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Quantitative Genetics of Cranial Nonmetric Traits in Randombred Mice: Heritability And Etiology

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ABSTRACT Cheverud and Buikstra (1981) demonstrated a tendency for nonmetric traits representing the number of foramina to have lower heritabilities than those representing hyperstotic or hypostotic traits in a sample of rhesus macaques. Based on this observation, Cheverud and Buikstra hypothesize that differences in the heritability of the two sets of traits may be due to differences in trait etiology. This study addresses the proposed relationship between trait heritability and etiology. Heritability values are calculated for 35 cranial nonmetric traits in a sample of 320 randombred mice using analysis of variance. The results are minimally consistent with the etiological hypothesis, but only 4 of the 35 traits showed statistically significant heritability values. These results are discussed with reference to the assumption that nonmetric traits have a strong genetic component. It is concluded that the developmental pathways that genetic variation traverses before being expressed in the form of nonmetric traits must be understood before variation in nonmetric traits can be used to its fullest potential.

Physical anthropologists have long considered nonmetric traits important variables for studying human skeletal remains. Discrete traits were initially used as descriptive features of the skeleton (e.g., Hooton, 1930) and later as indicators of stresses experienced by the individual (Deol and Truslove, 1957; Gruneberg, 1952; Ossenberg, 1970; Pucciarelli, 1974; Searle, 1954a,b,c). The discovery of the potential role of nonmetric traits in the study of biological relationships between human populations fostered their widespread use by anthropologists (Brothwell, 1959; Buikstra, 1976, 1980; Conner, 1976; Molto, 1983; Ossenberg, 1976; Suchey, 1975). In these studies it is assumed that variation in nonmetric traits has some genetic basis, but the degree to which this is true has not been firmly established for all nonmetric traits. Some argue strongly for the genetic basis of nonmetric traits (Berry and Berry, 1967; Berry, 1968; Berry, 1975). Others argue for a significant environmental component (Corruccini, 1974; Ossenberg, 1970; Suchey, 1975; Trinkaus, 1978).

The genetic basis of nonmetric traits is most easily studied in experimentally bred populations where genetic relationships are known. Laboratory control of diet, noise, temperature, and other features of the general environment allows evaluation of these factors in the development of the traits (Deol and Truslove, 1957; Howe and Parsons, 1967; Searle, 1954a,b,c). Since experimental populations cannot mimic genetic or environmental circumstances found in nature, the results of these studies must be applied with caution.

Alternatively, investigators have tested the genetic basis of variation in nonmetric traits directly by calculating heritability values for traits in nonexperimental, free ranging populations (Berry, 1963, 1964, 1973; Berry et al., 1967; Berry and Jakobsen, 1975; Berry et al. 1978; Cheverud, 1979; Cheverud and Buikstra, 1981; McGrath et al., 1984; Patton, 1975; Rees, 1969). In these studies principles developed in laboratory situations are applied to natural populations. A review of the cited research demonstrates that whatever the characteristics of the research environment, heritability values vary markedly from population to population.

Cheverud and Buikstra (1981) calculated heritability estimates for fourteen nonmetric traits expressed on the right side in a sample

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of free ranging rhesus macaques. In this sample hyperstotic/hypostotic traits had significantly higher heritability estimates than did foraminal traits. Hyperstotic/hypostotic traits result from variable ossification of connective tissue surrounding nerves or vessels (e.g., bridging of hypoglossal canal, Ossenberg, 1970). Foraminal traits represent variations in neurovascular bundles which travel through bone (Cheverud and Buikstra 1981). Cheverud and Buikstra (1981) hypothesize that the observed pattern of heritability estimates might be related to differences in determinants of ontogeny of trait types, that is, to differences in trait etiologies. Yet, in a recent study of the same rhesus population (McGrath et al., 1984) heritability estimates for the same traits on the left side do not match the pattern described by Cheverud and Buikstra (1981). This discrepancy suggests a need to test the etiological hypothesis more thoroughly.

If the pattern reported by Cheverud and Buikstra (1981) is found in other populations, use of hyperstotic/hypostotic traits may be more appropriate than foraminal traits in particular research designs. The potential implications for studies of biological (genetic) relationships between populations are evident. The purpose of this study is to evaluate the pattern of heritability estimates demonstrated by Cheverud and Buikstra (1981) in another study population. We test the hypothesis that a relationship exists between trait etiology and heritability estimates by calculating heritabilities for a set of nonmetric traits in a sample of mice raised in a crossfostering design. Since heritability estimates are population specific, correspondence between exact *values* of heritability estimates with previous studies is not expected. Instead, it is the *pattern* of heritability estimates among the traits that is of interest. Pattern here refers to the level of heritability values for a defined set of traits relative to the heritability values for another set of traits studied within the same population. Specifically, we are interested in the relative levels of heritability estimates for foraminal, hyperstotic/hypostotic, and fusion traits.

MATERIALS AND METHODS

Experimental design

Twenty-eight crossfostering pairs (n = 320 sibling mice) were chosen at random from a population of randombred ICR stock raised in a cross fostering design at the University of Wisconsin, Madison (N = 2693). All mice examined were 70-day-old adults, however some of the mice in the study sample had been used previously to study growth (Cheverud, 1984; Cheverud et al., 1983; Riska et al., 1984). For that purpose, mice were periodically sedated with metaphane to allow measurements to be taken. Heritability studies using the same skeletal population have shown metaphane to have little or no apparent effect on the heritability estimates of metric characters (Cheverud et al., 1983; Kohn and Atchley, 1985). Nonetheless, the effect is removed statistically in this analysis as described below.

In this crossfostering design litters born on the same day were standardized to eight pups, usually consisting of four males and four females. A random half of each litter was then exchanged between pairs of dams which were not full sibs so that a mother's post-natal, or nursing litter is composed of half of her genetic progeny and an equal set of siblings from her paired dam. Rearing progeny in a crossfostering design permits the separate estimation of direct genetic effects from the covariance among full sibs reared apart, as well as an estimation of maternal effects from the covariance among nurse litter-mates (Atchley et al., 1981; Rutledge et al., 1972). Maternal effect is the component of environmental variation that the mother contributes to the young via nursing and overall postnatal maternal care. It is rendered measureable through the implementation of the crossfostering design.

Observations from each crossfostering pair form a two-by-two table in which the values of the two factors are the two mothers (genetic and nursing). The pups are assigned to one of the four cells of the table by matching each pup to its nurse mother (column) and genetic mother (row). Each complete pair yields variation corresponding to one degree of freedom for the effect of genetic mother and one degree of freedom for the effect of nurse, and residual variance. Since different cross fostering pairs were scored by different observers, and additive genetic variance is estimated in this design from the variance of full sibs (see following), inter-observer error contributes minimally to the results as a source of additive genetic variance. Residual variance includes any mother-nurse interaction and variance among full sibs with a common nurse (Riska et al., 1984). (See Rutledge et al., 1972 for further discussion of a full-sib cross fostering design and statistical

methodology for estimasting gentic parameters using this design.)

In this study the use of metaphane (M), replication (R), and sex (S) of the individual were treated as a component of the linear model assumed for analysis of variance so as to remove their affects from analysis. The linear model for the experiment is:

$$Y_{ijklmno} = \mu + M_i + S_j + (SM)_{ij} + R_k + (RM)_{ik} + (RS)_{jk} + (RSM)_{ijk} + P_l + d_{m(l)} + n_{n(l)} + e_{mno(l)}, \quad (1)$$

where $Y_{ijklmno}$ is the occurrence of the trait of the o^{th} pup of the j^{th} sex in the k^{th} replicate (the cross fostering experiment was carried out as two replicates (R) due to space limitations) from the i^{th} metaphane treatment nursed by the n^{th} nurse born of the m^{th} dam nested in the l^{th} pair (Atchley et al., 1984). Effects due to dam (d), nurse (n), and the residual (e) were assumed to be random effects with zero means and variances V_d, V_n, and V_e (Atchley et al., 1984; see also Atchley and Rutledge, 1980; Rutledge et al., 1972). Non-subscript effects enclosed in parentheses denote interaction terms and the subscripts in parentheses denote that the preceding subscript(s) is nested within these terms (Atchley et al., 1984).

The components of variance and mean squares were calculated from data on 56 sibships within 28 crossfostering pairs using the VARCOMP procedure of SAS76 (Barr et al., 1982). The observed components of variance were equated to theoretical causal components assuming a genetic model of additive direct and additive maternal variances and covariances. The variance component due to dams is the covariance between full-sibs reared apart and is free of maternal and common environmental effects as they are included in the variance component which is due to nursing (Rutledge et al., 1972). The variance and covariance of full-sibs reared by different nurses under this design estimate one-half of the additive genetic variance (Atchley and Rutledge, 1980).

The crossfostering design allows the phenotypic variance to be expressed in terms of variance components attributable to different causes as shown in equation 2,

$$V_P = V_A + V_M + V_E, \quad (2)$$

where V_P is the phenotypic variance, V_A is the additive genetic variance, V_M is the variance due to maternal effect, and V_E is the residual environmental variance which includes variance due to environmental effect as well as that component of variation which cannot be ascribed to either genetic or known environmental factors. V_A, the additive genetic variance, is determined by observations on the similarity of full-siblings. V_A is estimated as twice the dam variance, V_D (Rutledge et al., 1972). V_M, the variance due to maternal effect, is estimated by the between-nurse variance. V_P, phenotypic variance, is calculated from observations on the phenotypes of the entire population under study. It includes variation due to all environmental and genetic factors.

Partitioning the phenotypic variance allows evaluation of the relative importance of each of the components of the variance. The heritability estimate, h^2, represents the degree to which trait variation is determined by genetic rather than environmental factors (Falconer, 1981). The heritability of a character is defined as the porportion of total phenotypic variance due to additive genetic effects as seen in equation 3,

$$h^2 = V_A/V_P. \quad (3)$$

The ratio expresses the extent to which phenotypes are determined by genetic information from the parents (Falconer, 1981). The proportion of variance due to post-natal maternal effects, m^2, is defined as,

$$m^2 = V_M/V_P, \quad (4)$$

where V_M is the foster mother component of variance.

Traits considered

Three major etiological categories of nonmetric traits are identified: 1) foraminal, 2) hyperstotic/hypostotic, and 3) fusion. Foraminal traits represent variations in presence, absence, branching of neurovascular bundles which travel through bone, or the relative positioning of bone with respect to the nodes of the branches during development. Foraminal traits were scored as present, absent, or by the number of foramina formed in a specific location. Hyperstotic traits are formed by excessive ossification of structures normally composed of soft tissue whereas hypostotic traits occur due to the lack of ossification of structures normally ossified. Several traits involving foramina are classified as hyperstotic/hypostotic traits (see

Table 1) because the *formation* of the foramen is not the structural variation under consideration. These traits (bridging of foramen ovale, hypoglossal bridging) involve accessory ossification within the foramen. Fusion traits involve the patency of a suture. Previous studies have classified certain sutural variations such as wormian bones in the hypostotic category (e.g., Ossenberg, 1970). Since the formation, growth, and fusion of cranial sutures are specialized processes (Enlow, 1975; Moss, 1957, 1960; Moss and Young, 1960), fusion traits are here considered as etiologically distinct.

A total of 62 cranial nonmetric traits were scored but 27 were dropped from the analysis due to lack of variability in expression (see Appendix A). The 35 traits used in analysis are grouped according to etiology in Table 1. Trait classification is based on past research focusing on the development of selected nonmetric traits (Bennett, 1965; Dodo, 1980; Lillie, 1917; Ossenberg, 1974; O'Rahilly and Muller, 1984), more generalized anatomical research (Greene, 1968; Sidman et al., 1971), and limited exploratory wet dissection of adult mice by Richtsmeier. The classifications are tentative until controlled dissection is conducted for all traits. All but two of the listed traits (anterior ethmoidal foramen for anterior ethmoidal branch of nasociliary nerve and wall separating foramen ovale and alisphenoid canal) have been discussed in earlier works (Table 1).

Each author scored one half of the sample using a binocular microscope. Entire cross-fostered pairs were scored by a single observer in order to minimize inter-observer error. Traits which occur bilaterally were scored separately by side and analyzed as independent nonmetric traits. An ordinal scale of trait expression was developed for those traits which are multi-state in nature (e.g., number of foramina palatina minora), as well as for traits which are considered to be continuous in development (e.g., bridges, tori, spicules) (Corruccini, 1974). Traits scored with more than two states of expression were dichotomized into categories of "present" and "absent" for analysis following a protocol designed by the authors. This procedure is in agreement with many of the traditional studies of nonmetric traits by skeletal morphologists.

RESULTS AND DISCUSSION

Of the 35 traits used in this analysis four have heritability values statistically significant at the .05 level (Table 2). The heritabil-

TABLE 1. Traits used in analysis grouped according to etiological category

Foraminal Traits
+ Frontal foramen (Berry and Searle, 1963)
+ Number of foramina palatina minora (Berry, 1963)
+ Maxillary foramen I (Berry, 1963)
+ Maxillary foramen II (Berry, 1963)
Foramen sphenoidal medium (Deol, 1955)
+ Postcondylar canal
+* Anterior ethmoidal foramen for anterior ethmoidal branch of the nasiocilliary nerve

Hyperstotic/hypostotic traits
+ Bridging of palatinum majus (Berry and Searle, 1963)
+ Bridging of foramen ovale ("foramen ovale double"; Deol, 1955)
+ Hypoglossal bridging (Deol, 1955)
Incomplete ossification of frontal bones ("parted frontals," Truslove, 1952)
+ Processus pterygoideus (Deol, 1955)
+* Wall separating foramen ovale and alisphenoid canal

Fusion Traits
Nasal fusion (Berry and Searle, 1963)
+ Squamosal-frontal fusion (Berry and Searle, 1963)
+ Squamosal-parietal fusion (Searle, 1954c)
+* Post tympanic hook of squamosal-parietal fusion
Basisphenoid-basioccipital fusion (Berry and Searle, 1956)
Basisphenoid-presphenoid fusion (Deol and Truslove, 1957)
+ Preoptic root (Truslove, 1954)

Traits which occur bilaterally are listed once, but their occurrence on each side is considered as a separate trait. References following each trait indicate the research in which the trait was previously defined and used in genetic analysis.
+ Trait occurs bilaterally and was scored and analyzed as two separate traits.
* Trait is newly defined for this study.

TABLE 2. Nonmetric traits with significant heritability values (p = .05)

Trait	h^2	Standard error
Squamosal-parietal fusion on the right side (RSQPF)	.1586	.089
Processus pterygoideus on the right side (RPROP)	.1556	.088
Wall separating foramen ovale and the alisphenoid canal on the left side (LOVWAL)	.1546	.088
Wall separating foramen ovale and the alisphenoid canal on the right side (ROVWAL)	.1951	.099

Abbreviations for traits used in text are listed after trait name.

ity values for these traits range in value from .155 to .195, indicating a low degree of nonmetric trait heritability in this sample. Portions of variability due to maternal factors were never greater than .125, and were not found to be significant at the .05 level. This suggests that the development of nonmetric traits is determined prenatally, or that if they have a postnatal origin, it is influenced by processes other than those affected by maternal care.

Three of the four traits with significant heritability values are hyperstotic/hypostotic traits (RPROP, LOVWAL, ROVWAL). The remaining trait (RSQPF) is a fusion trait. None of the foraminal traits have significant heritability values. These results provide tentative support for the etiological hypothesis proposed by Cheverud and Buikstra.

A more striking result of this study however, is the generalized lack of significant heritability values for the nonmetric traits analyzed. This does not match the findings of research previously cited which reports a large number of nonmetric traits with high heritability values in mammalian populations. In addition, our results do not match other quantitative genetic studies of the entire mouse population from which our sample was selected which report many heritable metric traits (Atchley et al., 1984; Cheverud et al., 1983; Kohn and Atchley, 1985). The sample size used in this study (n = 320) is small in comparison to other studies of nonmetric traits in mouse populations, but falls within the range defined by Falconer (1981) as being appropriate. Since the pattern of our heritability estimates are in accordance with results of Self and Leamy (1978) who used a much larger sample of mice, we do not feel that sample size is a major determinant of the low heritability values.

The relationship between population structure and genetic variability of nonmetric traits is not clear. The population of mice used in this study was obtained from a randombred stock and raised in a laboratory under a crossfostering design. Pioneering studies of the genetic variability of nonmetric traits in mice used one or more inbred stains (e.g., Deol, 1955; Deol and Truslove, 1957; Searle, 1954a; Truslove, 1954). Inbreeding increases genetic differentiation between lines (strains) and genetic uniformity within lines (Falconer, 1981). In a study of another randombred mouse population approximately 52 generations removed from an inbred strain (Self and Leamy, 1978), the majority of 11 nonmetric traits analyzed showed a general low level of heritability ($h^2 \cong 0.20$). Self and Leamy (1978) suggest that 52 generations of randombreeding may not have been sufficient time for increased genetic variability. Cheverud and Buikstra's (1981) results are based on a free-ranging population of rhesus macaques. Since Self and Leamy's (1978) explanation is not appropriate for our sample, the degree to which the structure of our study population (randombred) explains the overall lack of significant heritability estimates for nonmetric traits is undetermined.

Almost half of the traits used in this study were scored using three or more categories and later dichotomized in order to estimate heritability. Dichotomization requires that some categories be lumped together with a resultant loss of information which enters the analysis as measurement error. The type of measurement error introduced by dichotomization appears as environmental variance (Falconer, 1981: 124) and consequently lowers heritability values. When dichotomizing, discrete categorization is imposed on a process which is, in fact, continuous. Since

we cannot demonstrate the biological correctness of the chosen categories, lumping of trait expression may not reflect the biological bases of trait variation. Dichotomization of trait expression may have contributed to the overall lack of heritability in nonmetric traits by increasing phenotypic variance relative to genetic variance. The question that remains, however, is why the effect of dichotomization might be greater in our study than in previous works which have used the same scoring method (e.g., Cheverud and Buikstra, 1981; McGrath et al., 1984).

Richtsmeier et al. (1984) suggest that the development of nonmetric traits follows principles of the functional matrix hypothesis outlined for the formation of other cranial osseous elements (see Moss and Moss-Salentijn, 1979). If this is so (cf. Johnston, 1976), then nonmetric traits occur incidental to the development of other soft tissue features of the face and cranium and may be the outcome of numerous developmental interactions. As the number of developmental sources of trait variation increase, heritability values may decrease because variation in composite traits is often lower than variation in those parts that make them up (Cheverud and Buikstra, 1981). For example, the morphology of a foraminal trait depends on the composition of the neurovascular bundle, the mode of ossification of the bony structure, the position of the bone in relation to the branching of the neurovascular bundle, and various developmental parameters, such as the rate or timing of the various stages of development of all associated tissues. The combined ontogenetic processes of these factors define the possible morphological states of the trait. Each of these processes has a heritability of its own which is a function of the additive genetic variance and the genetic covariances of the developmental parameters (Atchley, 1985). Since a nonmetric trait is an accumulated result of several processes, heritability is a function of the additive genetic variance of each physiological property that makes up the trait and the genetic covariance between all parts. By definition, additive genetic variance has a positive value, but genetic covariances can be either positive or negative. Negative genetic covariances could potentially reduce positive heritability values. Lack of understanding of the developmental dynamics operative in the formation of nonmetric traits confounds an accurate interpretation of heritability estimates for these traits.

The results of this study are therefore inconclusive. The pattern of heritability is consistent with Cheverud and Buikstra's etiological hypothesis. However, we concede only a minimal understanding of the development and variation in mode of expression of these cranial nonmetric traits. The lack of understanding of trait etiology prevents a biologically meaningful method of trait dichotomization. The circularity of the circumstances is painfully obvious: demanding that the knowledge of the causes of trait development is prerequisite to studying trait heritability is an awkard way to end a study whose initial purpose was to examine the hypothesis that different etiological categories of traits have different heritability values.

The low heritability values of nonmetric traits and relatively large magnitudes of environmental variance are consistent with the results of Self and Leamy (1978) and Searle (1954a). This suggests that historically accepted assumptions about heritability of nonmetric traits require continued close scrutiny. Despite years of research in anthropology, genetics, and growth and development, the use of nonmetric traits is still plagued by questions concerning their true significance in skeletal research. Anthropologists interested in the biological relatedness of populations need to be especially careful when choosing nonmetric traits as the vehicle for analysis, and in their selection of the specific traits to be examined. Before generalizations of trait heritabilities are formed, heritability estimates should be calculated for populations from varying mammalian species, and research should be aimed at determination of the mode of development of nonmetric traits. Until a fuller understanding of nonmetric trait etiology is developed, the promise of nonmetric traits will remain unfulfilled.

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APPENDIX A. Nonmetric traits excluded from analysis due to lack of phenotypic variation

+	Preorbital foramen
	Interfrontal
	Frontal fontanelle
	Dorsal frontal fusion
+	Occipital-periotic fusion
+	Metopic root
+	Foramen sphenoidale ventral
+	Fenestra flocculi
+	Foramen ovale open posteriorly
+	Number of mental foramen
	Bregmatic wormian bone
	Lambdoidal wormian bone
+	Bone absorption of eminentia incisive of mandible
+	Extra sutural incisive foramen
+	Bridging of incisive foramen
+	Entrance to interior alveolar canal

+Trait occurs bilaterally.