Genetics of Anthropometric Asymmetry in an Indian Endogamous Population—Vaidyas

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ABSTRACT To understand the genetics of Fluctuating Asymmetry (FA) and Directional Asymmetry (DA), the present study comprised 14 bilateral morphometric traits from 200 Vaidya families including 824 individuals (of two generations) from North 24 Parganas, West Bengal. The statistical analysis included: Regression analysis to remove the age effect, Familial correlation, Heritability estimation, Principal Component Analysis and Segregation Analysis (SA) using genetic model test. The obtained results revealed little effect of genetic factor and considerable amount of environmental influence on anthropometric asymmetry. The results support the idea postulated by several previous authors that FA provides a measure of developmental instability in man. The contribution of heredity on these asymmetric variables is not unimportant but that of the common environment is very substantial. The magnitude of heritability of DA traits is slightly higher than that of FA traits. Five principal factors were detected from these asymmetric traits (three factors are on asymmetry on length, head, and breadth; while last two factors represent the asymmetry of diameters). SA did not suggest any evidence of major gene contribution. But the involvement of minor genes or polygenes could not be discarded. As the study on SA of asymmetry in man is limited, similar other studies are needed to confirm the result of the present study. Am. J. Hum. Biol. 19:399–408, 2007.

Most of the bilateral characteristics of an organism do not develop mirror images of one another, i.e. they showed some degree of asymmetry. It could be developmentally viewed as variation in growth about the median plane (Little et al., 2002). As genetic information for both sides is thought to be same (Potter and Nance, 1976), failure to buffer itself against developmental noise causes asymmetry (Bogle et al., 1994; Livshit and Kobyliansky, 1991; Palmer and Strobeck, 1997). Two types of asymmetries can be distinguished. Fluctuating asymmetry (FA) is the random difference between two sides (Livshit and Kobyliansky, 1989), whereas in Directional Asymmetry (DA) the dimension of one side of the body is consistently larger than the other (Palmer and Strobeck, 1986; Schell et al., 1985).

As there is limited number of studies dealing with this topic (Livshits and Kobyliansky, 1989), the cause of the development of asymmetry is yet to be clear. Asymmetries can develop in prenatal stage (Schultz, 1926), continue to develop in childhood (Van Dusen, 1939), and are found in adults (Malina and Buschang, 1984). It is suggested that in prenatal period, development of asymmetries presumably occur without influence of a preference for one side of the body in movement. The prenatal stress (physiological, nutritional, and disease factors) may cause a breakdown in the genetic linkage system that regulates bilaterally symmetric structure (Sharma and Bakshi, 2005). But after birth, during adolescence, it is due to the preference of the use of one side of the body (Plato et al., 1980; Schell et al., 1985), and therefore, differences in physical activity may affect anthropometric asymmetry (Little et al., 2002). Marked asymmetry is found to be associated with intensive unilateral activity such as in some sports and occupations (Kannus et al., 1996) involving "dominant/non-dominant arm differences in strength and muscle girth in highly skilled players/workers with a long history of such ac-

tivity" (Little et al., 2002). Apart from the above environmental influences, association between FA and season of birth was also reported (Benderlioglu and Nelson, 2004).

On the other hand, some earlier studies found that bilateral asymmetry can not be explained by purely environmental components. Schultz (1926) found marked asymmetry in the length of the foot in human fetuses of 4 months before any significant activity-related stress factors become operative. Garn et al. (1976) observed that the cortical thickness of the second metacarpal is greater on the right side in both right and lefthanders. Montoye et al. (1976) found no relationship between activity levels and degree of asymmetry in radiographic bone breadths of the second metacarpal. All these studies support that bilateral asymmetry may have some genetic basis. Palmer and Strobeck (1986) also suggested that individuals are genetically or developmentally directed to become asymmetrical and as a consequence, "the variation in R-L may no longer be a product of pure developmental noise".

But these previous studies could not elucidate the nature of asymmetry in the relative contribution of genetic and environmental factors. Hence, it is necessary to seek the answer whether the anthropometric asymmetry is inherited or is simply related to the preferred day-to-day use of one side of the body. Keeping this in mind the present communication is an attempt to understand whether it is under any genetic regulation, and if so, the magnitude and mode of heritable factors.

MATERIALS AND METHODS

Historical background

The studied population Vaidya is a Bengali caste group, who practices Hinduism. They are traditionally recognized as physician, though today they are giving up their ancestral occupations and increasingly taking to employment in diverse field. Their origin is a controversial issue. Some consider them to be an offshoot of the Brahmins who are intermarried with the other castes like Vaishya, Sudra, etc. But it is also claimed that they are direct descendents of Aryans who immigrated to Bengal. Genealogically, every Vaidya member is considered to be the descendent of one or the other ancient sages and is identified by the clan (*Gotra*). They are strictly endogamous since several generations, though they practice clan exogamy. Their endogamous nature makes them homozygous for different alleles. On the other hand, though there is some controversy (Leary, 1984, 1985), it has been estimated that the more homogeneous individual (Leary et al., 1983) or population (Kat, 1982) have higher FA (Livshits and Kobyliansky, 1987). This population is, therefore, an important source of information for this type of genetic study.

Sample

Data consists of 824 individuals from 200 Vaidya families inhabited at Barasat of North 24 Parganas in West Bengal. The pedigrees comprised living parent (Male: 200, Female: 200) with at least two children (Male: 229, Female: 195). Inquires were used to eliminate half-sib and adopted children. The families, which consist of the amputee members or handicapped individuals, are also excluded.

Some basic information including name, age, and sex were collected. Age range of the parental generation varied from 30 to 72 years, whereas that of the offspring generation ranged between 12 and 44 years. Using the standard anthropometric techniques and landmarks, 14 linear measurements (3 on head and 11 on limbs) were taken from each side of the body. Repeat visits to a household were required to obtain the data of all family members. Martin's slide and spreading calipers were used to take the measurements. Data was collected by a single investigator (first author) to exclude the inter-observer error. To minimize the intra-observer error, measurements were taken for three times and the average values were recorded. Same instruments were used for all individuals to minimize the instrumental error.

Statistical analysis

(a). FA and DA of each individual trait were measured respectively as the absolute and signed value of difference between the right (R) and left (L) hands (Jantz and Webb, 1980). The formulae are: $FA_i = |(X_iR - X_iL)|$, and $DA_i = (X_iR - X_iL)$. [where X_iR and X_iL are individual values for the trait on the right and left side of the body, respectively].

(b). To remove the age effect on anthropometric traits, the possible dependent variables were adjusted for age by regression analysis. These standardized residuals are the phenotype variables used for further analysis.

TABLE 1. Descriptive statistics of anthropometric asymmetry (FA and DA)

Variable	Male		Female	
	Mean	SD	Mean	SD
FA of physiognomic ear length	1.582	0.130	1.419	0.120
FA of physiognomic ear breadth	1.479	0.122	1.500	0.166
FA of ear length	1.174	0.108	1.292	0.114
FA of hand length	2.437	0.123	2.152	0.100
FA of hand breadth	1.295	0.063	1.569	0.059
FA of foot length	3.091	0.142	2.858	0.142
FA of foot breadth	1.926	0.166	2.429	0.531
FA of bicondylar diameter at elbow	1.479	0.118	1.515	0.110
FA of bistyloid diameter at wrist	1.984	0.475	1.619	0.118
FA of bicondylar diameter at knee	1.274	0.339	1.058	0.087
FA of bimalleolar diameter at ankle	3.037	2.898	2.472	0.125
FA of length of middle finger	1.177	0.094	1.028	0.089
FA of thumb length	1.149	0.093	1.183	0.095
FA of palm length	1.579	0.115	1.401	0.108
DA of physiognomic ear length	−0.440	0.200	−0.226	0.184
DA of physiognomic ear breadth	0.279	0.189	−0.058	0.224
DA of ear length	−0.035	0.159	−0.003	0.172
DA of hand length	0.237	0.188	0.279	0.150
DA of hand breadth	0.321	0.096	0.173	0.080
DA of foot length	0.484	0.234	0.376	0.231
DA of foot breadth	−0.226	0.266	0.520	0.582
DA of bicondylar diameter at elbow	0.716	0.175	0.855	0.167
DA of bistyloid diameter at wrist	−0.286	0.514	−0.051	0.200
DA of bicondylar diameter at knee	−0.293	0.361	−0.150	0.136
DA of bimalleolar diameter at ankle	−0.847	2.913	0.147	0.193
DA of length of middle finger	−0.544	0.141	−0.322	0.132
DA of thumb length	−0.084	0.148	0.086	0.151
DA of palm length	0.100	0.195	0.102	0.177

(c). The correlation coefficients (r) were used to measure the resemblance between the relatives. For husband-wife, parent-child and midparent-child, interclass correlations (by Karl Pearson) and for different sibling, intraclass correlation (by Fisher, 1958) were used. On all estimated correlation coefficient a t-test of significance was carried out.

(d). The heritability (h^2) of these traits was calculated by Falconer (1960). The average value obtained from parent-child regression and sib-sib correlations was used to get the best estimate of heritability.

(e). Using genetic correlation matrix between the studied traits, principal factors were extracted with varimax rotation of principal components. Factor scores were then computed for each individual of each pedigree sample.

(f). In final stage, to evaluate the mode of inheritance, complex segregation analysis was carried out using Maximum Likelihood Methods by "Pedigree Analysis Package" [PAP] (Hasstedt, 1994). The program estimates: the population frequency of the first of two major alleles A_1 and A_2 (P); three transmission probabilities (τ_g); the average trait value or genotypic value (μ_{gs}) of individuals having genotype g (A_1A_1, A_1A_2, and A_2A_2); the standard deviation (σ) in individuals having the same major gene (MG) genotype, and the trait variance (h^2).

This program finally finds the best fitting linear genetic model of the trait variability. The following genetic models have been used: (1) General Model, (2) Sporadic Model, (3) Environmental Model, (4) τ's equal to P, (5) Mendelian Model, and (6) No Polygenic Components for detail (Sengupta and Karmakar, 2004). The difference in the log-likelihood between the general and its sub-models is asymptomatically distributed as a χ^2 variable. The degrees of freedom (df) for different models depends on the number of constraints imposed by the sub-models.

RESULTS

As the age effect of asymmetric traits was not found, neither the adjustment to age on asymmetry level carried out nor the descrip-

tive statistics conducted for two generations separately could be estimated. The result including mean with standard deviation (SD) were presented in Table 1. For FA, the mean values range from 1.149 to 3.091 for male and from 1.028 to 2.858 for female. On the other hand, for DA, the values vary from −0.947 to 0.716 for male and −0.322 to 0.855 for female.

From Table 2 it is clear that the familial correlations of asymmetry are low in magnitude and most of them are statistically non-significant ($P > 0.05$). The result revealed that spouse correlation was very low and even some are negative. The correlations between other pairs of relationships are also low but positive. For FA, the values are statistically significant ($P < 0.05$) in few cases (34 out of 196 i.e. 17%). Out of 14 asymmetric traits, father-son correlation is higher than father-daughter for seven traits (50%); mother-daughter correlations are greater than both father-son and mother-son for eight traits (57%) and than father-daughter for 10 traits (71%); mother-child correlation is again higher than father-child for 10 traits (71%). Except bi-malleolar diameter at ankle ($r = 0.06$ for parent-child; $r = 0.05$ for sib-sib), other asymmetric traits have higher sib-sib correlation than parent-child, 64% of the sib-sib correlations are higher than midparent-child correlation.

For DA, only 45 combinations (out of 196 combinations) have significant correlations (22%). Out of 14 DA traits, father-son and mother-daughter correlations are respectively higher than father-daughter and mother-son for nine traits (64%). In the same way, father-son and mother-daughter correlations are respectively greater than mother-son and father-daughter for nine traits (64%). Seven variables out of 14 (50%) have higher similarity between mother and child than father and child. Except hand breadth ($r = 0.04$ for parent-child; $r = 0.03$ for midparent-child) and bi-malleolar diameter at knee ($r = 0.07$ for parent-child; $r = 0.05$ for midparent-child), parent-child correlation is lower than midparent-child correlation. Eight asymmetric traits (57%) have higher sibling resemblance than midparent-child. On the other hand, sib-sib correlation is higher than parent-child for all traits.

The last column of Table 2 represents the heritability coefficients, which are very low in magnitude. Sixty four percent of traits (i.e. 9 out of 14) have higher heritability of DA than of FA. The heritability coefficient of FA ranged between 8 and 18%. FA of palm length ($h^2 = 0.18$) has the highest heritability coefficient followed by FA of physiognomic ear length ($h^2 = 0.17$) and hand length ($h^2 = 0.17$). On the other hand, least heritability is found in FA of foot breadth ($h^2 = 0.08$). Heritability of DA traits varied between 10 and 20%. The highest asymmetry was found in the DA of thumb length ($h^2 = 0.20$) followed by foot length ($h^2 = 0.19$), whereas lowest value was found in bi-styloid diameter at wrist ($h^2 = 0.10$).

Table 3 represents the result of Principal Component Analysis. Five factors with eigen values greater than one explained 79.753% of the total variation. Factor I has highest loading on the asymmetry of length measurements of upper and lower extremities. The factor alone is responsible for 26.263%. It may be called "asymmetry of length measurements factors". Factor II explains 20.268% of the total variance and includes the asymmetry of head measurements (physiognomic ear length, physiognomic ear breadth, and eye length). Thus it may be termed as "asymmetry of head measurements factor". Factor III describes the asymmetries of breadth measurements of two extremities and explained 16.921% of the total variance. The factor may be called "asymmetry of length measurements factors". For factor IV and V, respectively FA and DA of different diameters of two extremities have high loading. They include 9.030% and 7.271% of the total variance, respectively. These factors can be respectively termed as "fluctuating and directional asymmetry of body diameter factor".

On the basis of the factor scores estimated from the principal component analysis, familial correlations have again been carried out. The result (Table 4) indicates that though all the values are very low as found in single asymmetric traits, 40% traits (30 traits out of 75) show significant difference.

Based on these factors, segregation analysis was carried out and presented in Table 5. Tables presented maximum likelihood estimates of the model parameters, $-2 \ln L$ values and respective χ^2 values with their degrees of freedom. The result of segregation analysis revealed that when five models were compared with the general model (Model 1), all models, except environmental model, were rejected ($P < 0.05$). The only model that could not be rejected by all five factors is environmental (model 3) or equal taus model ($\chi^2 <$

TABLE 2. Familial correlation and heritability estimation of anthropometric asymmetry

Variable	HW (200)	FS (229)	FD (195)	MS (229)	MD (195)	FC (424)	MC (424)	PC (848)	MidS (229)	MidD (195)	MidC (424)	BB (152)	BS (240)	SS (110)	Sib (502)	Mean (h^2)
FA of physiognomic ear length	0.02	0.08	0.13*	0.07	0.04	0.10*	0.06	0.08*	0.10	0.11	0.10*	0.15	0.09	0.12**	0.09*	0.17 ± 0.07
FA of physiognomic ear breadth	0.03	0.06	0.03	0.02	0.12	0.02	0.05	0.02	0.05	0.10	0.06	0.14	0.04	0.14	0.11**	0.13 ± 0.05
FA of ear length	0.01	0.02	0.01	0.01	0.06	0.02	0.03	0.01	0.01	0.04	0.01	0.18*	0.17**	0.29*	0.07	0.12 ± 0.02
FA of hand length	−0.02	0.10	0.11	0.13*	0.11	0.08	0.12*	0.06	0.06*	0.10	0.08	0.04	0.14*	0.08	0.06	0.17 ± 0.06
FA of hand breadth	0.01	0.03	0.04	0.05	0.06	0.02	0.05	0.03	0.02	0.05	0.03	0.03	0.15**	0.15	0.08	0.11 ± 0.01
FA of foot length	0.08	0.04	0.02	0.10	0.09	0.03	0.09	0.06	0.09	0.05	0.07	0.18*	0.14*	0.12	0.15**	0.16 ± 0.06
FA of foot breadth	0.07	0.04	0.03	0.04	0.05	0.03	0.05	0.04	0.01	0.05	0.02	0.07	0.11	0.09	0.07	0.08 ± 0.00
FA of bicondylar diameter at elbow	0.04	0.06	0.04	0.05	0.06	0.05	0.05	0.05	0.01	0.08	0.04	0.06	0.05	0.07	0.06	0.12 ± 0.02
FA of bistyloid diameter at wrist	0.04	0.09	0.12	0.11	0.06	0.09	0.08	0.07*	0.01	0.09	0.05	0.06	0.07	0.14*	0.07	0.14 ± 0.03
FA of bicondylar diameter at knee	−0.02	0.05	0.05	0.06	0.09	0.05	0.07	0.06	0.09	0.11	0.09*	0.10	0.02	0.06	0.06	0.11 ± 0.02
FA of bimalleolar diameter at ankle	0.09	0.08	0.03	0.03	0.05	0.08	0.04	0.06	0.16*	0.06	0.12*	0.05	0.06	0.03	0.05	0.10 ± 0.01
FA of length of middle finger	−0.10	0.02	0.01	0.06	0.10	0.01	0.08	0.04	0.05	0.08	0.06	0.04	0.12	0.14*	0.10*	0.12 ± 0.04
FA of thumb length	−0.03	0.01	0.02	0.14*	0.02	0.01	0.09	0.04	0.10	0.02	0.06	0.06	0.11	0.07	0.08	0.11 ± 0.01
FA Of palm length	0.06	0.04	0.06	0.14*	0.12	0.05	0.12*	0.08*	0.16*	0.12	0.14**	0.07	0.13	0.19*	0.09*	0.18 ± 0.07
DA of physiognomic ear length	−0.02	0.13*	0.06	0.08	0.04	0.09	0.06	0.08*	0.17*	0.10	0.14*	0.13	0.09	0.05	0.08*	0.15 ± 0.05
DA of physiognomic ear breadth	0.11	0.05	0.06	0.09	0.07	0.05	0.08	0.06	0.06	0.13	0.09	0.05	0.17**	0.10	0.10*	0.15 ± 0.06
DA of ear length	−0.09	0.08	0.13	0.03	0.07	0.10*	0.05	0.08*	0.08	0.06	0.07	0.04	0.12	0.11	0.10*	0.15 ± 0.06
DA Of hand length	0.02	0.07	0.00	0.03	0.21	0.04	0.11*	0.07	0.08	0.13	0.09*	0.11	0.23**	0.10	0.11**	0.17 ± 0.08
DA of hand breadth	0.10	0.04	0.01	0.02	0.04	0.04	0.00	0.04	0.04	0.04	0.03	0.11	0.23**	0.24**	0.12**	0.13 ± 0.04
DA of foot length	0.00	0.09	0.08	0.09	0.11	0.10*	0.09*	0.10**	0.16*	0.12	0.14**	0.06	0.12	0.09	0.10*	0.19 ± 0.05
DA of foot breadth	0.03	0.08	0.02	0.02	0.10	0.03	0.06	0.04	0.06	0.03	0.05	0.08	0.17**	0.15	0.04	0.13 ± 0.03
DA of bicondylar diameter at elbow	0.02	0.03	0.01	0.06	0.09	0.01	0.07	0.04	0.03	0.10	0.06	0.02	0.22**	0.09	0.04	0.11 ± 0.02
DA of bistyloid diameter at wrist	−0.04	0.05	0.06	0.07	0.02	0.05	0.04	0.03	0.12	0.03	0.10	0.04	0.05	0.08	0.06	0.10 ± 0.02
DA of bicondylar diameter at knee	0.09	0.07	0.06	0.06	0.09	0.08	0.04	0.07	0.07	0.05	0.05	0.12	0.07	0.09	0.07	0.13 ± 0.03
DA of bimalleolar diameter at ankle	−0.02	0.07	0.04	0.08	0.07	0.04	0.07	0.06	0.10	0.08	0.09*	0.08	0.14*	0.09	0.10	0.14 ± 0.05
DA of length of middle finger	0.07	0.06	0.14*	0.05	0.07	0.04	0.06	0.05	0.07	0.14*	0.06	0.07	0.14*	0.19*	0.11**	0.16 ± 0.05
DA of thumb length	0.00	0.12	0.10	0.07	0.14*	0.10*	0.10*	0.10**	0.13*	0.20**	0.16**	0.11	0.10	0.09	0.10**	0.20 ± 0.07
DA of palm length	0.05	0.03	0.04	0.05	0.05	0.04	0.05	0.03	0.06	0.05	0.05	0.24**	0.18**	0.16*	0.15**	0.17 ± 0.06

*$P < 0.05$; **$P < 0.01$.
Values in parentheses represent number of pairs. HS, Husband–Wife; FS, Father–Son; FD, Father–Daughter; MS, Mother–Son; MD, mother–Daughter; FC, Father–Child; MC, Mother–child; PC, Parent–Child; MidS, Midparent–Son; MidD, Midparent–Daughter; MidC, Midparent–Child; BB, Brother–Brother; BS, Brother–Sister; SS, Sister–Sister; Sib, Sib–Sib.

TABLE 3. Loading of variables by Principal Component Analysis of anthropometric asymmetry

Variables	Factor				
	I	II	III	IV	V
FA of hand length	0.954	–	–	–	–
FA of length of middle finger	0.915	–	−0.356	–	–
FA of foot length	0.898	–	–	–	–
FA of thumb length	0.837	–	–	–	–
FA of palm length	0.763	–	–	–	–
DA of hand length	0.728	–	–	–	–
DA of thumb length	0.692	–	–	–	–
DA of palm length	0.649	–	–	0.308	–
DA of foot length	0.550	–	–	–	–
DA of length of middle finger	0.473	–	–	–	–
FA of eye length	–	0.992	–	–	–
FA of physiognomic ear length	–	0.875	–	–	–
DA of physiognomic ear length	–	0.724	–	–	–
DA of eye length	–	0.662	–	–	–
DA of physiognomic ear breadth	–	0.573	–	–	–
FA of physiognomic ear breadth	–	0.496	–	–	–
FA of hand breadth	–	–	0.806	–	–
DA of foot breadth	–	–	0.794	–	–
FA of foot breadth	–	–	0.615	–	–
DA of hand breadth	–	–	0.583	–	–
FA of bistyloid diameter at wrist	–	–	–	0.724	–
FA of bimalleolar diameter at ankle	–	–	–	0.610	–
FA of bicondylar diameter at elbow	–	–	–	0.598	–
FA of bicondylar diameter at knee	–	–	–	0.516	–
DA of bimalleolar diameter at ankle	−0.311	–	–	–	0.743
DA of bistyloid diameter at wrist	–	–	–	–	−0.657
DA of bicondylar diameter at knee	–	–	–	–	−0.573
DA of bicondylar diameter at elbow	–	–	–	–	−0.461
V. P.	*14.459*	*11.672*	*8.816*	*6.583*	*5.821*
Cum. Var.	*26.263*	*46.531*	*63.452*	*72.482*	*79.753*

Rotation Method: Varimax with Kaiser Normalization, Loading values below 0.30 are omitted.V.P., the variance explained by each factor; Cum. Var., the cumulative proportion of explained.

5.99, df = 3, $P > 0.05$). Thus in Table 4 the results of only general model and environmental model (model 3) were presented.

DISCUSSION

The correlation coefficients between different pairs of relatives of the present study are low but positive in nature, which indicates that there may have been some family factors in these asymmetric traits. But such factors may be genes, common (shared) familial environment, or interaction among these causes (Feitosa et al., 2000). In the present study, much lower correlation values than the theoretical values (as suggested by Fisher, 1918 and Penrose, 1949) may indicate the contribution of environmental effect.

Very low and non-significant spouse correlation clearly discarded the possibility of assortative mating for these traits. The resemblance between mother and offspring exceed than that of father and offspring for most of the traits (71% for FA traits and 50% for DA traits), which may indicate the possibility of maternal effect on these asymmetric traits. Several earlier studies have already found significant positive correlation between asymmetry and maternal smoking, obesity (Kieser et al., 1997), alcohol use (Kieser, 1992; Wilber et al., 1993), parity (Lalumiere et al., 1999), gestational age, and the health status of mother (Livshits et al., 1988).

According to Mather and Jinks (1963), when X-linkage is involved, the correlations are affected proportionally by the number of shared chromosomes. The father-son correlation would be zero, as they do not share X-chromosome in common. Father-daughter, mother-son, and sister-sister correlations are increased and brother-sister correlation is reduced, while brother-brother and mother-daughter correlations remain unchanged. The present study showed that the hypothesis of the presence of X-linked loci is unlikely for asymmetry as most are in contradiction with the hypothesis of X-linked genes.

In the next step of the present study, heritability coefficients were calculated and result

TABLE 4. Familial correlation based on factor

Variable	HW (200)	FS (229)	FD (195)	MS (229)	MD (195)	FC (424)	MC (424)	PC (848)	MidS (229)	MidD (195)	MidC (424)	BB (152)	BS (240)	SS (110)	Sib (502)
Factor I	0.01	0.21*	0.14*	0.03	0.14*	0.17*	0.09	0.13*	0.10	0.10	0.10*	0.16	0.08	0.11	0.08*
Factor II	−0.04	0.17*	0.07	0.10	0.17*	0.09	0.14*	0.12*	0.06	0.15*	0.06	0.14	0.12	0.17*	0.14**
Factor III	0.02	0.09	0.02	0.15*	0.02	0.05	0.09	0.06	0.03	0.04	0.01	0.18*	0.14*	0.19*	0.17**
Factor IV	−0.05	0.18*	0.16*	0.09	0.02	0.16	0.06	0.10*	0.05	0.11	0.08	0.14	0.13	0.18*	0.15**
Factor V	0.03	0.12	0.09	0.15*	0.11	0.09	0.13*	0.07	0.04	0.08	0.03	0.19*	0.15**	0.09	0.14**

Values in parentheses represent number of pairs.
*$P < 0.05$; **$P < 0.01$.

TABLE 5. Segregation analysis of factors extracted from the asymmetric traits

	Factor I		Factor II		Factor III		Factor IV		Factor V	
Parameter	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3	Model 1	Model 3
P	0.317	0.317	0.513	0.613	0.617	0.528	0.504	0.604	0.617	0.392
τ_1	0.724	0.551	0.761	0.705	0.833	0.667	0.892	0.553	0.821	0.547
τ_2	0.317	0.551[a]	0.442	0.705[a]	0.504	0.667[a]	0.507	0.553[a]	0.503	0.547[a]
τ_3	0.108	0.551[a]	0.082	0.705[a]	0.124	0.667[a]	0.108	0.553[a]	0.110	0.547[a]
μ_1	0.317	0.338	0.512	0.631	0.607	0.831	0.689	0.382	0.392	0.732
μ_2	0.228	0.431	0.682	0.432	0.580	0.604	0.441	0.550	0.517	0.382
μ_3	−0.013	−0.510	0.315	0.522	−0.428	0.336	0.317	0.192	0.593	−0.112
σ_1	0.607	0.617	0.561	0.812	0.831	1.587	0.831	0.582	0.730	0.617
σ_2	0.711	0.382	0.731	0.690	0.644	0.620	0.699	0.731	0.504	0.392
σ_3	0.608	0.542	0.559	0.397	0.792	0.582	1.528	0.658	0.392	0.052
h^2	0.714	0.731	0.810	0.655	0.558	0.408	0.382	0.830	0.517	0.631
$-2 \ln L$	1984.521	1986.938	2029.824	2030.372	1943.843	1948.495	2154.553	2156.109	2207.741	2209.164
χ^2	–	2.417 (2)[ns]	–	0.548 (2)[ns]	–	4.652 (2)[ns]	–	1.576 (2)[ns]	–	1.423 (2)[ns]

(n) Number of degrees of freedom.
[a]Parameter constrained to equal parameter value listed above it.
[ns]$P > 0.05$.

showed weak heritability (8–18% for FA and 10–20% for DA), which do not indicate any evidence of genes. These low values are consistent with the heritability of other morphological asymmetric traits both in man (Bailit et al., 1970) as well as in different animal species (Leamy, 1986, 1997; Polak and Starmer, 2001). On the other hand, some studies on man have found significant heritability values of asymmetric traits ranging between 20 and 45% (Livshits and Kobyliansky, 1989; Moller and Thornhill, 1997). But the heritability values of the present study are too low to draw any specific conclusion about it.

The present results also revealed that the heritability of different asymmetric traits vary from each other. The result is consistent with the argument that FA is trait-specific (Soule and Cuzin-Roudy, 1982). The variation in the magnitude of heritability among traits may reflect the variation of the amount of heritable or non-heritable component. FA of physiognomic ear length, hand length, foot length and palm length and DA of all length measurements (except eye length) have heritability of >16%, whereas the other traits showed lower heritability. The present result of asymmetry is consistent with the general anthropometric traits in the respect that the contribution of hereditary components is higher for length measurements than breadth measurements (Kaur and Sing, 1981; Sanchez-Andrez and Mesa, 1994; Susanne, 1975). This similarity between the general anthropometric traits and their asymmetry may indicate the biological validity of the present findings.

The comparative result revealed that, both FA and DA of diameters of upper extremities (diameters of elbow and wrist) have lower heritability than that of the lower extremities (diameters of knee and ankle). It indicates that asymmetry of upper extremities are more influenced by environment than that of lower extremities. The result is not incompatible with the hypothesis of Trinkaus et al. (1994) that upper and lower body articular surface asymmetry is related to bipedal locomotion. This is because upper limb joints are more likely to experience unilateral mechanical loading, while joints in the lower limbs experienced roughly equal magnitude of mechanical loading during bipedal gait (Plochocki, 2004). Thus, higher environmental influence (bilateral functional differences) may lead to lower heritability of the asymmetry of diameters of upper extremities.

It appears that when significant correlations were observed, traits may be developmentally and functionally closely related (Livshits et al., 1998) and that the correlated variables are measuring something in common to some extent (Chopra, 1979). Several previous studies also showed some significant correlations among various morphological asymmetric traits (Dufour and Weatherhead, 1996; Leamy, 1994; Livshits et al., 1998). Thus the factors extracted from these asymmetric traits are as important and reliable for overall analysis as single trait is, since these factors are "at the same time more general and meaningful anatomically and more specific genetically" (Howells, 1953). In the present study, PCA was carried out to reduce the number of related traits to a small number of defined factors.

By this analysis five factors were extracted, which show low but significant correlations between the relatives. To search the cause of such familial resemblance, segregation analysis of factor score was carried out, which clearly reject the sporadic model indicating that the effect, which influence the asymmetry, is transmitted in the family. The nature of transmission is not consistent with no major gene hypothesis (τ's equal to P' model) indicating the possibility of existence of major gene (MG). But the rejection of "Mendelian model" failed to establish the evidence of the existence of any MG effect. To infer a putative major locus, the rejection of "no major gene effect" and "equal τ's model" as well as non-rejection of Mendelian model are required. Thus, the result failed to decipher the existence of MG effect on these asymmetric traits. The only model that is accepted for all five factors is the environmental model with equal τ's. However, all these factors also rejected "no-polygenic component" hypothesis, which indicates a possibility of the existence of minor gene (polygenes).

But the problem is that the present study probably represents the first application of genetic model test on anthropometric asymmetry and thus, due to the non-availability of literature, the present study could not be compared with the earlier studies. Asymmetry of morphological traits is mostly studied with respect to dental asymmetry (Keiser, 1992; Roberts and Potter, 1981) and handedness (McManus, 1991; Plato et al., 1980; Yeo et al., 1997) relative to the other body dimensions (Little et al., 2002; Schell et al., 1985). Some studies on the inheritance of functional asymmetry are available, the results of which are similar with the present findings. Family data

suggest that hand-clasping is under genetic control, although the data do not fit any straightforward recessive or dominant Mendelian model. On the other hand, Left-handed occurs 8% in the human population. The genetic models that successfully explain the data are those of McManus and Annett (McManus, 1991), which share the feature of incorporating a random component reflecting the biological phenomenon of FA.

However, weak genetic correlation, low heritability, and acceptance of the environmental model do not demonstrate genetic effect on asymmetry. The result, as a whole, are not inconsistent with the hypothesis proposed by the different authors (Corruccini and Potter, 1981) that, asymmetry may serve as an indicator of general buffering capacity of an individual's ontogenetic development.

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