Genetics of Dermatoglyphic Asymmetry in Vaidyas of West Bengal, India

M. SENGUPTA[1] AND B. KARMAKAR

Abstract In this study of the genetics of dermatoglyphic asymmetry, we collected bilateral finger and palm prints of 824 individuals from 200 families including 2 generations from an endogamous caste (Vaidya) in Barasat, North 24-Parganas District, West Bengal. Two main types of asymmetry (fluctuating asymmetry and directional asymmetry) were calculated between the two hands. The study includes familial correlation between first-degree relatives, principal-components analysis, and maximum-likelihood-based heritabilities (by pedigree analysis). We found, first, that familial correlations in all possible pairs of relationships (except spouse correlation) were weak but positive; some were even statistically significant. No indication of assortative mating was observed, but the influence of maternal environment could not be discarded. The results also showed that X-chromosome linkage does not seem to be involved. A second major finding is that five principal factors could be extracted from all these asymmetric traits, explaining 74.207% of the overall cumulative variance. Asymmetry of finger and palmar areas were clearly separated by factor. In addition, the heritabilities of the extracted five factors were in the range of 8–24%. These estimates are in agreement with some previously published data. The heritabilities of the factors describing palmar asymmetry are slightly lower than those describing finger asymmetry. The present results support the hypothesis that both types of asymmetry have a genetic basis and are influenced by the intrauterine environment.

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; others are acquired. Some features, such as asymmetry of dermatoglyphic patterns and location of the heart, lungs, liver, spleen, and so on, appear to be strongly predetermined and not easily perturbed by the environment. Other features, such as calvarial form, diaphyseal bone length, and the size of muscle attachment, may well be acquired through preferential use and disuse (Schultz 1937). In the literature two types of asymmetry are

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studied: (1) fluctuating asymmetry (FA), which is the random deviation (irrespective of sign) from perfect bilateral symmetry (Arrieta et al. 1993); and (2) directional asymmetry (DA), which reflects a consistent bias of a character toward systematically greater development on one side (considering sign) (Palmer and Strobeck 1986).

Inborn asymmetry has been found to be related to prenatal stress, including chromosomal disharmonies (Zankl et al. 2003), nutritional deprivation (Nosil and Reimchen 2001), infection (Ariji et al. 2002), health status of the mother (Kieser et al. 1997), inbreeding (Bailit et al. 1970), consanguinity (Hershkovitz et al. 1993), and heterozygosity of the individual (Leary et al. 1983) or population (Biemont 1983). Although it is generally believed that genetic information for both sides of an individual is the same (Potter and Nance 1976), an individual's inability to buffer against environmental (intrauterine) and genetic perturbations (Polak and Starmar 2001) causes asymmetry (specially FA). Asymmetry is thus considered a good indicator of overall developmental homeostasis (Palmer and Strobeck 1997).

Recently, a number of scientists have become interested in studying asymmetry because it has different applied aspects, including neurology (Verenich 1996), visual search (Nicholls et al. 2004), hormone levels (Sorenson et al. 1993), disease incidence (Toth et al. 2004), congenital anomalies (Katznelson et al. 1999), and population variation (Karmakar et al. 2001; Sengupta and Karmakar 2004a). However, the actual utility of asymmetry is limited because of inadequate knowledge of its genetic nature. Therefore a thorough understanding of the mode of inheritance of asymmetry is essential.

A variety of bilateral quantitative asymmetries have been examined. To understand the genetic mechanism of asymmetry, dermatoglyphics is more suitable because it is easily quantifiable through noninvasive methods and not subject to environmental alterations after birth. As far as the literature is concerned, few investigators have considered the genetics of asymmetry on finger pattern types (Bener 1981), finger ridge counts (Loesch and Martin 1982), or the main line index (Karmakar 1990). Moreover, the results of these studies vary widely. Some studies implicate an absence of any genetic component (Holt 1954; Arrieta et al. 1993; Bogle and Reed 1997). To the contrary, slight hereditary components of asymmetry of finger and palmar ridge counts have been reported (Singh 1970; Trimble et al. 1971; Mi and Rashad 1977; Jantz 1979; Martin et al. 1982; Poliukhov 1984), and some recent studies have found significant heritability values, varying between 20% and 45% (Livshits and Kobyliansky 1989; Moller and Thornhill 1997; Pechenkina et al. 2000). Another source of evidence implying a genetic basis for dermatoglyphic FA comes from well-established differences among populations (Jantz 1975; Dittmar 1998).

Thus, despite some efforts and approaches regarding the genetics of asymmetry, this enigma is still unsolved, probably because of the lack of application of appropriate statistical analysis in previous studies, even though statistical methods have rapidly progressed and computers have become widely available.

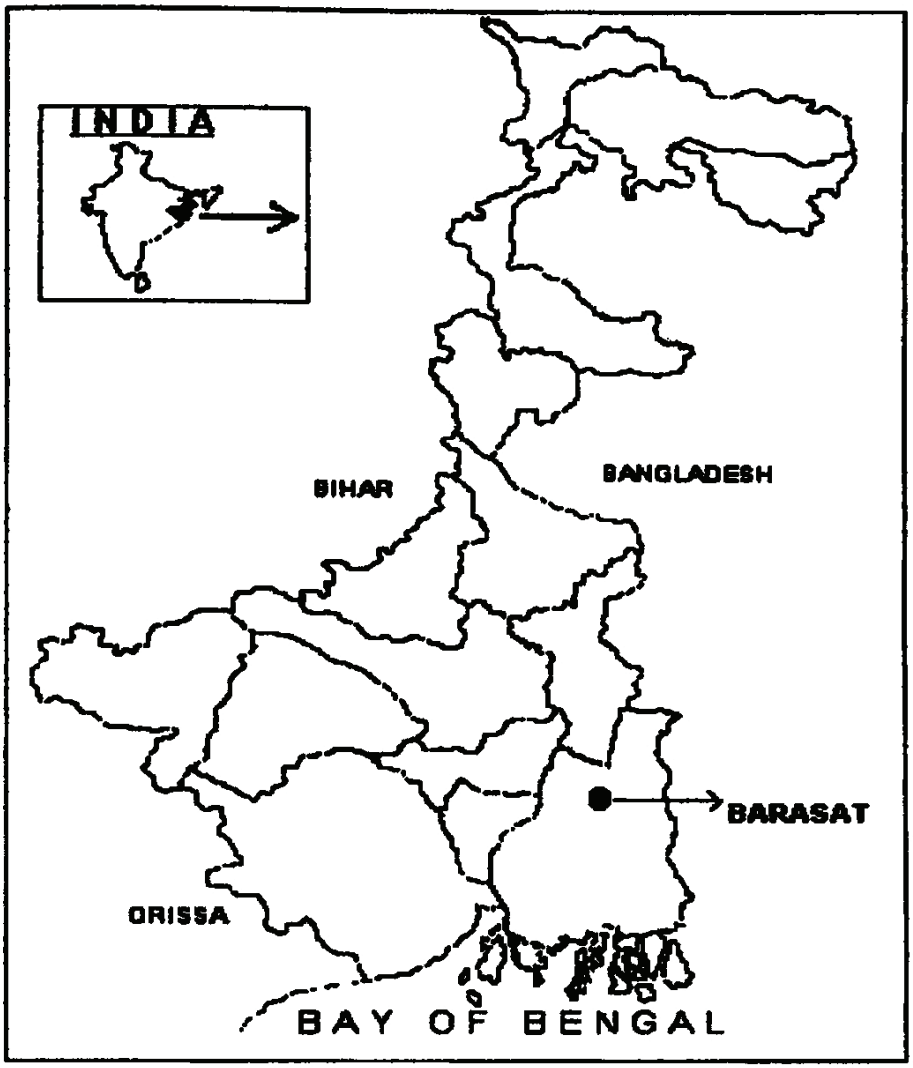

Figure 1. Map of West Bengal showing the location of the studied population in the North 24-Parganas District.

Therefore in the present study we examine the nature and extent of familial relationships of dermatoglyphic asymmetry (both FA and DA). Furthermore, we aim to identify the causal factors presumed to be operating on the asymmetric traits and to estimate their likelihood-based heritabilities.

Materials and Methods

Study Population. The present study has been confined to a Bengali Hindu caste group (Vaidyas) from the area of Barasat in the North 24-Parganas District of West Bengal, India. Data on 200 families, consisting of 824 individuals of 2 generations (living husband and wife with at least 2 children), were taken into consideration. The geographic location of the study area is shown in Figure 1.

Table 1. Sample Sizes for the Vaidya Study Population

Sex	*Parental Generation*	*Offspring Generation*	*Total*
Male	200	229	429
Female	200	195	395
Total	400	424	824

The sample size and the proportion of male and female children according to different family sizes are given in Tables 1 and 2, respectively.

In genetic analysis it is important to restrict the family data to an endogamous population because admixture of genetically heterogeneous populations leads to spurious estimates of parameters. Thus the marriage system of the Vaidyas is described here [a detailed description of the study population has been presented in our previous papers (Sengupta and Karmakar 2003, 2004b)]. Consanguineous marriage is prohibited among the Vaidyas. They adhere to an orthodox marriage pattern; that is, they are strictly endogamous but practice clan (*gotra*) exogamy. Of course, with the passage of time and the ensuing advances made in civilization, these barriers are no longer rigid or watertight. For this reason only the families of pure Vaidya descent were taken into account. Although the possibility of intercaste marriage in an ancestral generation cannot be totally ruled out, the sampled families have no such record in the last two to three generations.

Dermatoglyphic Variables. The prints were collected using the widely used traditional ink method proposed by Cummins and Midlo (1976). Seven traits from the fingers and five traits from the palm were taken into consideration, and each trait was measured in both hands.

Print Analysis. Following Galton's classification, we classified patterns into four types: arches, radial loops, ulnar loops, and whorls. Quantitatively, we analyzed the prints using the method of Holt (1968). All the data collection and print

Table 2. Proportion of Male and Female Children in Different Family Sizes

Number of Children in the Family	*Number of Families*	*Male Children*		*Female Children*		*Total Children*	
		N	*%*	*N*	*%*	*N*	*%*
2	179	193	45.52	165	38.92	358	84.43
3	18	29	6.84	25	5.90	54	12.74
4	3	7	1.65	5	1.18	12	2.83
Total	200	229	54.01	195	45.99	424	100.00

Table 3. Studied Traits and Their Abbreviations

Dermatoglyphic Asymmetry	*Abbreviation*
Fluctuating asymmetry (FA)	
FA of ridge count of finger 1	RC1FA
FA of ridge count of finger 2	RC2FA
FA of ridge count of finger 3	RC3FA
FA of ridge count of finger 4	RC4FA
FA of ridge count of finger 5	RC5FA
FA of total ridge count	TRCFA
FA of finger pattern intensity index	FPIFA
FA of a-b ridge count	ABFA
FA of b-c ridge count	BCFA
FA of c-d ridge count	CDFA
FA of atd angle	ATDFA
FA of palmar pattern intensity index	PPIFA
Directional asymmetry (DA)	
DA of ridge count of finger 1	RC1DA
DA of ridge count of finger 2	RC2DA
DA of ridge count of finger 3	RC3DA
DA of ridge count of finger 4	RC4DA
DA of ridge count of finger 5	RC5DA
DA of total ridge count	TRCDA
DA of finger pattern intensity index	FPIDA
DA of a-b ridge count	ABDA
DA of b-c ridge count	BCDA
DA of c-d ridge count	CDDA
DA of atd angle	ATDDA
DA of palmar pattern intensity index	PPIDA

analyses were done by a single investigator (M. Sengupta) to avoid interobserver error.

Asymmetry Analysis. Following the work of Jantz and Webb (1980), FA and DA were measured from each individual trait as the absolute and signed value of difference between the right (R) and left (L) hands, respectively:

$$\mathrm{FA}_i = (X_{i,R} - X_{i,L}), \tag{1}$$

$$\mathrm{DA}_i = (X_{i,R} - X_{i,L}), \tag{2}$$

where $X_{i,R}$ and $X_{i,L}$ are the individual values for the trait on the right and left side of the body, respectively. The studied variables are listed in Table 3.

Making Pairs of Relatives for Familial Correlation. Fifteen possible combinations of relatives were made for each family. The nomenclature for these pairs (along with their number) is presented in Table 4.

Table 4. Nomenclature of the Possible Relationships

Relationship	*Abbreviation*	*Number of Pairs*
Interclass		
Husband-wife	HW	200
Father-son	FS	229
Father-daughter	FD	195
Mother-son	MS	229
Mother-daughter	MD	195
Father-child	FC	424
Mother-child	MC	424
Parent-child	PC	848
Midparent-son	MidS	229
Midparent-daughter	MidD	195
Midparent-child	MidC	424
Intraclass		
Brother-brother	BB	152
Brother-sister	BS	240
Sister-sister	SS	110
Sib-sib	Sib	502

Statistical Analysis To normalize the data, we converted each value for a dermatoglyphic trait using Fisher's Z transformation:

$$Z = \frac{X_i - \overline{X}}{\mathrm{SD}}, \tag{3}$$

where X_i, $\overline{X}$, and SD are the individual measurement, the average, and the standard deviation of the trait, respectively.

Analysis of familial correlation (r) was done with the help of two methods: (1) interclass correlation and (2) intraclass correlation. The interclass correlation was calculated as the degree of resemblance between a parent and child or between a midparent and child and was computed with the usual Pearson's correlation. The intraclass correlation used Fisher's (1958) formula to estimate the degree of resemblance between two siblings. A t test of significance was carried out on the estimated correlation coefficient.

We also performed a principal-components analysis. Using a genetic correlation matrix between the studied traits, we constructed principal factors with varimax rotation of principal components to maximize the sum of squares of the loading of each factor. Factor scores were computed on the total pedigree sample and then used for further analysis. These analyses were carried out using Excel97 and SPSS (version 7.5).

Finally, we estimated heritability (h^2). Maximum-likelihood-based estimates of heritability were obtained using the Pedigree Analysis Package (PAP) (Hasstedt 1994).

Results

After the data were normalized using Fisher's Z transformation, they were subjected to further analyses. The results of the interclass and intraclass correlation coefficients are presented in Table 5.

For FA traits all pairs of relationships were positively correlated with respect to the asymmetric traits, with the exception of the husband-wife correlation. However, only 26 (15%) out of the 168 possible combinations (except spouse), were statistically significant ($p < 0.05$). Out of 12 asymmetric traits, father-son correlation was higher than father-daughter correlation for 8 traits (67%) and mother-daughter correlation was greater than mother-son correlation for 5 traits (42%); 33% of the traits showed similar mother-daughter and mother-son correlations. On the other hand, father-son correlation was higher than mother-son correlation for 9 traits (75%), and father-daughter correlation was greater than mother-daughter correlation for 6 traits (50%). All traits showed higher father-child similarity than mother-child similarity, except for the FA of digit 3 ($r = 0.09$ for mother-child; $r = 0.06$ for father-child), b-c ridge count ($r = 0.08$ for mother-child; $r = 0.04$ for father-child), and c-d ridge count ($r = 0.07$ for mother-child; $r = 0.04$ for father-child). Other traits had higher midparent-child correlation than parent-child correlation, except for the FA of digit 3 ($r = 0.07$ for parent-child; $r = 0.01$ for midparent-child) and digit 4 ($r = 0.07$ for parent-child; $r = 0.04$ for midparent-child). A higher similarity between siblings (sib-sib) than between parent and child was observed for all traits, except the FA of c-d ridge count ($r = 0.06$ for parent-child; $r = 0.04$ for sib-sib).

For signed bilateral difference (DA), husband and wife were almost noncorrelated ($p > 0.05$). Although the other combinations of relationships were positively correlated, only 38 combinations were statistically significant out of 168 (23%). The DA of a-b ridge count was not statistically significant for any pair of relationship. For 7 asymmetric traits out of 12 (58%), father-son correlations were higher than father-daughter correlations, and mother-daughter correlations were higher than mother-son correlations for 8 traits (67%). On the other hand, mother-son correlation was greater than father-son correlation for 7 traits (58%), whereas mother-daughter correlation was higher than father-daughter correlation for 8 traits (67%). Fifty percent of the asymmetric traits (6 out of 12 traits) had higher similarity between mother and child than between father and child. Others DA traits showed higher midparent-child correlation than parent-child correlations, with the exception of digit 5 ($r = 0.07$ for midparent-child; $r = 0.09$ for parent-child), total ridge count ($r = 0.06$ for midparent-child; $r = 0.10$ for parent-child), finger pattern intensity ($r = 0.11$ for midparent-child; $r = 0.15$ for parent-child), and atd angle ($r = 0.05$ for midparent-child; $r = 0.06$ for parent-child). For all traits except signed asymmetry of c-d ridge count ($r = 0.07$ for parent-child; $r = 0.06$ for sib-sib) and finger pattern intensity ($r = 0.15$ for parent-child; $r = 0.13$ for sib-sib), parent-child correlation is lower than sib-sib correlation.

Table 5. Inter- and Intraclass Familial Correlation of Dermatoglyphic Asymmetry

Variable	*HW*	*FS*	*FD*	*MS*	*MD*	*FC*	*MC*	*PC*	*MidS*	*MidD*	*MidC*	*BB*	*BS*	*SS*	*Sib*
n^a	200	229	195	229	195	424	424	848	229	195	424	152	240	110	502
RC1FA	0.04	0.13[b]	0.06	0.04	0.04	0.04	0.04	0.03	0.18[c]	0.02	0.09	0.05	0.01	0.13	0.09
RC2FA	−0.07	0.02	0.10	0.03	0.03	0.03	0.00	0.02	0.01	0.12	0.06	0.18[b]	0.18[c]	0.13	0.15[c]
RC3FA	0.02	0.06	0.06	0.02	0.16[b]	0.06	0.09	0.07[b]	0.01	0.04	0.01	0.02	0.03	0.17	0.11[b]
RC4FA	0.09	0.09	0.07	0.08	0.08	0.08	0.08	0.07[b]	0.04	0.07	0.04	0.09	0.09	0.12	0.10[b]
RC5FA	−0.10	0.15[c]	0.08	0.03	0,02	0.12[b]	0.02	0.02	0.15[b]	0.03	0.07	0.14	0.04	0.05	0.08
TRCFA	−0.03	0.11	0.03	0.00	0.01	0.07	0.01	0.04	0.22[c]	0.09	0.16[c]	0.10	0.15[b]	0.18	0.09[b]
FPIFA	−0.08	0.12[b]	0.07	0.07	0.05	0.09	0.06	0.06	0.19[c]	0.11	0.10[b]	0.07	0.13	0.17	0.12[b]
ABFA	−0.07	0.04	0.08	0.06	0.09	0.08	0.08	0.07[b]	0.02	0.13	0.07	0.01	0.05	0.10	0.08
BCFA	−0.06	0.09	0.02	0.03	0.09	0.04	0.08	0.06	0.08	0.18[c]	0.13[c]	0.08	0.11	0.13	0.08
CDFA	−0.03	0.07	0.05	0.07	0.05	0.04	0.07	0.06	0.10	0.19[c]	0.14[c]	0.08	0.12	0.06	0.04
ATDFA	0.13	0.05	0.06	0.02	0.02	0.00	0.00	0.00	0.07	0.05	0.01	0.03	0.01	0.05	0.01
PPIDA	−0.08	0.09	0.01	0.01	0.02	0.05	0.00	0.03	0.08	0.01	0.05	0.07	0.10	0.00	0.07
RC1DA	0.02	0.01	0.07	0.03	0.09	0.04	0.06	0.05	0.04	0.05	0.05	0.22[c]	0.13[b]	0.04	0.13[b]
RC2DA	−0.09	0.10	0.04	0.05	0.14[b]	0.07	0.09	0.08[c]	0.03	0.06	0.04	0.12	0.07	0.15[b]	0.08
RC3DA	−0.04	0.00	0.03	0.06	0.00	0.01	0.03	0.01	0.11	0.12	0.01	0.06	0.02	0.18[b]	0.09[b]
RC4DA	0.08	0.01	0.07	0.05	0.04	0.03	0.01	0.02	0.10	0.07	0.09	0.01	0.19[c]	0.09	0.11[b]
RC5DA	0.02	0.12[b]	0.10	0.08	0.05	0.11[b]	0.07	0.09[c]	0.10	0.04	0.07	0.19[b]	0.16[c]	0.08	0.14[c]
TRCDA	−0.06	0.12[b]	0.11	0.14[b]	0.04	0.11[b]	0.09	0.10[c]	0.08	0.04	0.06	0.13	0.25[c]	0.03	0.14[c]
FPIDA	−0.01	0.20[c]	0.18[b]	0.06	0.20[c]	0.19[c]	0.12[c]	0.15[c]	0.13[c]	0.09	0.11	0.13	0.18[c]	0.09	0.13[c]
ABDA	−0.09	0.02	0.02	0.04	0.08	0.02	0.06	0.04	0.08	0.10	0.09	0.06	0.06	0.04	0.05
BCDA	0.07	0.07	0.02	0.01	0.07	0.05	0.04	0.03	0.20[c]	0.12	0.17	0.10	0.06	0.02	0.05
CDDA	−0.07	0.02	0.01	0.06	0.07	0.01	0.06	0.07[b]	0.15[c]	0.17[c]	0.16[c]	0.09	0.08	0.05	0.07
ATDDA	−0.08	0.09	0.01	0.01	0.02	0.05	0.01	0.03	0.08	0.01	0.05	0.07	0.10	0.01	0.09[b]
PPIDA	0.06	0.02	0.05	0.08	0.11	0.03	0.09	0.06	0.02	0.12[b]	0.05	0.08	0.02	0.08	0.06

a. Number of pairs.
b. $p < 0.05$.
c. $p < 0.01$.

Table 6. Factor Analysis of Dermatoglyphic Asymmetry and Heritability of Each Factor[a]

Variable	*Factor I*	*Factor II*	*Factor III*	*Factor IV*	*Factor V*	*Likelihood-Based Heritability (h^2)*
TRCFA	0.957	–	–	–	–	0.19
TRFFA	0.833	–	–	–	–	
RC1FA	0.581	–	–	–	–	
RC3FA	0.519	–	–	–	–	
RC5FA	0.505	–	–	–	–	
RC4FA	0.499	–	–	–	–	
RC2FA	0.354	–	–	–	–	
TRCDA	–	0.821	–	–	–	0.24
TRFDA	–	0.711	–	–	–	
RC3DA	–	0.699	–	–	–	
RC4DA	–	0.479	–	–	–	
RC2DA	–	0.417	–	0.308	–	
RC5DA	–	0.395	–	–	–	
RC1DA	–	0.392	–	–	–	
CDFA	–	–	0.864	–	–	0.17
CDDA	–	–	0.783	–	–	
BCFA	–	–	0.771	–	–	
BCDA	–	–	0.680	–	–	
TRPDA	–	–	–	0.620	–	0.12
ATDDA	–	–	–	0.598	–	
ATDFA	–	–	–	−0.524	–	
TRPFA	–	–	–	−0.468	–	
ABDA	–	–	–	–	0.730	0.08
ABFA	–	–	–	–	−0.693	
VP[b]	12.991	10.539	9.636	6.066	5.974	
CV[c]	24.263	43.531	56.166	66.233	74.207	

a. Rotation method: Varimax with Kaiser normalization, loading values below 0.30 are omitted.
b. Variance explained by each factor.
c. Cumulative proportion of explained variance.

In the principal-components analysis five principal components were extracted (Table 6), which explained 74.207% of the overall cumulative variance. Factor structure showed that asymmetry of finger and asymmetry of palm had no association. Factor I, which includes the FA of finger ridge counts, described 24.263% of the total variance. This factor can be called the FA of finger ridge count factor. Factor II (the DA of finger ridge count factor) explained 19.268% of the total variance. Factor III (12.635% of the total variance) consists of asymmetry of digits 2 and 3 interdigital ridge counts, and factor IV (the asymmetry of atd angle and palmar pattern intensity factor) explained 10.067% of the total variance. Factor V includes only the asymmetry of a-b ridge counts. This is a unique factor and explains 7.974% of the total variance.

For each factor the maximum-likelihood-based heritabilities were estimated using the model parameters in PAP. The results are presented in the last column of Table 6. Factor II, which contains DA of finger ridge counts, showed the highest heritability, followed by factor I ($h^2 = 0.19$); factor V (asymmetry of a-b ridge count) revealed the lowest value ($h^2 = 0.08$).

Discussion

Familial correlations in the present study unambiguously show weak but positive involvement of family factors in the manifestation of asymmetry. Fourteen percent of FAs and 21% of DAs are significantly different from 0, suggesting the contribution of some genetic factors to bilateral asymmetry; this finding supports several earlier works (Parsons 1973; Martin et al. 1982; Arrieta et al. 1993; Pechenkina et al. 2000). However, it is well known that when additive genes with independent effects without dominance are present, the correlation is 0.5 for the parent-child and sib-sib pairs (Fisher 1918) and 0.71 for the midparent-child pair (Penrose 1949). The strength of correlation of both FAs and DAs 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 (Jantz 1979; Malhotra 1987). Our results are also consistent with Martin et al. (1982), who suggested that "there is a genetic component in asymmetry variation between hands but environmental factors are more important" (cited by Arrieta et al. 1993, p. 561).

The correlation coefficients for both FA and DA in the present study 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 (1988) 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. 1991). This similarity between general dermatoglyphic traits and their bilateral asymmetry is again compatible with the suggestion of Jantz (1975) that the genetic mechanisms responsible for ridge counts may also mediate their bilateral asymmetry.

Departures from random mating and consanguinity lead to changes in the correlation coefficient. But very low (even negative) spouse correlation in the present study indicates the absence of any assortative mating for these asymmetric traits. Thus the present correlation values between different pairs of relatives (other than husband-wife) may not be affected by assortative mating. Absence of assortative mating on the asymmetry of qualitative dermatoglyphic traits has been reported in other studies (Bener and Erk 1979; Bener 1981).

According to Mather and Jinks (1963), if X-chromosome-linked genes are involved in bilateral differences, the father-daughter and mother-son correlations would be expected to be higher than the mother-daughter correlation, and the father-son correlation would be expected to be lowest, because in this case there is no X-chromosome contribution. On the other hand, brother-brother and sister-sister correlations would be higher than the brother-sister correlation. The present results of both FA and DA traits contradict this hypothesis, however; 75% of the FA traits and 67% of the DA traits showed that father-son correlation was either higher than or equal to father-daughter correlation. Thus X-chromosome linkage does not seem to be involved in these asymmetric traits. Bener and Erk (1979) found that X-chromosome linkage does not appear to be operating in the development of dermatoglyphic asymmetry in a Polish population. But it has been shown in the case of sex chromosome aneuploidies that extra X and Y chromosomes have a considerable effect on finger ridge count and their asymmetry (Barlow 1973; Jantz 1977).

Parent-child and midparent-child resemblance in the present study differed among different traits, suggesting varying degrees of genetic components. Twenty-five percent of FA traits and 50% of DA traits showed higher mother-offspring than father-offspring correlation (although the differences are mostly nonsignificant); this would indicate a possibility of maternal effect on these asymmetric traits. To the contrary, Bener and Erk (1979) observed low correlation between mother and daughter in bilateral whorl asymmetry in 539 Polish families. They explained this finding as "a remarkable absence of maternal influence through either egg organization or maternal-fetal hormonal effects" (p. 353). Except for this study, most of the earlier studies have reported a significant maternal effect on bilateral asymmetry (Pons 1961; Parsons 1973).

With some exceptions, sib-sib correlations in the present study are slightly higher than parent-child correlations. Pons (1961) also observed higher sib-sib correlation ($r = 0.15$) than parent-child correlation ($r = 0.09$) in the asymmetry of main line transverseness in 400 Spaniards. Pons suggested that this was due to intrauterine environmental influence; during prenatal development "sibs share a relatively more homogeneous environment than parents and offspring who belong to two different generations" (Kaur and Singh 1981, p. 337). Another reason for lower parent-child than sib-sib correlation may be genetic dominance, which also decreases parent-child and midparent-child correlations to a much greater extent than sib-sib correlation (Matsuda 1973). However, no evidence of genetic dominance on asymmetry of dermatoglyphic traits has been reported in the literature (Bener 1979; Bener and Erk 1979). Thus the higher sib-sib than parent-child correlation may be due to environmental factors rather than dominance.

In order to reduce the number of interrelated variables to a few factors, we performed a principal-components analysis on the studied asymmetric traits. Froehlich (1976) 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 (Micle and Kobyliansky 1986, 1992; Karmakar et

al. 2001). In the first two factors (out of the five extracted factors) of the present study, the FA and DA of digital ridge counts were clearly separated. But for the last three factors (which explain the asymmetry of palmar dermatoglyphics), both FA and DA fall into the same factor, indicating some relationship between the genetic factors of DA and FA.

In a previous study, Micle and Kobyliansky (1986) studied 46 dermatoglyphic traits, including FA and DA, where correlation matrices of the traits were used in a principal-components analysis. Ten factors (68%) were extracted, of which five were clearly identified: (1) digital pattern size, (2) palmar lines, (3) a-b ridge count, (4) finger ridge count diversity, and (5) FA factor. Later, Micle and Kobyliansky (1992) performed a principal-components analysis of 42 dermatoglyphic traits, including the indexes of intra-individual diversity (differences between nonhomologous fingers) and asymmetry, in a Jewish population. Ten factors were extracted, explaining 70% of the total variance. The investigators found that, in both sexes, factor I had high loading for the FA of ridge counts (digits 2 and 4) and factor II had high loading for DA of some diversity indexes for left and right hands. Sex differences occurred in the extraction order of the remaining factors. Another study by Karmakar et al. (2001), on five endogamous populations of India, should be mentioned here. Karmakar and colleagues conducted a principal-components analysis on 38 dermatoglyphic variables representing the indexes of intra-individual diversity and asymmetry. Ten factors were extracted, of which four were clear: (1) intra-individual finger ridge count diversity, (2) DA, (3) FA, and (4) bilateral asymmetry factor. Our results here do not contradict these studies, but because we considered only asymmetric traits (not diversity), we extracted five principal components rather than ten. However, the universality of the first factor (digital pattern size) and of the separation of the a-b ridge count from other palmar dermatoglyphic traits by principal-components analysis (Karmakar et al. 2002) is also observed in the present asymmetric traits. This similarity between general dermatoglyphic traits and their asymmetry may indicate biological validity of underlying genetic components.

The results of the maximum-likelihood-based heritability estimation showed that there is a small hereditary component for all factors (8–24%), which supports the results of the present familial correlation. The heritability of factor II (DA of digital dermatoglyphics) is higher than that of factor I (FA of digital dermatoglyphics). This is not inconsistent with the concept that DA is genetically controlled, but FA is a poorly inherited trait and is considered a result of the inability of the organism to buffer the negative influences of disturbing developmental factors (Micle and Kobyliansky 1992). On the other hand, factors that represent finger dermatoglyphic asymmetry (factors I and II) were slightly higher than those representing palmar dermatoglyphic asymmetry (factors III–V), especially factor V (a-b ridge count), which showed the lowest heritability ($h^2 = 0.08$). By examining 92 female and 105 male twin pairs, Arrieta et al. (1993) also found low heritability (for females, $h^2 = 0.317$; for males,

$h^2 = -0.180$) of asymmetry of a-b ridge count. They explained that the "interdigital area develop[s] over a longer time period than do digital dermal ridges" (p. 561), and as a result the interdigital area is exposed to the intrauterine environment for a longer time, causing lower heritability. A similar interpretation was made by Malhotra et al. (1991) for an Indian population. Malhotra and colleagues studied ridge count asymmetry among different dermatoglyphic areas and postulated that certain areas such as the "interdigital area a-b are more vulnerable to developmental stress/environmental insults compared to other areas" (p. 165).

We have already mentioned that few inheritance studies consider dermatoglyphic asymmetry. Especially in India, such studies are rare. For this reason we compared the present Indian population with other non-Indian populations. The present results are similar to the results of a study by Mi and Rashad (1977), who evaluated the heritability of bimanual asymmetry of finger ridge counts ($h^2 = 6$–22%) in 711 Hawaii families. Asymmetries of separate digits had higher heritability than in the present study, whereas asymmetry of total ridge count had a lower heritability than in the present study. Based on 221 pairs of twins and 80 pairs of opposite-sex siblings, Loesch and Martin (1982) found a combined estimate of heritability for all fingers ($h^2 = 0.28 \pm 0.07$) that is closer to the heritability found in the present study. Another study on a Russian sample (Pechenkina et al. 2000) revealed that FA showed weak but significant heritabilities, with values falling within the 20–35% range, which is slightly higher than the present results. Pechenkina and co-workers explained their high heritability estimates as a result of high genetic diversity of the people living in Moscow. But the present Vaidya population is endogamous, leading to lower genetic diversity, for which the present Bengali groups might have lower heritability than the Russian group. All the other discrepancies in the heritability values of the present study and earlier studies may be due to different methodologies, various sample sizes, or different ethnic groups having different geographic and environmental backgrounds. These differences in results are consistent with the suggestion of Mi and Rashad (1977) that the regressions on bimanual differences of pattern type counts vary from one ethnic group to another.

Conclusion

Our results indicate that asymmetry of dermatoglyphic traits has a genetic basis ($h^2 = 8$–24%), although effects of intrauterine environment are also present. The results support the idea postulated by several investigators that asymmetry provides a measure of developmental instability in humans. The principal-components analysis of asymmetry clearly separated finger and palmar traits. Lower heritability of interdigital palmar dermatoglyphic traits than of digital dermatoglyphic traits suggests that the palmar dermatoglyphic traits may be a better indicator of developmental homeostasis than finger dermatoglyphic traits.

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