
β	0.070 ± 0.092	-0.015	0.274	[0]	[0]	[0]
ε	0.272 ± 0.0595	0.025	0.124	[0]	[0]	[0]
τ_1	0.904 ± 0.0791	[1]	0.360!(=p)	[1]	0.919	0.514!(=p)
τ_2	0.513 ± 0.071	[0.5]	0.360!(=p)	[0.5]	0.502	0.514!(=p)
τ_3	0.116 ± 0.069	[0]	0.360!(=p)	[0]	0.080	0.514!(=p)
Log LH	-426.628	-429.060	-435.507	-429.703	-428.557	-444.999
χ^2		4.864 (1)	17.758 (1)	6.150 (1)	2.292 (4)	32.884 (5)
d.f.		3	3	7	3	3
P		0.18	0.0005	0.52	0.51	$3.4 \cdot 10^{-7}$

Parameter constraints: ! Parameter is equal to parameter above specified in parentheses; [] parameter was fixed to the specified value; + parameter achieved the limit of the valid range. In the χ^2 row the digit in parentheses specifies the number of model for LRT comparison.

Table 5. Segregation Analysis of Factor 2

Parameter	General Model (1)	Mendelian τ-s (2)	τ-s equal to p (3)
P	0.609 ± 0.037	0.6384	0.655
μ_{m1}	-0.725 ± 0.072	-0.735	-0.678
μ_{m2}	0.809 ± 0.091	0.823	0.850
μ_{m3}	0.565 ± 0.500	0.434	0.338
μ_{f1}	-0.861 ± 0.093	-0.866	-0.707
μ_{f2}	0.636 ± 0.077	0.642	0.7189
μ_{f3}	-1.835 ± 0.181	-1.846	-2.080
σ^2	0.354 ± 0.044	0.354	0.319
β	0.4580 ± 0.012	0.459	0.324
ε	-0.510 ± 0.104	-0.481	0.030
τ_1	1+	[1]	0.655!(=p)
τ_2	0.628 ± 0.063	[0.5]	0.655!(=p)
τ_3	0+	[0]	0.655!(=p)
Log LH	-429.588	-431.615	-437.749
χ^2		4.054	16.322
d.f.		1	3
p		0.044 (1)	0.001 (1)

Notes: Same as Table **4**.

Table 6. Segregation Analysis of Factor 3

Parameter	General Model (1)	Mendelian τ-s (2)	τ-s equal to p (3)
P	0.865 ± 0.042	0.173	0.088
μ_{m1}	-0.559 ± 0.122	-2.243	-3.596
μ_{m2}	0.680 ± 0.197	0.871	-0.716
μ_{m3}	0.915 ± 0.411	-0.267	0.161
μ_{f1}	-0.335 ± 0.122	0.899	-0.025
μ_{f2}	0.562 ± 0.222	0.521	-0.025
μ_{f3}	0.223 ± 0.507	-0.270	-0.025
σ^2	0.665 ± 0.097	0.655	0.848
β	0.209 ± 0.092	0.242	0.250
ε	-0.156 ± 0.206	-0.073	0.010
τ_1	0.953 ± 0.060	[1]	0.088!(=p)
τ_2	0 +	[0.5]	0.088!(=p)
τ_3	0 +	[0]	0.088!(=p)
Log LH	-439.616	-442.333	-442.915
χ^2		5.434	6.598
d.f.		2	3
p		0.066	0.086

Notes: Same as Table **4**.

support this field theory. The three factors of the present study are exactly the same as our previous findings [5] of five Indian populations, which suggest that the internal structure of the dermatoglyphic variables represented by the factors are common irrespective of different ethnic/ geographical populations.

Familial Correlations Based on Individual Traits

The spouse correlations exhibit low values and some are even negative ($p > 0.05$); indicating the absence of assortative matting in the studied population for 22 dermatoglyphic traits (Table **1**). It is evident that the strength of correlations of different traits is different, perhaps due to genetic interaction with the environment and thus certain dermatoglyphic characters revealed highly heritable than some other dermatoglyphic traits. There is no striking difference in the correlation values of the three kinds of parent-child combinations, but these correlations are positive and significant, with few exceptions. These results suggest the strong involvement of family factors (presumably genetics) in determining the variation of dermatoglyphic traits, which was supported by several earlier studies on family resemblance [4,5,7,9,33,53,56-58]. Similar results also appear from family correlations based on factors (Table **3**). Falconer [59] indicated that the correlations between genetically related individuals are significantly different, which is fully supported by our present results. The correlation values are slightly lower than the theoretical value (0.5) in the case of parent-offspring and sib-sib pairs. However, none of these correlations is significantly different from the expected value.

Segregation Analysis Based on Factors

The goal of the present report is to use family data to identify Mendelian mechanisms with respect to dermatoglyphic traits. Unfortunately, the existing information is very limited [33,5]. Therefore, we are unable to provide an accurate explanation of our present results compared with the earlier studies. We can only discuss here the results of our comprehensive analyses. Two traditional criteria are required to derive a major gene effect. (1) The environmental hypothesis must be rejected with a chi-square test in which $p<0.05$, indicating that the general model fits better than the environmental hypothesis. (2) The Mendelian hypothesis must be accepted with a chi-square test in which $p>0.05$, indicating that the general model does not fit significantly better than the Mendelian hypothesis. Evidence of a major gene effect (the Mendelian model was favored) was found for Factor 1 and Factor 2 where the environmental model was strongly rejected. However, in analyzing Factor 3 (Table **6**), we were not able to reject either the Mendelian model ($p=0.066$), or the Equal τ values or the Environmental model ($p=0.086$). Thus, the major gene assumption was not accepted for this trait. This result fully supports our earlier findings [5] involving five Indian populations. Earlier investigations support polygenic effects on the a-b count trait [30,60-63] attempted to test both polygenic and major gene effects on a-b ridge count with Brazilian families, but they

were unable to draw any conclusions between Mendelian transmission and the lack of transmission models. Therefore, hypothesis of an accident occurring in the developmental process of a-b count has to be investigated further. Our present result is in good agreement with the results and interpretation of segregation analysis on a-b ridge count [30, 34].

CONCLUSION

Major gene involvement with Mendelian expectation regarding finger dermatoglyphics is confirmed for all analyzed traits. However there is no evidence of significant support for major gene effect or environmental effect on palmar a-b ridge counts.

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