

Genetic Correlations Between Sides and Heritability of Asymmetry for Nonmetric Traits in Rhesus Macaques on Cayo Santiago

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ABSTRACT The use of nonmetric traits for estimation of biological distance is a long-standing practice in biological anthropology. Nonmetric traits can be scored using either the individual or the side of the individual as the unit of measure. If sides of the individual are genetically correlated the use of sides would produce redundant genetic information. For this reason, Korey (*Am. J. Phys. Anthropol.* 53:19-23, 1980) argues for the use of individuals as the unit of measure for nonmetric traits. Ossenberg (*Am. J. Phys. Anthropol.* 54:471-479, 1981), however, argues that bilateral occurrence of nonmetric traits indicates greater genetic liability for the trait and that therefore the sides are the more biologically correct unit of measure.

Genetic correlations for 13 cranial nonmetric traits are estimated for a sample of rhesus macaque skeletons from Cayo Santiago. In addition, heritability of asymmetry is estimated for these 13 traits as a test of Ossenberg's contention that asymmetry is genetically influenced.

Significant genetic correlations between sides support Korey's contention that nonmetric traits should be scored by individual. Only two asymmetry heritabilities were significantly different from zero, providing no significant support for Ossenberg's contention that asymmetry is genetically determined.

Our results support the theory that asymmetry represents a measure of the ability of an organism to buffer stresses. Therefore, a measure of the heritability of asymmetry is a measure of the heritability of the ability to buffer stresses. This ability does not appear to be heritable in this sample.

Nonmetric skeletal traits are of significance to physical anthropologists because their apparent genetic component permits the study of biological distance between skeletal populations (Brothwell, 1959; Rightmire, 1972; Buikstra, 1976; Ossenberg, 1976, 1977; Jantz, 1974). Ideally, such studies require the use of genetically based traits (Uebelaker, 1978) that are free of age or sex effects or cultural influences such as cranial deformation (Buikstra, 1976). In practice, many traits are used that do not meet these requirements. The common assumption behind biological studies of paleopopulations is that small populations exhibit genetic differences

that are primarily due to stochastic processes such as genetic drift. If similarities are found in the occurrence of genetically influenced traits, this is assumed to represent the presence of natural selection or nonselective processes such as gene flow (Jantz, 1974). It is on this basis that biological distance studies are used to attempt to determine biological and cultural affinities of prehistoric populations.

Years of research involving nonmetric skeletal traits have failed to resolve the question

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of how to properly score trait frequencies. There are two methods currently under discussion. The first uses the individual as the unit of measure and figures trait frequencies as the proportion of individuals exhibiting the trait on either or both sides. This is based on the assumption that there is a significant degree of genetic correlation in the bilateral expression of nonmetric traits. The alternative method uses the side of the individual as the unit of measure and figures trait frequencies as the proportion of sides exhibiting the trait. Unless corrections for correlations have been made, the use of sides as the unit of measure assumes that the expression of nonmetric traits is independent between sides. Each side of the individual must then be considered as a separate unit, or underenumeration of trait frequencies will result. Clearly the fundamental difference between these two methods is whether or not nonmetric traits are assumed to be bilaterally independent.

Recently several authors have addressed the question of how to score nonmetric traits. These works focus on which method provides the least statistically biased but most genetically and biologically accurate method of scoring nonmetric traits. Green et al. (1979) are concerned with statistical bias in biological distance measures caused by phenotypic correlations of nonmetric traits between sides. Green and his colleagues devised a statistical method to control for this type of bias.

But Green et al.'s (1979) correction applies to phenotypic correlations only. It does not address the problem of whether sides provide redundant genetic information and is, in practice, difficult to use. Phenotypic correlations include correlations due to both genetic and environmental effects. As a result the true genetic relationship between sides may be masked by environmental factors. Since evolutionary inferences are being sought, it is this genetic relationship between sides that is important in determining the biologically optimal scoring method. This genetic relationship is measured by genetic correlations, which are caused by pleiotropy and/or linkage disequilibrium (Falconer, 1981).

Green et al. (1979) reject the use of the individual to score nonmetrics because this will lead to an underestimation of the true population frequency in cases of poorly preserved material. If sides are strongly genetically correlated, however, the issue of poorly preserved remains becomes unimportant as

examination of either side is adequate to obtain an estimate of the true population frequency.

Korey (1980) addresses the question of whether sides are genetically correlated. If two sides of an individual are completely correlated, then a given nonmetric trait will always be present on both sides. Certainly this is not the case and therefore it is the meaning of asymmetrical expression of nonmetric traits that must ultimately be addressed. Fluctuating asymmetry occurs when trait expression is random with respect to sides. Earlier investigators have argued that fluctuating asymmetry is due to environmental influence (Gruneberg, 1952; Beardmore, 1960; Bader, 1965; Finkel, 1971; Buikstra, 1976; Doyle and Johnston, 1977; Di Bennardo and Bailit, 1978; Trinkaus, 1978; Owsley, 1980; Sciulli et al., 1979). Korey tests this theory by examining the occurrence of supraorbital foramen in 124 Haida crania from several sites in British Columbia. He observes that the proportion of bilateral occurrences of supraorbital foramen increases with developmental age and concludes that unilateral occurrence is an "ontogenetically transitional phase" of "little or no genetic significance" (Korey, 1980, p.21). "That over the course of skeletal maturation the genotypic classes underlying modes of trait expression should not be uniformly maintained, becoming instead progressively differentiated" (Korey, 1980, p.22) fits best with the model of asymmetry in response to environmental influences. The crux of Korey's hypothesis is that additive genetic effects are the same for both sides of an individual.

The alternative point of view is represented by Ossenberg (1981). Ossenberg is also concerned with the genetic meaning of bilateral versus unilateral expression of nonmetric traits. She argues that bilateral expression of a nonmetric trait represents greater genetic liability for that trait and that therefore it is critical to use sides for frequency counts in order to weight such occurrences properly.

Ossenberg tests her hypothesis by examining the pattern of trait occurrences of mylohyoid bridging in 1,215 adult mandibles and third molar agenesis in 1,266 adult mandibles from a sample of American Indian and Eskimo skeletons. She observes a pattern of increased bilateral occurrence of these traits associated with an overall increase in trait

occurrence. This, she argues, suggests that as genetic liability for a trait increases the probability of bilateral occurrence also increases. This further suggests to Ossenbergs that unilateral (i.e., asymmetrical) expression is due to genetic as well as environmental influences. If this is the case, then treating the entire individual as the unit of measure essentially ignores potential genetic information.

Ossenbergs hypothesis, however, is not consistent with the concept of liability as applied to nonmetric traits. Liability is defined by Falconer (1965) as a graded "attribute immediately related to the causation" (p. 52) of the trait. Liability includes both genetic and environmental factors underlying trait expression. This underlying attribute is defined as being normally distributed, thereby permitting the degree of trait liability to be expressed in standard deviation units. But trait liability itself is a property with a physical basis, presumably related to the mechanics of development. Liability is something "that could in principle be measured. . ." (Falconer, 1981, p. 270). Ossenbergs fails to offer an explanation of the physical or mechanical process that would result in trait expression on only one side of an individual but that, when carried to further extremes, results in bilateral expression. The threshold model for nonmetric trait expression states that there is an underlying continuous liability for a trait with a threshold of trait expression imposed upon it (Gruneberg, 1952) (see Fig. 1A). If trait liability is below this threshold the trait is not expressed. If the threshold is surpassed trait expression results. Ossenbergs presents, however, a model such as that diagrammed in Figure 1B. She posits an additional threshold on trait liability above which asymmetry will be expressed, below which it will not. It is this threshold that seems logically inconsistent with the definition of trait liability. Ossenbergs does not explain how liability can form a continuum such that bilateral expression *requires* more liability than unilateral expression.

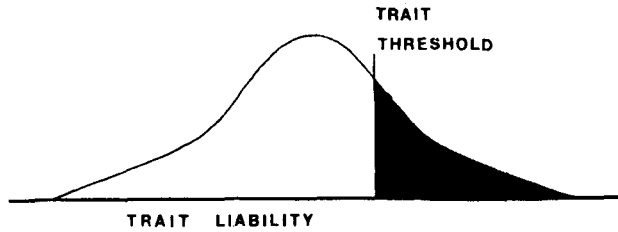
If trait liability is plotted against the probability of asymmetry, Ossenbergs theory can be diagrammed as in Figure 2A. The probability of asymmetry is zero below the unilateral threshold and rises to one as this threshold is surpassed. Probability again drops to zero as the bilateral threshold is surpassed. Therefore, only those individuals falling between the two thresholds exhibit

asymmetry. We propose a model in which the probability of asymmetry is greater the closer the individual is to the threshold of trait expression (Fig. 2B). Individuals on either side of the threshold are equally likely to have unilateral expression as a result of their proximity to the trait threshold. Individuals at a distance from the trait threshold—on either side—also have an equal, if lower, probability of asymmetry. Therefore, it is the proximity to the trait threshold that is important. In our model, Ossenbergs unilateral and bilateral thresholds are not thresholds at all but correspond to the curve describing trait liability and probability of asymmetry.

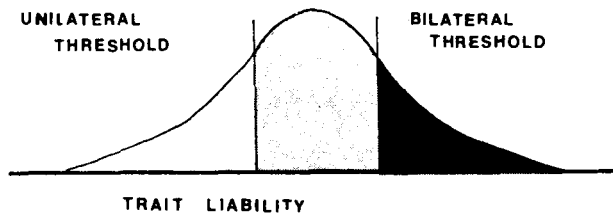
A direct way to test the genetic influence on asymmetry is to measure the heritability of asymmetry (Reeve, 1960; Singh, 1970; Soule and Cuzin-Roudy, 1982). Heritability is a measure of the proportion of total phenotypic variance which is due to additive genetic variance. It represents a measure of the "degree of correspondence between phenotypic value and breeding value" (Falconer, 1981, p. 149). The breeding value of an individual is the sum of the average effects of its genes (Falconer, 1981). If asymmetry is influenced by genetic factors then heritability of asymmetry estimates should be greater than zero. That is, there should be some evidence that some phenotypic variance in asymmetry is explained by genetic factors.

The test for the validity of these scoring methods is a test of whether the genetic basis of the two sides of an individual is the same. This is tested by calculating genetic correlations. Heritability of asymmetry estimates serve to test Ossenbergs suggestion of a genetic influence on asymmetry. The difference between heritability and genetic correlation estimates is fundamental. Heritability measures the relationship between phenotypic values and additive effects of genes and represents the degree to which phenotypic variation is influenced by genes. Genetic correlations measure the relationship between breeding values for each side and represent the degree of similarity between entities. Therefore, these two measures are theoretically distinct and high values for both sets of estimates is not an uninterpretable result.

The results of this test have implications for the study of asymmetry in general. If the genetic correlation between sides is one, then nongenetic factors are responsible for side differences in trait expression. That is, asym-



**A. THRESHOLD MODEL OF TRAIT
EXPRESSION**



B. OSSENBERG'S MODEL

Fig. 1. Frequency distribution of trait liability. The threshold model of nonmetric trait expression states that there is an underlying continuous liability for the trait with a threshold of trait expression imposed upon it. A) A threshold of trait expression divides the distribution into two phenotypic classes: absence of the trait (un-

shaded) and presence of the trait (shaded). B) Osseberg's model: Two thresholds divide the distribution into three phenotypic classes: absence of the trait (unshaded), unilateral presence of the trait (light shading), and bilateral presence of the trait (dark shading).

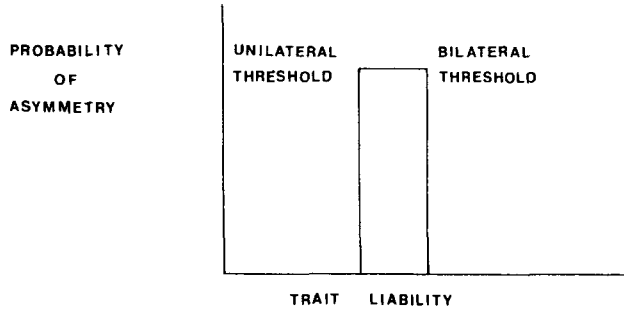
metry is due to environmental effects. The heritability of asymmetry can thus be considered as a measure of the heritability of the ability of an individual to buffer such environmental effects (i.e., the ability to canalize development) (Mather, 1953). If this is indeed the case then it may be feasible to use sides of an individual as genetic "twins," in a sense, for studying the effects of environmental factors on the same genes. This suggests at least one important potential use for studies of asymmetry of nonmetric traits.

MATERIALS AND METHODS

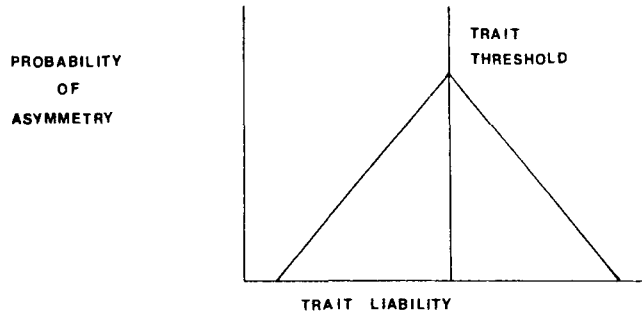
Data from individuals of known relationship are necessary in a quantitative analysis of this nature. Such data are available from the sample of rhesus macaque skeletons (*Macaca mulatta*) from the research colony on Cayo Santiago, Puerto Rico. The macaques

on Cayo Santiago are a free-ranging population established in 1938. The history of the island is presented elsewhere (Altmann, 1962; Buettner-Janusch et al. 1974; Carpenter, 1972; Cheverud, 1979; Koford, 1965; Sade et al., 1976). In 1969 retrieval and identification of the remains of animals dying on the island was initiated. Groups E, H, A, and K were systematically removed and some individuals, including all of group K, were added to the skeletal collection (Sade et al., 1976). The present study uses 442 individuals, including 133 mother-offspring pairs.

A set of 13 nonmetric traits of the cranium were scored for each individual (Table 1). J.M.C. scored one third of the individuals and J.E.B. scored the remaining individuals. Scoring was cross-checked and found to be concordant between scorers. The traits are all bilateral and have been demonstrated to



A. OSSENBERG'S MODEL



B. PROPOSED MODEL

Fig. 2. Two models of trait liability plotted against the probability of asymmetry. A) Ossenbergs model: The probability of asymmetry is zero to the left of the unilateral threshold and to the right of the bilateral threshold.

The probability rises to one between the two thresholds. B) Proposed model: Individuals on either side of the threshold may exhibit asymmetry but the probability of liability increases with the proximity to the threshold.

TABLE 1. Nonmetric cranial traits

1. Fronto-nasal contact (FNC)
2. Supraorbital notch (SON)
3. Accessory supraorbital structures (ASOS)
4. Extrasutural position of the zygomatico-frontal foramen (ZFFP)
5. Number of zygomatico-frontal foramina (ZFFN)
6. Number of infraorbital foramina (IOF)
7. Accessory zygomatico-facial foramen (FZZ)
8. Divided hypoglossal canal (DHYC)
9. Lateral pterygoid bridging—medial (LPBM)
10. Extrasutural position of the occipital foramen (OCCFP)
11. Number of occipital foramina (OCCFP)
12. Infraorbital suture (IOS)
13. Lateral pterygoid bridging—lateral (LPBL)

be independent of age and sex (Cheverud, 1979). For all traits partial expressions are scored as "present." The majority of the traits are scored as present or absent but some are scored for presence of more than two foramina (e.g., ZFFN) or for positioning of a foramen outside of a suture (e.g., ZFFP).

Genetic correlations between sides depend on the heritability of that trait. Therefore, the heritability of each trait is calculated first, followed by an estimate of the genetic correlation. Heritability, as defined above, measures the proportion of total phenotypic

variance which is due to additive genetic variance:

$$h^2 = V_A/V_P, \quad (1)$$

where h^2 is the heritability estimate, V_A is the additive genetic variance, and V_P is the phenotypic variance (Falconer, 1981). Heritability scores indicate the degree of genetic influence on variation in trait expression.

Since nonmetric traits are discrete and considered to be quasi-continuous (Gruneberg, 1952; Falconer, 1981), a modified method of calculating heritability estimates is required. Falconer (1965) has devised a method of calculating the heritability of discontinuously expressed traits based on the continuously distributed liability for trait expression. Falconer's method uses the regression of offspring on parent values to estimate heritability of trait liability for quasi-continuous traits. Heritability is equal to twice the regression of offspring on mother:

$$h^2 = 2b, \quad (2)$$

where b is the regression coefficient and

$$b = (x_g - x_r)/a_g, \quad (3)$$

where x is the mean liability as measured in standard deviation units from the threshold and g and r represent the general population and the relatives of those individuals exhibiting the trait respectively; and a_g represents the deviation of the mean liability of affected individuals from the mean liability of the general population (Falconer, 1965). The mean values of x_g , x_r , and a_g are obtained from the data on trait incidence in the population (Falconer, 1965).

An alternative method of calculating heritability scores focuses on the trait itself, rather than trait liability. Phi coefficients derived from a chi-square analysis represent the Pearson Product Moment correlations for 0-1 traits. Heritability is twice the correlation coefficient between the mother and the offspring:

$$h^2 = 2\phi. \quad (4)$$

This method is also employed to calculate trait heritabilities and the results of both methods are compared.

Both methods of estimating heritabilities are easily adapted to estimation of heritability of asymmetry. A new trait is derived by scoring "0" if the trait is present or absent on both sides and "1" if it is present on only one side. Each new asymmetry trait is designated by the original trait name preceded by "A" (e.g., AFNC, ASON). The newly defined asymmetry traits are used in heritability estimates as described above.

Using heritability estimates from each side of an individual, genetic correlations between sides are obtained as follows:

$$r_G = (b_{ij} + b_{ji}) / \sqrt{h_i^2 h_j^2}, \quad (5)$$

where b is the cross-covariance between mother and offspring and h^2 is the heritability (Cheverud, 1979)¹. The subscripts refer to sides i and j , respectively. Using Falconer's method this equation gives an estimate for the genetic correlation of the trait liability. If phi coefficients are inserted in place of the cross-covariances, b_{ij} and b_{ji} , the genetic correlation between sides for the trait itself is estimated.

For completeness, phenotypic and environmental correlations are also presented here. Phenotypic correlations are calculated as the regression of offspring on parent using equation 3 (see Falconer, 1965, 1981). Phenotypic correlations represent both environmental and genetic influences on trait expression. Environmental correlations represent the correlations present between sides that is due to commonality of environmental factors. It is defined as

$$r_E = (r_p - h_i h_j r_G) / e_i e_j, \quad (6)$$

where r_p is the phenotypic correlation, h represents the square root of heritability for the sides i and j , respectively, and r_G is the genetic correlation (Falconer, 1981). The elements of the denominator are defined as

$$e = \sqrt{1 - h^2}.$$

¹The formula as it appears in Cheverud 1979 is:

$$r_G = (b_{ij} + b_{ji}) / 2 \sqrt{h_i^2 h_j^2}.$$

The "2" in the denominator is a typographic error and has been removed here.

If there are no genetic correlations, then the environmental and phenotypic correlations are equal.

These tests produce contrasting expectations for Korey's and Ossenbergs theories. If Korey is correct then genetic correlations between sides should be at or near one. If Ossenbergs theory is correct, the genetic correlations should be at or near zero. These are statements of the extremes of these theories but intermediate results do not necessarily invalidate either hypothesis. In addition it is expected that the pattern of results obtained will be the same using both Falconer's method and phi coefficients. Ossenbergs (1981) expects high heritability of asymmetry estimates as support for her claim that asymmetry is genetically influenced.

RESULTS

The heritability estimates for the 13 non-metric traits are given in Table 2. There are ten (41.7%) heritability estimates that are significantly different from zero at the .05 level using Falconer's method and five (20.8%) heritability estimates that are significant at the .05 level using phi coefficients. The range of heritability scores is large, but

all have high standard errors. Heritability estimates are theoretically limited to values between zero and one. Values exceeding these limits occur in this study because the covariance of relatives is used as an estimate of the sample value in lieu of the sample additive genetic variance (Cheverud and Buikstra, 1981b). Nevertheless, there is evidence for a relatively high ratio of additive genetic to phenotypic variance. There is no significant correlation between heritability estimates for each side as a whole ($r_S = .027, P = .466$). A Spearman's rank correlation coefficient (r_S) compares Falconer's method and phi coefficients for each side. For the right side $r_S = .668, P = .01$. For the left side $r_S = .82, P = .022$. Therefore, both methods render similar results.

Cheverud and Buikstra (1981a) examine the heritability of these traits for the right sides only. They compare those traits that score the number of foramina with those that occur as variation in ossification of connective tissue (hyper- or hypostotic traits; Ossenbergs, 1970). Cheverud and Buikstra (1981a) find a tendency for nonmetric traits of the first type to have lower heritabilities than hyper- or hypostotic traits. A similar comparison using the Mann-Whitney U test (Blal-

TABLE 2. Heritability of 13 nonmetric cranial traits for sides using Falconer's method (h^2F) and phi coefficients (h^2) (one-tailed test)

Trait	h^2F^{\dagger}	SE h^2	P = .0	h^2	P = .0
RFNC	.698*	.328	.017	.588*	.001
LFNC	.725*	.322	.012	.422*	.019
RSON	.680*	.377	.036	.352	.078
LSON	-.227	.41	.29	.076	.919
RASOS	.439	.436	.157	.206	.534
LASOS	1.105*	.447	.007	.748*	.0005
RZFFP	.345	.327	.146	.266	.239
LZFFP	.334	.335	.16	.138	.656
RZFFN	.56	.475	.119	.31	.133
LZFFN	-.005	.52	.496	.122	.725
RIOF	.370	.281	.094	.264	.13
LIOF	-.054	.352	.439	-.12	.488
RZFF	.205	.52	.347	.252	.340
LZFF	1.237*	.523	.008	.604	.146
RDHYC	.735 [†]	.394	.031	.222	.201
LDHYC	.593	.465	.101	.052	.764
RLPBM	1.08*	.354	.001	.46*	.008
LLPBM	1.034*	.335	.001	.26	.134
ROCCFP	1.113*	.375	.001	.654*	.002
LOCCFP	††	—	—	-.182	.73
ROCCFN	.219	.418	.300	.124	.718
LOCCFN	.206	.381	.294	.242	.315
RIOS	.484	.384	.104	.156	.626
LIOS	††	—	—	††	—
RLPBL	.672	.442	.069	.29	.097
LLPBL	.814*	.403	.022	.252	.145

[†]The heritability values for the right sides of these traits are reported by Cheverud and Buikstra (1981a). Differences with those reported here are due to rounding error.

^{††}Undefined values.

*Significant at the .05 level.

ock, 1972) for the left sides does not indicate the same pattern ($P = .451$). Therefore, the left sides do not provide evidence that the heritabilities of these two types of traits differ significantly.

Phenotypic, genetic, and environmental correlations for the 13 nonmetric cranial traits are presented in tables 3 and 4. All phenotypic correlations are significantly different from zero at the .05 level or better. Falconer's method results in five (38.5%) undefined values for genetic correlations. Genetic correlation values are undefined if heritabilities are less than zero or there is zero trait frequency in relatives. Of the remaining eight traits, six (71%) have correlations that are significantly different from zero at the .01 level. Only one of the eight genetic

correlations with defined values is significant at the .05 level using phi coefficients. However, the results of the methods are comparable ($r_S = .905$, $P = .008$). Standard errors are seemingly quite low. This is explained by the fact that the phenotypic and genetic correlations are generally close in value. As the difference between phenotypic and genetic values increases so does the standard error for genetic correlations. Since the values are similar standard errors are low. In addition, there are high heritabilities for these traits which also serve to lower the standard error.

Eight values for environmental correlations (Falconer's method) are undefined (61.5%) while four of the remaining traits (80%) are significant at the .01 level. Envi-

TABLE 3. Phenotypic, genetic, and environmental correlations for 13 nonmetric traits (Falconer's method)

Trait	r_p	SEr_p	r_G	SEr_G	r_E	SEr_E
FNC	1.046*	.108	1.009 [†]	.011	1.14	.093
SON	.608*	.09	†	—	—	—
ASOS	.336*	.134	.218	.174	†	—
ZFFP	.799*	.095	.766	.143	.817*	.068
ZFFN	.833*	.071	†	—	—	—
IOF	.502*	.101	†	—	—	—
ZFF	.99*	.118	1.446*	.380	†	—
DHYC	.609*	.116	1.306*	.144	-.771	.762
LPBM	1.074*	.038	1.067*	.019	—	—
OCCFP	.489*	.120	†	—	—	—
OCCFN	.575*	.108	.554	.395	.580 [‡]	.111
IOS	.639*	.118	†	—	—	—
LPBL	.802*	.110	.741*	.073	1.028 [‡]	.174

[†]Undefined values.

[‡]Significant at the .01 level.

*Significant at the .05 level.

TABLE 4. Phenotypic, genetic, and environmental correlations for 13 nonmetric cranial traits (phi coefficients)

Trait	r_p	$P = .0$	r_G	$P = .0$	r_E	SEr_E
FNC	.699*	0	1.082**	.019	.327**	.049
SON	.462*	.0000	†	—	-.008**	.035
ASOS	.146*	.0073	.229	1.000	.125	.076
ZFFP	.565*	0	1.028	1.000	.463**	.015
ZFFN	.620*	0	†	—	.558**	.012
IOF	.35*	.0000	†	—	.257**	.018
ZFF	.632*	0	1.179	.149	.316**	.043
DHYC	.31*	.0000	2.33	.764	.070**	.024
LPBM	.661*	0	1.24	.134	.119	.062
OCCFP	.298*	.0000	†	—	.128**	.038
OCCFN	.358*	.0000	.710	.718	.288**	.017
IOS	.334*	.0000	††	—	—	—
LPBL	.523*	0	.673	.532	.468**	.018

[†]These correlations were eliminated in Table 3.

^{††}Undefined values.

[‡]Significant at the .01 level.

**These values are significant at the .05 level.

ronmental correlation values are undefined if heritabilities are less than zero (this produces an undefined denominator) or r_G is undefined. Using the alternative method one value (7.7%) is undefined and ten of the remaining traits (83.3%) are significantly different from zero at the .05 level.

It is interesting to test the relationship between phenotypic and genetic correlations for each method. A comparison of values estimated from Falconer's method shows a positive relationship ($r_S = .619$, $P = .05$) suggesting that those traits with relatively high phenotypic correlations can be expected to have high genetic correlations as well. The same comparison using phi coefficient estimates, however, indicates no such relationship ($r_S = -.200$, $P = .25$).

The heritability of asymmetry estimates are given in Table 5. Only two (15.4%) heritability of asymmetry estimates are significant for each method. Falconer's method and phi coefficients as applied to asymmetry also produce comparable results ($r_S = .733$, $P = .008$). These results provide little evidence of a strong genetic influence on asymmetrical expression of these 13 traits.

There is no relationship between genetic correlations and heritability of asymmetry estimates in this sample ($r_S = -.5$, $P = .111$ [Falconer's method]; $r_S = -.5$, $P = .264$ [alternative method]). This means that there is not a significant relationship between the genetic correlation and the degree to which genetic variance accounts for phenotypic variance in asymmetry in these traits.

DISCUSSION

The evidence from our study supports Korey's call for scoring of nonmetric trait frequencies by individual. Korey (1980) is correct in noting that "the pertinent question becomes whether additive genetic factors are to account for these differences in the form of expression" (p. 20). The results of the examination of genetic correlations indicate that sides are neither perfectly correlated nor perfectly independent. If genetic correlations of greater than .5 are considered high it can be seen that the range of values for this sample tends to be quite high, with several values even surpassing the theoretical limit of 1.0. That there is some degree of genetic correlation supports Korey's contention that scoring by individual is less likely to produce redundant genetic information than scoring by sides.

Ossenberg's contention that unilateral expression provides useful genetic information is not supported by our data. Ossenberg's examination of patterns of trait occurrence is inadequate to evaluate genetic influence on trait expression. Therefore, heritability of asymmetry estimates are used. The majority of heritability of asymmetry values are low and nonsignificant (Table 5), providing no evidence for genetic influence on asymmetry. Additive genetic variance does not explain a significant proportion of phenotypic variance in liability for asymmetry or for asymmetry itself.

The wide range of genetic correlations observed for these 13 traits suggests that researchers must choose carefully which set of nonmetric traits are most appropriate for their research. Our data do not necessarily reflect the situation in other mammalian populations because genetic correlations and heritability estimates are population specific. Our study uses a nonhuman sample and caution is also necessary when generalizing to the study of human remains.

Two methods of analysis are used in this study. The first is based on the threshold theory of quasi-continuous traits (Falconer, 1965) and is concerned with estimates pertaining to the underlying trait liability. The second method uses phi coefficients and derives estimates pertaining to the discrete trait itself. In all cases there is a significant correlation between the results of each method. Although phi coefficients routinely produce lower values than Falconer's method this is an artifact of the method and is expected mathematically. The results are comparable regardless of which technique is employed. However, the interpretation of each is quite different.

The implication of these results goes beyond the issue of how to score nonmetric traits. It is the meaning of asymmetrical occurrence that is of ultimate interest. The most common type of asymmetry occurs when either side of an individual is equally likely to display the trait, that is, when there is no direction to the asymmetry (Van Valen, 1962). Such asymmetry is termed "fluctuating asymmetry" (Van Valen, 1962). The generally accepted model states that fluctuating asymmetry is a result of random disruptions in development. If sides are correlated, as they appear to be, it may be possible to consider the sides of an individual as "twins" in a genetic sense and to view asymmetry as a

TABLE 5. Heritability of asymmetry in 13 nonmetric cranial traits by Falconer's method (h^2F) and phi coefficients (h^2)

Trait	H^2F	SEh^2	$P = .0$	h^2	$P = .0$
AFNC	.57	.834	.247	.108	1.0
ASON	.225	.31	.234	.366	.067
AASOS	1.037*	.346	.001	.66*	.0006
AZFFP	†	—	—	-.24	.356
AZFFN	.78	.558	.081	.276	.268
AIOF	.172	.521	.371	.004	.986
AZFF	.546	.465	.121	.254	.264
ADHYC	.427	.465	.181	.160	.356
ALPBM	-.384	.525	.232	-.104	.805
AOCCFP	1.14*	.477	.008	.548*	.006
AOCCFN	-.392	.465	.2	-.004	1.0
AIOS	.044	.460	.462	-.004	1.0
ALPBL	.04	.616	.474	.074	.962

†Undefined values.

*Significant at the .01 level.

result of differences between sides with respect to nongenetic factors.

By the model stated above asymmetrical expression reflects the degree of developmental homeostatis during ontogeny (Bader, 1965; Beardmore, 1960; Di Bennardo and Bailit, 1978; Lerner, 1954; Sciulli et al., 1979; Siegel and Doyle, 1975; Siegel et al., 1977, 1980; Siegel and Smookler, 1973) and can be considered a measure of development stability for a specific character (Soule and Cuzin-Roudy, 1982). Developmental stability reflects an organism's ability to canalize development. Canalization of development is the tendency for development to "run in grooves" (Waddington, 1957), that is, to remain stable under varying conditions. The better an organism is able to buffer the environmental stresses that disrupt canalization the more likely it is to achieve developmental stability. Given this, asymmetry represents a measure of the ability of an organism to buffer stresses (i.e., to canalize development).

Our study supports the model of nongenetically induced asymmetry. Therefore, what we have tested with heritability of asymmetry estimates is the heritability of the ability to canalize development. This ability is not highly heritable in this sample. Future testing and consideration of this possibility is imperative.

We conclude that scoring nonmetric trait frequencies by individual is the more geneti-

cally and biologically correct method of scoring trait frequencies. We also propose that given that asymmetry reflects developmental disruption by nongenetic factors, then the heritability of asymmetry is an appropriate estimate of the heritability of an organism's ability to canalize development.

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